Published in final edited form as:

J Abnorm Psychol. 2005 November ; 114(4): 612–626. doi:10.1037/0021-843X.114.4.612.

Conjoint Developmental Trajectories of Young Adult Alcohol and Tobacco Use

Kristina M. Jackson and Kenneth J. Sher

Midwest Alcoholism Research Center and Department of Psychology, University of Missouri—Columbia

John E. Schulenberg

Institute for Social Research and Department of Psychology, University of Michigan

Abstract

Developmental and etiological advances have set the stage for considering trajectories of problem behavior across the life course, but little work thus far addresses co-occurring problem behavior trajectories. Although recent work characterizes drinking and smoking trajectories, none has explored the course of concurrent drinking and smoking. Using panel data from the Monitoring the Future Project ($N = 32,087$), the authors applied growth mixture modeling to 4 waves of heavy drinking and smoking in a young-adult sample. The authors extracted a single latent group membership factor from heavy drinking and smoking. Associations between trajectory classes and risk factors were relatively unique to the substance being predicted. The association of smoking with alcohol expectancies and delinquency appeared to exist by virtue of smoking's comorbidity with drinking.

Keywords

alcohol; tobacco; comorbidity; trajectory; developmental

In the past decade, much research has been focused on modeling age trends in substance use and on examining both predictors and outcomes of such trends. Such research on the natural history of substance use has demonstrated that, on average, substance use tends to increase throughout adolescence, decline over young adulthood, and level off by the fourth decade of life (Bachman, Wadsworth, O'Malley, Johnston, & Schulenberg, 1997; Chen & Kandel, 1995). Despite the importance of identifying these normative age trends, however, the emphasis on average change belies the growing evidence that the developmental course of substance use is systematically heterogeneous across the life course with distinct variation in the timing and degree of escalation and duration. Identifying and modeling distinct courses of substance use can reveal the extent to which different risk and protective factors contribute to divergent developmental courses, and may have implications for intervention and treatment utilization, content, timing, and outcome (Schulenberg, Maggs, Steinman, & Zucker, 2001). This "person-centered" or "pattern-centered" emphasis (as opposed to a more "variable-centered" emphasis) has recently gained popularity and is based on understanding individual trajectories over time, rather than understanding average growth over time (Bates, 2000). More broadly, this reflects the growing understanding about the heterogeneity in the course of

Copyright 2005 by the American Psychological Association

Correspondence concerning this article should be addressed to regarding this article should be sent to Kristina M. Jackson, who is now at the Center for Alcohol and Addiction Studies, Department of Community Health, Brown University, Box G-BH, Providence, RI 02912. Kristina_Jackson@brown.edu..

psychopathologies across the life course (Cicchetti & Rogosch, 2002). A key assumption of these “developmental typology” groupings is that the experience of those within a given group is distinctive; for example, those in the heavy chronic use groups use substances (and engage in related behaviors) in distinctive ways compared to those in low substance use groups.

Developmental Course of Alcohol Use and Tobacco Use

During recent years, theoretical and empirical research has begun to chart the longitudinal course of alcohol involvement during adolescence and young adulthood. Theoretical (Zucker, 1987, 1994; Zucker, Fitzgerald, & Moses, 1995) and empirical (e.g., Bennett, McCrady, Johnson, & Pandina, 1999; Chassin, Pitts, & Prost, 2002; Colder, Campbell, Ruel, Richardson, & Flay, 2002; Schulenberg, O'Malley, Bachman, Wadsworth, & Johnston, 1996; Schulenberg, Wadsworth, O'Malley, Bachman, & Johnston, 1996; Tucker, Orlando, & Ellickson, 2003) work suggests that, although clearly there is some variation in course, several prototypical courses have emerged, including a nonuser/stable-low-user course, a chronic or high use course, a “developmentally limited” course (transitioning or maturing out of drinking), and some evidence for a late-onset (increasing) course. Trajectories derived from adolescent samples tend to include more late-onset courses, whereas samples that include young adults tend to show more courses that remit (developmentally limited).

Although there is considerably less research examining developmental course of smoking, there is evidence for developmental pathways of smoking that are similar to these drinking courses. In addition, some researchers have identified a stable light- or moderate- smoking group (which may comprise “chippers,” i.e., those who indulge only occasionally). Colder et al. (2001) identified five patterns of use over six waves (as well as an *a priori* nonsmoking group): early rapid escalators, low moderate escalators, late slow escalators, stable light smokers, and stable puffers. Chassin, Presson, Pitts, and Sherman (2000) characterized six smoking trajectories over six waves: a nonsmoking group and an erratic group were identified *a priori*; and then an early onset stable group, a late-onset stable group, a quitter group, and an experimenter group were empirically identified. Finally, White, Pandina, and Chen (2002) identified three trajectories of smoking over five waves: a heavy-regular smoking group, an occasional-maturing out group, and a nonsmoking-experimental group; however, the two trajectories of smokers were indistinguishable based on risk factors. Note that while these studies generally span late adolescence through early adulthood, the definition and meaning of the trajectory groups tend to vary by ages included.

Developmental Course of Alcohol-Tobacco Comorbidity

One of the fundamental aspects of alcohol involvement is its high co-occurrence with other substances, particularly the strong association between alcohol involvement and tobacco use (henceforth termed *comorbidity*). Alcoholics are more likely to smoke than nonalcoholics (Bien & Burge, 1990; Gulliver et al., 1995; Kozłowski et al., 1993; Martin, Kaczynski, Maisto, & Tarter, 1996; York & Hirsch, 1995), and social drinkers are also more likely to smoke than nondrinkers (Istvan & Matarazzo, 1984). In a similar manner, individuals with a diagnosable tobacco use disorder exhibit greater risk for an alcohol use disorder (Breslau, 1995), and smokers are more likely to drink than nonsmokers (Bien & Burge, 1990; Breslau, 1995; DiFranza & Guerrera, 1990; Torabi, Bailey, & Majd-Jabbari, 1993; Zacny, 1990). Concurrent alcohol and tobacco use interact synergistically to produce greater health risks than expected from use of either substance alone (Bien & Burge, 1990), including elevated rates of esophageal (Munoz & Day, 1996), laryngeal (Flanders & Rothman, 1982), and oral cancers (Blot et al., 1988).

Comorbidity has traditionally been viewed as a cross-sectional phenomenon; that is, the existence of two or more conditions occurring at a single point in time. Implicit in this approach

is that each comorbid condition is adequately characterized as a static entity. Recent data, however, emphasize the importance of course of single disorders or conditions, suggesting that comorbidity should be viewed in the context of the longitudinal course of each co-occurring condition. Despite the recent surge of longitudinal research on comorbidity, however, “too little attention has been given to the implications of diagnostic course, both singly and across related disorders” (Widiger & Clark, 2000, p. 956). Correspondingly, empirical research in the area of alcohol–tobacco comorbidity has generally failed to consider both course and comorbidity, with no research to date examining the concurrent relation between trajectories of smoking and drinking. White, Johnson, and Buyske (2000) used growth mixture modeling to extract univariate trajectories of drinking and smoking of 15-to-28-year-olds, and they examined the predictive utility of parental modeling and parenting behaviors on these patterns of substance use. However, consistent with the emphasis of their study, trajectories of alcohol and tobacco use were independently derived, and there was no discussion of the association between the two. Using five waves of data from a mixed-gender young adult sample ($N = 449$), we examined in our previous work trajectories of combined alcohol use disorders and tobacco dependence using latent class analysis (Jackson, Sher, & Wood, 2000). We extracted five longitudinal types of alcohol–tobacco use disorder over time: (a) nondiagnosing, (b) developmentally limited alcohol use disorder, (c) chronic alcohol use disorder, (d) chronic tobacco use disorder, and (e) comorbid alcohol and tobacco use disorder. This procedure, however, did not explicitly model the temporal ordering of the manifest variables. There exists the potential for correlated errors between an alcohol use disorder and a tobacco use disorder at a given measurement occasion. Muthén (2001) reanalyzed the same data with a mixture modeling approach and identified three classes of alcohol use disorders (AUD) and three classes of tobacco dependence (TD). Next, he estimated joint probabilities between the classes. Although most AUD classes had low TD, nearly a third of chronic and decreasing AUD were increasing in TD; 12% of decreasing AUD and 5% of chronic AUD had chronic TD. The results of these analyses mapped onto what was done by Jackson et al. (2000a)—the five trajectory groups were represented by the five most prevalent cells in Muthén (2001).

This earlier work has demonstrated the feasibility and promise of jointly considering course of multiple disorders but has been limited by both relatively small samples that may be inadequate for detecting relatively rare but clinically important classes and data that are not nationally representative. Also, in contrast to our earlier work focusing on substance use disorders, identification of subtypes based on longitudinal profiles of continuous alcohol and tobacco use can more clearly resolve intensity of substance use than profiles based on dichotomous variables with a relatively high threshold. This resolution may lead to the identification of additional subtypes that better permit us to distinguish between those who are experiencing a “developmental disturbance” (Schulenberg, Maggs, Long, et al., 2001; Schulenberg & Zarrett, in press) versus those who are at risk for continued heavy use, or problems—for example, distinguishing between Zucker’s (1987, 1994) developmentally limited and life-course persistent courses. Nationally representative data are more characteristic of the general population, which is particularly important when examining certain forms of substance use, as college students are more likely to binge drink and less likely to smoke than their noncollege peers (Johnston, O’Malley, Bachman, & Schulenberg, 2004; O’Malley & Johnston, 2002). Moreover, large samples permit detection of relatively rare classes that may be important from a clinical standpoint.

In addition, extant literature on trajectories of alcohol and tobacco involvement fails to consider more than a single cohort.¹ Monitoring the Future (MTF) panel data (Johnston et al., 2004) show secular changes in drinking and smoking over the past 3 decades such that drinking has

¹Although data used by White et al. (2000) contained three cohorts, analyses were restricted to the middle cohort.

become less prevalent, whereas smoking, which decreased in prevalence over the 1980s, has in recent years become more prevalent (particularly among women). Data from the National Household Survey on Drug Abuse (NHSDA) suggest that the recent increase in smoking has been due to cigars, but also to a lesser extent, to cigarettes (Department of Health & Human Services [DHHS], 2000). When considering courses of substance use, it is important to isolate cohort and historical differences from actual developmental trends.

Third-Variable Analyses

Determining the extent to which risk factors distinguish among courses of comorbidity can provide construct validity for the trajectories and can not only illuminate the nature of comorbidity but can provide a better understanding of alcohol use and smoking in general. We explored three general questions, subsequently described.

The first question concerns the extent to which heavy drinking and smoking possess common risk factors, that is, whether alcohol and tobacco use have *common versus unique correlates*. To do this, we compared risk factors for courses that have comparable trajectories of substance use but are discriminated by the specific substance (e.g., courses with relatively chronic high drinking and persistent low smoking vs. relatively persistent low drinking and chronic high smoking). For example, we might expect alcohol expectancies to be substance-specific and predict only drinking, or alcohol expectancies may reflect a general expectation about the effects of all substances and would predict smoking to the same degree as drinking.

Our second question concerns whether variables associated with comorbid classes are similar to or different from classes characterized by use of a single substance. To address this question, we examined the extent to which predictors of comorbid trajectories are similar or distinct from those of single-substance trajectories. Several patterns of association are possible that are associated with additive effects, synergistic effects, or comorbid-specific correlates. This type of analysis permits the discovery of “masked effects” attributable to confounding. For example, an effect for smoking may exist by virtue of smoking’s relation with drinking, or vice versa. To test this comparison, we compared the prediction of comorbidity with the prediction of single substance courses.

Our third question concerns the extent to which it is possible to distinguish among comorbid classes characterized by similar trajectories of the same substance but by different trajectories of the second substance. Our approach was to examine correlates of temporal patterns of comorbidity with constant levels of a target substance. Controlling for the comorbid substance permits examination of different levels of a given substance and provides better resolution of risk factors for a given substance. For example, if two trajectories have similar smoking course but are differentiated by drinking course, we can compare these trajectories to examine risk factors for drinking. Given the high comorbidity between drinking and smoking, this is a much more illustrative approach than simply comparing risk factors for univariate drinking courses and univariate smoking courses.

The Current Study

The goal of the current study was to describe the concurrent course of heavy alcohol use and tobacco use during early adulthood (ages 19–26), the time of transition from the high school environment to college, military service, and/or part- or full-time occupation. We used a longitudinal developmental framework and validated these courses using available etiologically relevant predictors. We examined heavy alcohol use rather than alcohol quantity or frequency because heavy drinking increases risk for onset (or continuation) of alcohol problems and alcohol use disorders (Wechsler & Austin, 1998) and is common in this developmental period of life (Bachman et al., 1997; Wechsler, Lee, Kuo, & Lee, 2000). A full

41% of college students and 34–42% of those aged 21 to 26 binge drank at least once in the past 2 weeks (Johnston et al., 2004). We chose smoking quantity because “regular smoking” is inconsistently defined (White et al., 2002) and because smoking frequency data were not available in this sample.

We used a mixture modeling procedure (Jones, Nagin, & Roeder, 2001; Muthén, 2001; Muthén et al., 2002; Muthén & Muthén, 2000; Nagin, 1999; for applications of this technique in the substance use area, see Colder et al., 2001; 2002; Li, Barrera, Hops, & Fisher, 2002). General growth mixture modeling is a form of latent growth modeling, but with the addition of an unobserved categorical variable that models variability via discrete homogeneous classes of individuals (rather than via a parameter measuring variability around the latent growth factors). This technique has some important advantages over other techniques used to derive developmental courses of substance use (e.g., cluster analysis), because it treats group membership as a latent (error-free) variable, and accounts for the temporal ordering of prospective data. In the current study, we examined alcohol-tobacco comorbidity by using a dual-trajectory model, in which we explicitly modeled comorbidity. Prior to deriving these trajectories of comorbidity, however, we first examined alcohol and tobacco use individually, in line with prior research, and we examined the association between the two substances. This allowed us to compare the two approaches to simultaneously studying developmental course and comorbidity of different forms of substance use. Finally, we examined the extent to which the courses of drinking, smoking, and comorbidity were associated with six etiologically relevant risk factors: sex, race, alcohol expectancies, delinquency, religiosity (reflecting, to some extent, conventionalism), and parent education.

Panel data were drawn from the MTF study, a large national dataset ($N = 32,087$) that allows fairly broad generalizability to young adults in the United States. In addition, the data enabled us to avoid potential confounds between developmental change and secular change, because MTF is a multiple-cohort study collected over a long historical period, with cohorts beginning in 1976 (and ongoing today). Previously, Schulenberg, O’Malley, et al. (1996) and Schulenberg, Wadsworth, et al. (1996) identified trajectories of heavy drinking with MTF panel data using conceptual groupings and cluster analysis;² the current study extends this work by focusing on the course of comorbid heavy drinking and smoking, using mixture modeling which models growth as a process.

Method

Respondents and Procedure

The MTF project (e.g., Bachman et al., 1997; Johnston et al., 2004), funded by the National Institute on Drug Abuse (NIDA), is an ongoing national study of adolescents and young adults, particularly focusing on substance use. Beginning in 1975, approximately 17,000 twelfth-grade students have completed self-administered questionnaires each year in their classrooms (national samples of 8th and 10th graders were added in 1991). A multistage random sampling procedure is used, in which particular geographic areas were selected, followed by the selection (with probability proportionate to size) of schools in each area. In the third stage, classes within each school were randomly selected, within which up to 350 students were selected. Beginning with the class of 1976, approximately 2,400 respondents were randomly selected for biennial follow-up from each cohort through mail surveys, with about half being surveyed 1 year later and the other half being surveyed 2 years later (and each half followed biennially thereafter). Respondents who reported heavy drug use at baseline were oversampled for follow-up.³ Panel

²A number of our heavy drinking classes are consistent with those found in Schulenberg, O’Malley, et al. (1996) and Schulenberg, Wadsworth, et al. (1996), including the nonheavy drinking class, the late-onset (increase) class, the developmentally limited (decrease) class, and the chronic class.

data are based on the follow-up data for senior-year cohorts 1976–97: Waves 2 to 5 (henceforth termed Times 1–4). Respondents were, on average, 18 years old at Wave 1, 19 to 20 years old at Wave 2, 21 to 22 years old at Wave 3, 23 to 24 years old at Wave 4, and 25 to 26 years old at Wave 5. However, there was variability around these ages. Given the current study's focus on developmental trajectories, we sought to retain homogeneity in age (age at Time 1 ranged from 17 to 23 years, resulting in greater age range within a year than between years). Therefore, we restricted the sample to the modal ages (subsequently described in greater detail)—that is, those who were 18 to 20 years old at Time 1 ($N = 32,087$; $M = 19.31$; 44% male; 82% Caucasian).

Retention rates for any one follow-up survey averaged 75% to 80%. Previous attrition analyses with similar MTF panel samples have shown that, compared with those excluded, those retained in the longitudinal sample were more likely to be female, White, higher on high school GPA and parental education level, and lower on high school truancy and senior year substance use (e.g., Schulenberg, O'Malley, Bachman, & Johnston, 2000; Schulenberg, O'Malley, et al., 1996, Schulenberg, Wadsworth, et al., 1996:). Fortunately, relatively new missing data techniques in Mplus have removed the necessity to restrict the sample to respondents present at all waves. This technique, which assumes that data are missing at random (MAR), estimates the model using full information maximum likelihood (FIML).

Measures

Substance use measures for these analyses included heavy (binge) drinking and current tobacco use. The MFT substance use items have been used for decades in both the project's surveys and by other researchers. They have been shown to demonstrate excellent psychometric properties, and their reliability and validity have been reported and discussed extensively (e.g., Johnston & O'Malley, 1985; O'Malley, Bachman, & Johnston, 1983). Although alcohol and tobacco use as well as sex, race, age, parent education, and religion were assessed on all participants, some psychosocial scales (alcohol expectancies and delinquency) were systematically given to random subsamples of the full respondent sample; analyses using these variables reflect this reduced sample size.

Heavy alcohol use—A single ordinal item assessed frequency of “binge” drinking (operationalized as five or more drinks in a row) in the past 2 weeks. Item responses included 1 (*never drink*), 2 (*once*), 3 (*twice*), 4 (*3–5 times*), 5 (*6–9 times*), and 6 (*10 or more times*); (Time 1 $M = 1.96$).

Tobacco use—A single ordinal item assessing the quantity of cigarettes smoked per day in the past 30 days was assessed. Item response categories included 1 (*not at all*), 2 (*less than one cigarette per day*), 3 (*one to five cigarettes per day*), 4 (*about one half pack per day*), 5 (*about one pack per day*), 6 (*about one and one half packs per day*), and 7 (*two packs or more per day*); Time 1 $M = 1.94$.⁴

Background variables—Age, sex (recoded 1 = male, 0 = female), and race were assessed at baseline. We coded race broadly into five categories: White, Black, Hispanic, Asian, and

³To account for this selective probability of retention, we reestimated our primary analyses (i.e., the mixture models for heavy drinking, smoking, and drinking/smoking) with a weight statement, down-weighting the heavy drug users. The pattern of trajectories was virtually identical, but the weighted results showed more individuals in the nondrinker–nonsmoker categories (i.e., 68% vs. 64% for nonheavy drinkers; 74% vs. 69% for nonsmokers; 62% vs. 57% for nonheavy drinkers–nonsmokers), which is consistent with the oversampling of the heavy drug users.

⁴These values differ slightly from other work using MTF panel data (e.g., Bachman et al., 1996; Schulenberg et al., 2000; Schulenberg, O'Malley, et al., 1996) because of differences in sample definition and cohorts involved.

Other (including Native American and other ethnic minorities) and created four dummy codes with White as the reference group.

Alcohol expectancies were assessed using 15 items, including items assessing drinking to get drunk (similar to Wechsler & Isaac, 1992), drinking to cope (similar to Jessor & Jessor, 1977), and drinking for tension reduction and social facilitation (Goldman, Brown, & Christiansen, 1987; Goldman, Del Boca, & Darkes, 1999; $\alpha = .58$). The binary items included “to relax or relieve tension,” “to feel good or get high,” and “because it tastes good.” We did not have a measure of smoking expectancies. Past-year delinquency was the mean of scores ranging from 1 (*not at all*) to 5 (*5 or more times*) for 15 items, including such items as “got in a serious fight in school or at work” and “been arrested and taken to a police station.” Internal consistency was good ($\alpha = .79$). Religiosity (a proxy for conventionalism) was assessed with 2 items: “importance of religion” and “attendance at religious services” (interitem $r = .62$). Ratings for “importance of religion” ranged from 1 (*not important*) to 4 (*very important*), and ratings for “attendance at religious services” ranged from 1 (*never*) to 4 (*about once a week or more*). Last, parent education was computed by taking the mean of ratings for maternal and paternal education (interitem $r = .55$), which ranged from 1 (*completed grade school or less*) to 6 (*graduate or professional school after college*).

Analytic Procedure

We used general growth mixture modeling (GGMM), using Mplus 3.01 (Muthén & Muthén, 1998–2004). GGMM is based on a latent growth model (LGM) context. Like LGM, growth is represented by latent growth factors (usually an intercept and one or more slope factors). However, in GGMM, homogeneous clusters (or “mixtures”) of individual trajectories are identified and are represented by a categorical latent variable. The extent to which LGM parameters differ across mixtures or classes is modeled. In addition, class prevalence is given, and each participant receives a probability of class membership for each class, ranging from 0 to 1.0. Finally, the influence of external predictors can be explored in a latent variable context in Mplus, by using a multinomial logistic regression procedure. Note that although drinking and smoking are ordinal in nature, they are approximated as continuous variables and thus are appropriate for the GGMM technique.

We identified classes based on the mean of the growth factors alone (i.e., we did not allow the growth factor variances to differ across classes) because freeing the variances across classes typically resulted in model nonconvergence. Other applications of GGMM have also distinguished classes based on growth factor means only (e.g., Colder et al., 2002; Tucker et al., 2003). Although Li et al. (2002) were able to model growth factor variances across class, their model was limited to two classes, which is a relatively simple analytic model. We allowed variances to be nonzero and constrained to be equal to each other, which still allowed us to consider minor variations within class (rather than assuming more “pure” classes and setting variances to zero). We used the (accelerated) expectation maximization (EM) algorithm and ROBUST maximum likelihood estimation, which gives (full-information) maximum likelihood parameter estimates and robust SEs (Muthén & Muthén, 1998–2004).

Results

First, we examine our two outcome variables, frequency of heavy drinking and smoking quantity, and we discuss the effects of birth cohort. Following, we briefly present the results of the mixture models for both heavy alcohol use and smoking, and we examine comorbidity between the two. Then, we present the mixture model for comorbidity. Finally, we explore prediction of alcohol–tobacco comorbidity courses by six etiologically relevant variables is explored.

Preliminary Analyses

On average, heavy drinking frequency slightly decreased over the course of the study when respondents were between ages 18 and 26: Time 1 $M = 1.96$ ($SD = 1.34$); Time 2 $M = 1.98$ ($SD = 1.32$); Time 3 $M = 1.83$ ($SD = 1.24$); Time 4 $M = 1.71$ ($SD = 1.17$).⁵ Although average growth was negative, the standard deviations suggest that individuals did not have the same pattern of growth, and graphs of heavy drinking (not shown) indicated great heterogeneity in the data. Average smoking quantity did not change over the course of the study, with substantial variability in smoking scores; Time 1 $M = 1.97$ ($SD = 1.52$); Time 2 $M = 1.99$ ($SD = 1.57$); Time 3 $M = 1.97$ ($SD = 1.59$); Time 4 $M = 1.93$ ($SD = 1.59$).

Cohort Effects

Prior to discussion of our models, we briefly discuss the nature of our sample. Data were collected using a multicohort design (see Table 1); within each cohort, there was significant age heterogeneity (in part because of the process of collecting follow-up data at biennial intervals). As a consequence, respondents were born in years ranging from 1955 through 1978, creating potential birth cohort effects. Using Cohort $\times$ Time repeated measures analyses of variance, we examined the effect of cohort on heavy drinking and smoking. Cohort had a significant linear effect on heavy drinking, $F(1, 19103) = 28.62$, $p < .001$, $\eta^2 = .002$, but no quadratic or cubic effect (see Figure 1, top panel). Likewise, cohort had a significant linear effect on smoking $F(1, 19276) = 85.95$, $p < .001$, $\eta^2 = .005$, and a significant quadratic effect, $F(1, 19276) = 33.70$, $p < .001$, $\eta^2 = .001$, but no cubic effect (see Figure 1, bottom panel).

In addition, Figure 1 shows that, consistent with preliminary analyses, there was a decrease in heavy drinking but not smoking as respondents age. Given the clear linear effect for cohort (birth year, ranging from 1957 to 1976) on both heavy drinking and smoking, we modeled cohort in all analyses. As discussed earlier, we restricted our sample to those who were aged 18 to 20 (66% of the sample, $N = 32,087$) at Time 1 to remove the age heterogeneity, and we controlled for birth cohort (i.e., birth year) by treating it as an exogenous variable predicting Times 1–4 drinking and smoking. In all subsequent models, the cohort effect is significant for all time points for both drinking and smoking models.⁶

Mixture Modeling: Extracting Trajectories

We based the general growth mixture models on a basic latent growth model with an intercept and linear and quadratic slopes.⁷ Model fit was evaluated using information criteria fit indices (Bayesian information criterion, BIC; Schwartz, 1978; and Akaike's information criterion, AIC; Akaike, 1987), as well as using the Vuong–Lo–Mendell–Rubin likelihood ratio test for

⁵To make the trajectories more interpretable with respect to drinking unit, heavy drinking was recoded to range from 0 to 10 episodes of binge drinking in the past 2 weeks by taking the midpoint of an item (e.g., 3–5 times in the past 2 weeks would be recoded to 4; $M = 1.31$, $M = 1.31$, $M = 1.10$, $M = 0.93$ for Times 1–4, respectively). Analogous to the binge-drinking item, smoking quantity was recoded to range from 0 to 2 packs per day, or 40 cigarettes, ($M = 3.71$, $M = 3.98$, $M = 4.01$, $M = 3.95$ for Times 1–4, respectively). However, the distribution of these variables were much more skewed than the original variable and resulted in poorer model convergence (skew ranged from 2.03 to 2.63 for heavy drinking and from 2.12 to 2.23 for smoking; kurtosis ranged from 4.07 to 7.67 for heavy drinking and from 3.65 to 4.32 for smoking). Transformations such as taking the logarithm (Neter, Wasserman, & Kutner, 1990) did not remedy the problem sufficiently. Hence, we retained the original variables (skew ranged from 1.12 to 1.57 for heavy drinking and from 1.34 to 1.47 for smoking; kurtosis ranged from 0.08 to 1.53 for heavy drinking and from 0.37 to 0.72 for smoking).

⁶We further probed cohort effects by examining them at the level of the intercept and slope factors. The only significant cohort effects were on intercept; that is, cohort did not predict linear or quadratic slope for either heavy drinking or smoking. This analysis assumes that all of the cohort effect was transferred through the intercept (i.e., Time 1), which we believe is less tenable than cohort having an effect through Times 1–4 heavy alcohol use and Times 1–4 smoking. To further examine time trends, we explored whether cohort had a nonlinear effect on these assessments. We created quadratic (cohort2) and cubic (cohort3) variables and tested the extent to which these variables predicted Times 1–4 heavy alcohol use and Times 1–4 smoking. None of the cubic trends were significant and the quadratic trend was significant for tobacco use only (standardized $\beta = .03$). Given our goal of parsimony, as well as identification problems in including the quadratic and cubic cohort variables (given limited degrees of freedom, inclusion of these variables as well as the linear cohort variable necessitated that their values be constrained to be equal across Times 1–4), we freely estimated the parameters between cohort (linear trend only) and each of the manifest variables.

k versus $k - 1$ classes (Lo, Mendel, & Rubin, 2001; Muthén et al., 2002), which is significant if k classes show improvement over $k - 1$ classes. In addition, given the large sample size (which affects values of AIC and BIC), we considered three other criteria: class prevalence (we tended not to consider classes that included less than 5% of the sample as they were unlikely to be replicable), class interpretability (the extent to which an additional class provided unique information) and stability (the extent to which the nature and prevalence of the classes changed when demographic variables were controlled) See Colder et al. (2002) for a more extended explanation of these criteria. We noted significant variability around the mean for the intercept and slope factors, suggesting individual differences and the likelihood of distinct classes of heavy drinkers and smokers over the observation period.

Prior to fitting the dual trajectory model for alcohol and tobacco use, we estimated separate (single-domain) models for alcohol and tobacco use. We identified four trajectories of frequency of heavy drinking, including nonheavy drinkers (including nondrinkers) (64%), chronic heavy drinkers (12%), developmentally limited (decrease) heavy drinkers (16%), and late-onset (increase) heavy drinkers (8%), and five trajectories of smoking quantity, including nonsmokers (69%), chronic smokers (12%), late-onset (increase) smokers (6%), developmentally limited (decrease) smokers (6%), and moderate smokers (7%). A cross-tabulation of group membership for heavy drinking by smoking revealed that heavy drinking and smoking were associated, $\chi^2(12, N = 31,853) = 2,449.78, p < .001; \Phi = .28$; Cramér's $V = .16$.⁸ A first-order configural frequency analysis technique (von Eye, 2002)⁹ that tested observed versus expected cell frequencies revealed that, although there were 20 (4×5) different potential trajectories of smoking and drinking, some of these particular combinations of smoking and drinking were less likely to occur than chance (antitypes) (e.g., nonheavy drinkers with smoking classes; nonsmokers with drinking classes), and correspondingly, some combinations were more expected to occur than chance (types; e.g., cells along the diagonal; chronic heavy drinkers with chronic high smokers and moderate smokers; chronic smokers with developmentally limited drinkers). On the basis of these findings, we examined prospective comorbidity by modeling both substances simultaneously to determine which of these conjoint types were most clearly represented in our sample, expecting that some of these "types" would have increased likelihood of being identified as a conjoint trajectory.

Identification of Trajectories

We tested two- through eight-group solutions (the nine-group model would not converge on a solution; see Table 2). According to BIC and AIC, we observed significant improvements in model fit up to eight classes, although according to the Vuong–Lo–Mendell–Rubin likelihood ratio test, the six-class model best fit the data. However, the seven-class model contained the moderate–moderate class (6% of the sample) that we believed added additional information. In addition, based on stability of the models in the presence of different exogenous covariates, class prevalences, and interpretability, the seven-class model appeared to be the best model. As such, we chose the seven-class model; Figure 2 presents mean growth from Times 1–4 in frequency of heavy alcohol use and smoking quantity by class, weighted by estimated class probabilities. Classes were as follows: a nonheavy drinking–nonsmoking class (56%), a chronic heavy drinking and chronic heavy smoking class (6%), a low drinking but heavy

⁷The intercept was centered at Time 1 which corresponds to modal ages 19–20. Based on our previous work looking at (negative) growth in substance use (Parra, Sher, Krull, & Jackson, 2003), we modeled the negative relation between the intercept and the slope factors as a directional relation, rather than as a covariance, in order to address the phenomenon that when modeling negative growth, the higher an individual is at Time 1, the greater he or she falls over time (suggesting perhaps a floor effect for those low at Time 1).

⁸For this analysis, we determined group membership by assigning an individual to the class to which he or she was most likely to belong. We also examined comorbidity using weighted estimates (weighted by probability of group membership in both groups). As might be expected by high entropy in our models, weighted estimates and corresponding tests of association were very similar to those using unweighted estimates.

⁹We used Lehman's approximation to the binomial probability (with Küchenhof's correction for continuity, cf. von Eye, 2002).

smoking class (8%), a heavy drinking but low smoking (perhaps chippers; Shiffman, 1989) class (14%), a moderate-drinker, late-onset heavy smoking class (5%), a moderate-drinker, developmentally limited heavy smoking class (5%), and a moderate drinking–smoking class (6%). There appears to be more variability in smoking—moderate drinking is accompanied by three types of smokers (moderate, late-onset, and developmentally limited smokers). Yet, there were four heavy smoking groups—two developmentally graded, two chronic; the chronic smoking classes were distinguishable by drinking (low heavy drinking vs. heavy drinking).

Prediction of Trajectory Group Membership

Next, also within the Mplus framework, we examined the extent to which the trajectory groups differed on several etiologically relevant predictors taken from baseline (Time 1). Zero-order correlations between drinking, smoking, and our six predictors (sex, race, alcohol expectancies, delinquency, religiosity, and parent education) are shown in Table 3. Table 4 presents means and proportions on the predictors for each of the trajectory groups. To test group differences, we conducted a series of multinomial logistic regressions. Predictors, which were tested univariately because of the differing number of participants and nonoverlapping samples (discussed previously) for each, were modeled as exogenous to the class membership variable in the context of the general growth mixture model. Prior to analysis, these variables were standardized to increase interpretability of coefficients (odds ratios; OR). Note that these coefficients are not (derived from) partial regression coefficients, as would be obtained in a multivariate regression procedure. Means and proportions with the same subscript (within a row) in Table 4 were not significantly different using a pairwise odds ratio (drawing from a method used by Tucker et al., 2003), according to the multinomial logistic regressions (changing the reference group accordingly). Although there exists a large number of possible pairwise contrasts, we were specifically interested in three sets of contrasts based on our research questions using a priori comparisons. To illustrate these contrasts, we focused primarily on the courses that show chronic drinking and/or smoking, because these are highly clinically relevant and have less ambiguity than some of the other drinking and smoking courses, although we did consider additional courses for our third question. To reduce possibility of Type I error, especially given our large sample size, we applied a Bonferroni correction and reported tests that were significant at $p < .001$ ($\alpha = .05/48$, the total number of tests; $8 \text{ comparisons} \times 6 \text{ variables} = 48$).

For our first set of contrasts, *common versus unique correlates*, we compared trajectories that had similar course (level and slope) of a given substance but were discriminated by the specific substance. That is, to what extent is prediction of a given substance due to that particular substance versus to the course of substance use in general? More specifically, we tested a set of three comparisons. In our first two comparisons, we examined prediction of single chronic substances (chronic high drinking–low smoking and low drinking–chronic high smoking) to the nonusing reference group (nondrinking–nonsmoking) to examine whether risk factors differ for prediction of chronic alcohol use than for the prediction of chronic tobacco use. If correlates were *common*, we would expect to find similar results for the two comparisons. If correlates were *unique*, we would observe some differential prediction. Analyses revealed that relative to the nondrinker–nonsmoker group, higher alcohol expectancies (OR = 1.89),¹⁰ higher delinquency (OR = 2.54), lower religiosity (OR = 0.62), higher parent education (OR = 1.22), being male (OR = 3.70), and not being Black (OR = 0.18), Asian (OR = 0.22), Hispanic (OR = 0.58), or Other ethnicity (OR = 0.67) increased the odds of being in the chronic drinker–low smoker group, and higher alcohol expectancies (OR = 1.62), higher delinquency (OR =

¹⁰Despite multiple start values, the models that included alcohol expectancies as a predictor failed to converge. The estimates shown herein are for models with the latent class part of the model constrained to equal the values in the full model (with no exogenous risk factors). Note that when delinquency was modeled this way, estimates were extremely similar to those from the fully estimated model (presented herein), with no substantive differences.

1.73), lower religiosity (OR = 0.59), lower parent education (OR = 0.74), being Other ethnicity (OR = 1.53) and not being Black (OR = 0.24), Asian (OR = 0.06), or Hispanic (OR = 0.17) significantly increased the odds of being in the low drinker–chronic smoker group. The different direction of effect for parent education between the two comparisons as well as the similar risk factors (alcohol expectancies, delinquency, religiosity, not being Asian or Hispanic) for the two suggests that there are some general similarities as well as some specific differences in prediction of the two substances.

Although the greater magnitude of some of the effects in one substance versus the other (i.e., alcohol expectancies, delinquency) in the above comparisons suggests differential prediction of the two substances, it does not explicitly test this issue. This led us to make a third comparison, in which we compared the chronic high heavy drinking–low smoking trajectory with the low drinking–chronic high smoking trajectory. Findings revealed that higher delinquency (OR = 1.47), higher parent education, (OR = 1.65), being male (OR = 4.34), not being in the Other ethnic group (OR = 0.53), and being Hispanic (OR = 3.42) significantly increased the odds of being in the chronic high drinker–low smoker group relative to the low drinker–chronic high smoker group, indicating that delinquency, parent education, being male, and being Hispanic or Caucasian are associated with chronic high drinking more so than chronic high smoking. This provides further support that the risk factors are more unique than they are common.

In sum, this comparison revealed that, despite most predictors' being associated with both drinking and smoking (see Table 3), trajectory analyses suggested that most predictors were differentially related to the use of one substance versus the other. Higher delinquency, higher parent education, being male, being Hispanic, and not being in the Black or Other ethnic groups were associated with greater drinking and less smoking. Alcohol expectancies had limited support as a unique risk factor, because they were more highly associated with chronic high drinking than chronic high smoking but the explicit comparison failed to reach significance. Religiosity, however, did not differentiate between trajectories that were characterized by similar course but different substances, suggesting that this factor is more common than unique.

In our second set of contrasts, *comorbidity versus single substance correlates*, we performed three contrasts. In the first, we predicted the comorbidity trajectory (chronic drinking–chronic smoking) by using the nondrinker–nonsmoker group as reference group. Higher expectancies (OR = 2.20); higher delinquency (OR = 2.64); lower religiosity (OR = 0.49); being male (OR = 2.35); and not being Black (OR = 0.16), Hispanic (OR = 0.20), or Asian (OR = 0.24) significantly increased the odds of being in the chronic drinker–chronic smoker group relative to the nondrinker–nonsmoker group. For alcohol expectancies, delinquency, religiosity, and being Black, this was a similar pattern of findings as the single-substance comparisons but with parameters generally greater in magnitude (although the extent to which this is true cannot be explicitly tested), which suggests that their prediction of comorbidity is additive or perhaps even synergistic. Next, we examined alcohol use with versus without comorbid tobacco use and we examined tobacco use with versus without comorbid alcohol use. Specifically, we examined prediction of the comorbid course (chronic drinking–chronic smoking) to the single chronic substance courses (chronic drinking–low smoking and low drinking–chronic smoking). Analyses revealed that lower religiosity (OR = 0.79), lower parent education (OR = 0.76), being female (OR = 0.64), not being Hispanic (OR = 0.34), and being in the Other ethnic group (OR = 1.89) increased the odds of being in the chronic drinker–chronic smoker group relative to the chronic drinker–low smoker group, and higher alcohol expectancies (OR = 1.36), higher delinquency (OR = 1.53), lower religiosity (OR = 0.83), higher parent education (OR = 1.25), and being male (OR = 2.76) significantly increased the odds of being in the chronic drinker–chronic smoker group relative to the low drinker–chronic smoker group.

In sum, some risk factors predicted comorbidity above and beyond a single substance, suggesting perhaps an additive effect. Certain risk factors predicted comorbidity relative to the chronic drinking course (low parent education, being female, not being Hispanic, and being in the Other ethnic group), suggesting that drinking with smoking is different from drinking alone. Still others predicted comorbidity relative to the chronic smoking course (alcohol expectancies, delinquency, lower religiosity, high parent education, and being male), suggesting that smoking with drinking is different from smoking alone.

Finally, in our third contrast, correlates of patterns of comorbidity with constant levels of a single substance, we explored prediction of a given substance while controlling for the other. Although our empirical trajectories did not allow for a comparison of smoking course while holding drinking constant, we were able to examine divergent alcohol trajectories while holding smoking constant. Specifically, we compared the moderate drinking–moderate smoking group with two groups: moderate drinking–late onset (increase) smoking and moderate drinking–developmentally limited (decrease) smoking. Findings revealed that being female ($OR = 0.40$) and being Black ($OR = 1.61$) or Hispanic ($OR = 4.00$) significantly increased the odds of being in the moderate drinker–moderate smoker group relative to the moderate drinker–late onset smoker group; and having high religiosity ($OR = 1.20$) and being Black ($OR = 4.17$) significantly increased the odds of being in the moderate drinker–moderate smoker group relative to the moderate drinker–developmentally limited smoker group. In sum, being female, being of high religiosity, and being Black or Hispanic increased risk for stable moderate smoking, as opposed to time-delimited smoking that remits after early young adulthood (ages 18–22) or escalates during late young adulthood (ages 22–26).

Discussion

Substance use tends to peak during the transition to adulthood. Yet, this normative trend does not apply to all, or even most, young people. Especially during this transition time, when diversity in life paths increases and changes in contexts are often pervasive and simultaneous, it is essential to identify distinct courses of substance use (Schulenberg & Maggs, 2002). In turn, these distinct courses can assist in advances in the understanding of the causes, correlates, and consequences of substance use during the transition to adulthood. Our findings have implications regarding the etiology of substance use and of psychopathology in general.

To characterize the nature of comorbidity over the course of development, we took the approach of modeling conjoint trajectories using nationally representative, prospective, multiwave data.¹¹ We identified seven co-occurring trajectories of alcohol and tobacco use, controlling for secular changes occurring over 2 decades. In addition, we examined the extent to which available covariates (specifically, sex, race, alcohol expectancies, delinquency, religiosity, and parent education) predicted course of alcohol–tobacco comorbidity.

Implications for Studying Comorbidity

The present study demonstrates both the importance and empirical feasibility of considering both developmental course and comorbidity in the characterization of alcohol–tobacco comorbidity during early young adulthood. Our findings extend to problems of clinical concern, and our techniques generalize to (multiple) psychiatric disorders in general. Although

¹¹The dilemma in modeling prospective comorbidity of two substances is whether to model the univariate course of each substance separately and to then examine the association between the two, or whether to examine multiple courses of comorbidity itself. Although both approaches have intuitive appeal to the study of course of comorbidity, the dual trajectory approach is a more parsimonious, pragmatic approach, especially when it comes to exploring the relation of comorbidity to etiological predictors of interest. If external predictors were explored in the context of the single-domain trajectories, it would be necessary to examine 20 combinations of drinking and smoking, rather than simply seven, and perhaps reify dual substance use trajectories that are unlikely to exist.

the explicit diagnostic criteria sets introduced in the third edition of the Diagnostic and Statistical Manual of Mental Disorders and subsequent revisions (e.g., American Psychiatric Association, 1980, 1987, 1994) signify a major leap forward in psychiatric phenotype definition by rejuvenating the Kraepelinian approach to diagnosis, they represent only a partial embrace of a Kraepelinian approach that equally emphasized syndrome description by using specific behavioral indicators and longitudinal course (Widiger & Clark, 2000). To a large extent, formal diagnostic nosology has not kept up with either theory or data that highlight the importance of considering both longitudinal course and co-occurring comorbidity as critical phenotypes. Our approach could be applied to the study of any set of problem behaviors that exhibits a developmental time course and tends to be comorbid with other co-occurring conditions or symptoms. Additionally, by extension, more than two disorders or behaviors theoretically could be studied using this approach (e.g., alcohol, depression, and marital discord), although at the present time, practical constraints (especially sample size and number of longitudinal measurement occasions) limit the number of domains that can be modeled simultaneously.

Alcohol–Tobacco Comorbidity

This is the first study to explicitly identify such trajectories of concurrent drinking and smoking. Although the majority of respondents tended to be nonheavy drinker–nonsmoker, nearly half were either moderate-to-high chronic drinkers or smokers, or some combination thereof. Identification of common drinking and smoking groups might provide information for targeted prevention or treatment initiatives. For example, a full two fifths of individuals who smoke chronically also binge drink chronically, suggesting that chronic smoking could be an index for other addictive syndromes. In addition, individuals who binge drank moderately tended to belong to one of three smoking groups: moderate, late-onset, or developmentally limited. Although research has long since established that heavy drinkers tend to smoke, it is relatively silent about the extent to which moderate drinkers smoke, other than noting a dose-dependent association between drinking and smoking (Madden, Bucholz, Martin, & Heath, 2000). Although the present work suggests that moderate drinking could be an antecedent (perhaps even a cause), a consequence, or simply a cooccurring condition with smoking, the present study is limited in its ability to resolve this issue. Rather than using more variable-centered approaches such as cross-lagged panel models, multivariate latent growth curve models, or state-trait models (Sher & Wood, 1997), which resolve the extent to which comorbidity is attributable to uni- or bidirectional relations between alcohol and tobacco involvement versus a function of common third variables, we selected our approach to explore the developmental courses of co-occurring drinking and smoking and to identify correlates of these courses. This approach of modeling “developmental comorbidity” is in some ways a more fundamental portrayal of comorbidity than variable-centered alternatives which fail to resolve observable comorbid “types” and provide prevalence estimates for these. We do note that prior work suggests reciprocal causation between alcohol use disorders and tobacco dependence (Sher et al., 1996), suggesting that both directions of influence occur, although common third-variable influences are also likely (Jackson et al., 2000b). However, such “third variables” would need to be differentially expressed as a function of development in order to explain the range of comorbid types revealed by the current set of analyses.

Our model also revealed the extent to which comorbidity changed over time. For example, the most chronic group reported less heavy drinking over time, tracking the decline in drinking following adolescence (Johnston et al., 2004; Muthén & Muthén, 2000), presumably due to the adoption of a more conventional lifestyle (Bachman et al., 1997; Fillmore, 1988; Jessor, Donovan, & Costa, 1991). Despite considerable cross-sectional comorbidity, patterns of use can diverge over time and there is some degree of functional independence, in a developmental sense, of tobacco and alcohol use, at least in a subset of the population.

Prediction of Courses of Alcohol–Tobacco Comorbidity

Although our data set was somewhat limited in its assessment of etiologically relevant covariables, consideration of patterns of prediction is nonetheless informative. There is some evidence that parent education, gender, and race were unique risk factors that may have exhibited an “additive” effect in associations with cooccurring drinking and smoking (by virtue of larger estimates for comorbid vs. single substance comparisons). Also consistent with a unique, additive effect is that prediction of comorbidity (a) showed low parent education and being female to be associated with drinking only when accompanied by smoking and (b) showed high parent education and being male to be associated with smoking only when accompanied by drinking. Consistent with findings for chronic smoking, being female, being of high religiosity, and being Black or Hispanic were associated with increased risk of stable moderate smoking, whereas being male, being of low religiosity, and being White were associated with time-delimited courses of smoking (drinking held constant). This suggests that these risk factors are also specific to *course* within a substance.

Low religiosity, reflecting to some extent low conservatism, appeared to be a relatively *common* risk factor for substance use that exhibited an additive effect when considering alcohol–tobacco comorbidity. It was negatively associated with both drinking and smoking, but it did not differentiate between drinking and smoking trajectories with similar course, suggesting that it was more common than unique. In addition, it predicted a course of comorbidity above and beyond a single substance course, suggesting that it has an additive effect. In sum, sex, race, parent education, and religiosity tended to be additive in nature, suggesting that comorbidity may simply be sign of severity for this particular set of predictors.

Perhaps of greatest interest, although alcohol expectancies and delinquency seemed to be relatively unique risk factors, these risk factors actually had a “masked” effect whereby their association with smoking could be attributed to a relation with drinking via smoking’s association with drinking—that is, when we controlled for drinking, smoking no longer showed an effect; its effect existed only by virtue of its comorbidity with drinking. The opposite was not true: When we controlled for smoking, alcohol expectancies and delinquency still predicted drinking. Specifically, alcohol expectancies and delinquency were each positively bivariately associated with drinking and smoking, as well as with drinking trajectories and smoking trajectories; however, when we examined drinking and smoking in a comorbidity framework, these risk factors were associated with smoking only if there was comorbid drinking.

Given (a) that previous research has shown similar expectancies across substances (Stacy, Galaif, Sussman, & Dent, 1996) and similarity between motivations for drinking and motivations for smoking (Johnson & Jennison, 1992), and (b) that work in our own laboratory (using different data) has shown a robust relation between alcohol expectancies and tobacco dependence (Jackson et al., 2000b), it might be tempting to conclude that expectancies are relatively common across substance. However, the present findings suggest that any observed association between alcohol expectancies and smoking is most likely due to smoking’s comorbidity with drinking. Likewise, although work has shown that conduct disorder and delinquency predict tobacco use, dependence, or both (Bardone et al., 1998; Bryant, Schulenberg, Bachman, O’Malley, & Johnston, 2000; Windle, 1990), our findings suggest that these latter relations may be due in part to smoking’s association with drinking. Masked effects such as those observed for alcohol expectancies and delinquency permit us to learn new information that cannot be obtained by separately examining predictors of drinking and predictors of smoking. We note that these masked effects are consistent with previous findings in alcohol–tobacco comorbidity (Jackson et al., 2000a) that revealed informative associations in the context of comorbidity that were not apparent from considering the single-domain relations alone. Specifically, Jackson et al. showed childhood stressors to be associated with an increased likelihood of belonging to the comorbid class. However, in the absence of a co-

occurring AUD, childhood stressors were not a risk factor for TD. This conditional effect was not detected from a single-domain approach. Univariate substance-specific (alcohol-only or tobacco-only) approaches may obscure specific relations that can only be observed when explicitly modeling comorbidity, and future research exploring risk factors for a behavior or disorder that is highly comorbid with another behavior or disorder must consider the risk factors in the context of the co-occurring behavior.

Strengths and Limitations

Data for the current study were taken from a large, nationally representative sample with multiple cohorts, allowing us to control for secular effects; in a single-cohort study, it is unclear how generalizable the findings are to other historic periods. However, characteristics of the data set also somewhat limited our study. Our participants were age 18 or older at the first assessment, and we certainly may have missed important developmental changes because much of the onset of substance use (particularly smoking) occurs in early-to-middle adolescence. Retention rates (65%) were acceptable (especially given that data were collected through lowcost mail surveys over a 6-year period), but attrition was somewhat differential with respect to variables important in this analysis. This suggests that our findings reflect a more conservative population in terms of substance use. Furthermore, we did not have syndromal diagnostic data. Although the heavy or “binge” drinking criteria of five or more drinks per occasion (or four or more for women) has been the topic of much debate, the association between binge drinking and alcohol consequences, problems, and dependence is robust, and data suggest that the five-drink measure is indeed a meaningful threshold (Wechsler & Austin, 1998).¹² Also, we believe that the limitations of using consumption measures are offset by their consistent assessment over four waves, and the present study complements previous findings on alcohol–tobacco comorbidity that used structured interviews but were limited with respect to generalizability, sample size, and multiple cohorts (Jackson et al., 2000b). We note that we have found using other data (Jackson & Sher, 2004) moderate agreement between classification of heavy drinking and alternate measures of alcohol consumption, including alcohol use disorders.

We also were limited to examining a set of risk factors that were relatively demographic in nature or were administered to a small random subgroup of participants. Presumably, work in behavioral genetics or prevention–treatment outcome studies can further establish the construct validity of the trajectory groups. However, multiple cohorts over 2 decades, multiple waves with consistent assessment of drinking and smoking, and a large representative sample provide a unique opportunity to characterize joint trajectories of behaviors that are closely related to clinical problems, and these strengths outweigh any of the limitations discussed above.

Although we used a state-of-the-art modeling technique to identify ordered trajectories of substance use, we note that this technique is not without its drawbacks. In mixture modeling analyses, mixtures can be extracted even when none exist, if the data are non-normal but contain only a single population (Bauer & Curran, 2003; but see Muthén, 2003). Although our findings from the single-domain approach were consistent with both theory and extant empirical research on trajectories of drinking and smoking, we still exercise caution in drawing conclusions about comorbidity from these data until the replicability of these comorbid groups is established. Regardless of replicability of the current work, the general approach represents an important step forward in psychiatric epidemiology by demonstrating the feasibility of modeling comorbidity and course within a person-centered approach to data analysis.

¹²We note that in other work in our lab, using a prospective (six-wave) sample of young adults (Sher, Walitzer, Wood, & Brent, 1991), trajectories of heavy drinking showed a moderate degree of overlap (percent agreement ranged from 60% to 69%; kappas ranged from .28 to .38) with trajectories of interview-based alcohol use disorder diagnoses and questionnaire-based measures of alcohol consequences and alcohol dependence (Sher & Jackson, 2003) as well as with alcohol consumption ($\kappa = .50$).

Acknowledgments

Preparation of this paper was supported by National Institute on Alcohol Abuse and Alcoholism Grants R21 AA12383 and K01 AA13938 to Kristina M. Jackson; the Monitoring the Future Project, from which the data were obtained, is funded by the National Institute on Drug Abuse Grant DA01411 to Lloyd D. Johnston.

We thank Tammy Chung, Jennifer Krull, Denis McCarthy, Bengt Muthén, Linda Muthén, Patrick O'Malley, Gilbert Parra, Lance Swenson, and Phillip Wood for their assistance in analyses and helpful comments on previous drafts of this article.

References

- Akaike H. Factor analysis and AIC. *Psychometrika* 1987;52:317–332.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd ed.. Author; Washington, DC: 1980.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd ed.. Author; Washington, DC: 1987. rev.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed.. Author; Washington, DC: 1994.
- Bachman, JG.; O'Malley, PM.; Johnston, LD.; Rodgers, WL.; Schulenberg, J.; Lim, J.; Wadsworth, KN. Changes in drug use during ages 18–32. Institute for Social Research; Ann Arbor, MI: 1996. Monitoring the Future Occasional Paper No. 39
- Bachman, JG.; Wadsworth, KN.; O'Malley, PM.; Johnston, LD.; Schulenberg, JE. Smoking, drinking, and drug use in young adulthood: The impacts of new freedoms and new responsibilities. Erlbaum; Mahwah, NJ: 1997.
- Bardone AM, Moffitt TE, Caspi A, Dickson N, Stanton WR, Silva PA. Adult physical health outcomes of adolescent girls with conduct disorder, depression, and anxiety. *Journal of the American Academy of Child & Adolescent Psychiatry* 1998;37:594–601. [PubMed: 9628079]
- Bates ME. Integrating person-centered and variable-centered approaches in the study of developmental courses and transitions in alcohol use: Introduction to the special section. *Alcoholism: Clinical & Experimental Research* 2000;24:878–881.
- Bauer DJ, Curran PJ. Distributional assumptions of growth mixture models: Implications for over-extraction of latent trajectory classes. *Psychological Methods* 2003;8:338–363. [PubMed: 14596495]
- Bennett ME, McCrady BS, Johnson V, Pandina RJ. Problem drinking from young adulthood to adulthood: Patterns, predictors and outcomes. *Journal of Studies on Alcohol* 1999;60:605–614. [PubMed: 10487729]
- Bien TH, Burge J. Smoking and drinking: A review of the literature. *International Journal of Addiction* 1990;25:1429–1454.
- Blot WJ, McLaughlin JK, Winn DM, Austin DF, Greenberg RS, Preston-Martin S, Bernstein L, Schonenberg JB, Stemhagen A, Fraumeni JF. Smoking and drinking in relation to oral and pharyngeal cancer. *Cancer Research* 1988;48:3282–3287. [PubMed: 3365707]
- Breslau N. Psychiatric comorbidity of smoking and nicotine dependence. *Behavior Genetics* 1995;25:95–101. [PubMed: 7733862]
- Bryant AL, Schulenberg J, Bachman JG, O'Malley PM, Johnston LD. Understanding the links among school misbehavior, academic achievement, and cigarette use: A national panel study of adolescents. *Prevention Science* 2000;1:71–87. [PubMed: 11521961]
- Chassin L, Pitts SC, Prost J. Binge drinking trajectories from adolescence to emerging adulthood in a high-risk sample: Predictors and substance abuse outcomes. *Journal of Consulting and Clinical Psychology* 2002;70:67–78. [PubMed: 11860058]
- Chassin L, Presson CC, Pitts SC, Sherman SJ. The natural history of cigarette smoking from adolescence to adulthood in a Midwestern community sample: Multiple trajectories and their psychosocial correlates. *Health Psychology* 2000;19:223–231. [PubMed: 10868766]
- Chen K, Kandel DB. The natural history of drug use from adolescence to the mid-thirties in a general population sample. *American Journal of Public Health* 1995;85:41–47. [PubMed: 7832260]

- Cicchetti D, Rogosch FA. A developmental psychopathology perspective on adolescence. *Journal of Consulting and Clinical Psychology* 2002;70:6–20. [PubMed: 11860057]
- Colder CR, Campbell RT, Ruel E, Richardson JL, Flay BR. A finite mixture model of growth trajectories of adolescent alcohol use: Predictors and consequences. *Journal of Consulting and Clinical Psychology* 2002;70:976–985. [PubMed: 12182281]
- Colder CR, Mehta P, Balanda K, Campbell RT, Mayhew KP, Stanton WR, Pentz MA, Flay BR. Identifying trajectories of adolescent smoking: An application of latent growth mixture modeling. *Health Psychology* 2001;20:127–135. [PubMed: 11315730]
- Department of Health and Human Services. Summary of findings from the 1999 National Household Survey on Drug Abuse. Author; Rockville, MD: 2000.
- DiFranza JR, Guerrera MP. Alcoholism and smoking. *Journal of Studies on Alcohol* 1990;51:130–135. [PubMed: 2308350]
- Fillmore, KM. Alcohol use across the life course. Addiction Research Foundation; Toronto, Canada: 1988.
- Flanders WD, Rothman KJ. Interaction of alcohol and tobacco in laryngeal cancer. *American Journal of Epidemiology* 1982;115:371–379. [PubMed: 7064973]
- Goldman, MS.; Brown, SA.; Christiansen, BA. Expectancy theory: Thinking about drinking. In: Blane, HT.; Leonard, KE., editors. *Psychological theories of drinking and alcoholism*. Guilford Press; New York: 1987. p. 181-266.
- Goldman, MS.; Del Boca, FK.; Darkes, J. Alcohol expectancy theory: The application of cognitive neuroscience. In: Leonard, KE.; Blane, HT., editors. *Psychological theories of drinking and alcoholism*. 2nd ed.. Guilford Press; New York: 1999. p. 203-246.
- Gulliver SB, Rohsenow DJ, Colby SM, Dey AN, Abrams DB, Niaura RS, Monti PM. Interrelationship of smoking and alcohol dependence, use, and urges to use. *Journal of Studies on Alcohol* 1995;56:202–206. [PubMed: 7760567]
- Istvan J, Matarazzo JD. Tobacco, alcohol, and caffeine use: A review of their relationships. *Psychological Bulletin* 1984;95:301–326. [PubMed: 6544436]
- Jackson KM, Sher KJ. Similarities and differences of longitudinal phenotypes across alternate measures of alcohol involvement. 2004 Manuscript submitted for publication.
- Jackson KM, Sher KJ, Wood PK. Trajectories of conjoint substance use disorders: A developmental, typological approach to comorbidity. *Alcoholism: Clinical and Experimental Research* 2000a; 24:902–913.
- Jackson KM, Sher KJ, Wood PK. Prospective analyses of comorbidity: Tobacco and alcohol use disorders. *Journal of Abnormal Psychology* 2000b;109:679–694. [PubMed: 11195992]
- Jessor, R.; Donovan, JE.; Costa, FM. Beyond adolescence: Problem behavior and young adult development. Cambridge University Press; New York, NY: 1991.
- Jessor, R.; Jessor, S. Problem behavior and psychosocial development. Academic Press; New York: 1977.
- Johnson KA, Jennison KM. The drinking–smoking syndrome and social context. *International Journal of the Addictions* 1992;27:749–792. [PubMed: 1618582]
- Johnston, LD.; O'Malley, PM. Issues of validity and population coverage in student surveys of drug use. In: Rouse, BA.; Kozel, NJ.; Richards, LG., editors. *Self-report methods of estimating drug use: Meeting current challenges to validity*. National Institute on Drug Abuse; Washington, DC: 1985. p. 31-54. NIDA Research Monograph No. 57
- Johnston, LD.; O'Malley, PM.; Bachman, JG.; Schulenberg. National survey results on drug use from the Monitoring the Future study, 1975–2003. Volume I: Secondary school students. Volume II: College students and young adults. National Institute on Drug Abuse; Bethesda, MD: 2004.
- Jones BL, Nagin DS, Roeder K. A SAS Procedure Based on Mixture Models for Estimating Developmental Trajectories. *Sociological Methods & Research* 2001;29:374–393.
- Kozlowski LT, Henningfield JE, Keenan RM, Lei H, Leigh G, Jelinek LC, Pope MA, Haertzen CA. Patterns of alcohol, cigarette, and caffeine and other drug use in two drug abusing populations. *Journal of Substance Abuse Treatment* 1993;10:171–179. [PubMed: 8510191]
- Li F, Barrera MJR, Hops H, Fisher KJ. The longitudinal influence of peers on the development of alcohol use in late adolescence: A growth mixture analysis. *Journal of Behavioral Medicine* 2002;25:293–315. [PubMed: 12055779]

- Lo Y, Mendell NR, Rubin D. Testing the number of components in a normal mixture. *Biometrika* 1991;88:767–778.
- Madden PAF, Bucholz KK, Martin NG, Heath AC. Smoking and the genetic contribution to alcohol-dependence risk. *Alcohol Health & Research World* 2000;24:209–214.
- Martin CS, Kaczynski NA, Maisto SA, Tarter RE. Polydrug use in adolescent drinkers with and without *DSM-IV* alcohol abuse and dependence. *Alcoholism: Clinical and Experimental Research* 1996;20:1099–1108.
- Munoz, N.; Day, NE. Esophageal cancer. In: Schoffenfield, D.; Fraumani, JF., Jr, editors. *Cancer epidemiology and prevention*. Oxford University Press; New York: 1996. p. 681-706.
- Muthén, B. Latent variable mixture modeling. In: Marcoulides, GA.; Schumacker, RE., editors. *New Developments and techniques in structural equation modeling*. Erlbaum; Mahwah, NJ: 2001. p. 1-33.
- Muthén BO. Statistical and substantive checking in growth mixture modeling: Comment on Bauer and Curran. *Psychological Methods* 2003;8:369–377. [PubMed: 14596497]
- Muthén B, Brown CH, Masyn K, Jo B, Khoo ST, Yang C-C, Wang C-P, Kellam SG, Carlin JB, Liao J. General growth mixture modeling for randomized preventive interventions. *Biostatistics* 2002;3:459–475. [PubMed: 12933592]
- Muthén B, Muthén LK. Integrating person-centered and variable-centered analyses: Growth mixture modeling with latent trajectory classes. *Alcoholism: Clinical & Experimental Research* 2000;24:882–891.
- Muthén, LK.; Muthén, BO. *Mplus user's guide*. 3rd ed.. Muthén and Muthén; Los Angeles: 1998–2004.
- Nagin DS. Analyzing developmental trajectories: A semiparametric, group-based approach. *Psychological Methods* 1999;4:139–157.
- Neter, J.; Wasserman, W.; Kutner, MH. *Applied linear statistical models: Regression, analysis of variance, and experimental designs*. Irwin; Homewood, IL: 1990.
- O'Malley PM, Bachman JG, Johnston LD. Reliability and consistency of self-reports of drug use. *International Journal of the Addictions* 1983;18:805–824. [PubMed: 6605313]
- Parra GR, Sher KJ, Krull JL, Jackson KM. Heavy drinking occasions and peer alcohol involvement: A latent growth analysis over 11 years. 2003 Manuscript submitted for publication.
- Schulenberg JE, Maggs JL. A developmental perspective on alcohol use and heavy drinking during adolescence and the transition to young adulthood. *Journal of Studies on Alcohol* 2002;S14:54–70.
- Schulenberg J, Maggs JL, Long SW, Sher KJ, Gotham HJ, Baer JS, Kivlahan DR, Marlatt GA, Zucker RA. The problem of college drinking: Insights from a developmental perspective. *Alcoholism: Clinical & Experimental Research* 2001;25:473–477.
- Schulenberg, J.; Maggs, JL.; Steinman, K.; Zucker, RA. Development matters: Taking the long view on substance abuse etiology and intervention during adolescence. In: Monti, PM.; Colby, SM.; O'Leary, TA., editors. *Adolescents, alcohol, and substance abuse: Reaching teens through brief intervention*. Guilford Press; New York: 2001. p. 19-57.
- Schulenberg, J.; O'Malley, PM.; Bachman, JG.; Johnston, LD. "Spread your wings and fly": The course of health and well-being during the transition to young adulthood. In: Crockett, L.; Silbereisen, R., editors. *Negotiating adolescence in times of social change*. Cambridge University Press; New York: 2000. p. 224-255.
- Schulenberg J, O'Malley PM, Bachman JG, Wadsworth KN, Johnston LD. Getting drunk and growing up: Trajectories of frequent binge drinking during the transition to young adulthood. *Journal of Studies on Alcohol* 1996;57:289–304. [PubMed: 8709588]
- Schulenberg J, Wadsworth KN, O'Malley PM, Bachman JG, Johnston LD. Adolescent risk factors for binge drinking during the transition to young adulthood: Variable- and pattern-centered approaches to change. *Developmental Psychology* 1996;32:659–674.
- Schulenberg, JE.; Zarrett, NR. Mental health during emerging adulthood: Continuity and discontinuity in courses, causes, and functions. In: Arnett, JJ.; Tanner, J., editors. *Emerging adulthood*. American Psychological Association; Washington DC: in press
- Schwartz G. Estimating the dimension of a model. *Annals of Statistics* 1978;6:461–464.
- Sher KJ, Gotham HJ, Erickson DJ, Wood PK. A prospective high-risk study of the relationship between tobacco dependence and alcohol use disorders. *Alcoholism: Clinical and Experimental Research* 1996;20:485–491.

- Sher, KJ.; Jackson, KM. Similarities and differences of longitudinal phenotypes across alternate measures of alcohol involvement. In: Russell, M., editor. Connecting the dots: Drinking patterns and alcohol problems from a life course perspective; Symposium to be conducted at the meeting of the Research Society on Alcoholism; Fort Lauderdale, FL. June, 2003; 2003. Chair
- Sher KJ, Walitzer KS, Wood PK, Brent EE. Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. *Journal of Abnormal Psychology* 1991;100:427–448. [PubMed: 1757657]
- Sher, KJ.; Wood, PK. Methodological issues in conducting prospective research on alcohol-related behavior: A report from the field. In: Bryant, KJ.; West, SG.; Windle, M., editors. The science of prevention: Methodological advances from alcohol and substance abuse research. American Psychological Association; Washington, D.C.: 1997. p. 3–41.
- Shiffman S. Tobacco “chippers”: Individual differences in tobacco dependence. *Psychopharmacology* 1989;97:539–547. [PubMed: 2498951]
- Stacy AW, Galaif ER, Sussman S, Dent CW. Self-generated drug outcomes in high-risk adolescents. *Psychology of Addictive Behaviors* 1996;10:18–27.
- Torabi MR, Bailey WJ, Majd-Jabbari M. Cigarette smoking as a predictor of alcohol and other drug use by children and adolescents: Evidence of the “gateway drug effect.”. *Journal of School Health* 1993;63:302–306. [PubMed: 8246462]
- Tucker JS, Orlando M, Ellickson PL. Patterns and correlates of binge drinking trajectories from early adolescence to young adulthood. *Health Psychology* 2003;22:79–87. [PubMed: 12558205]
- von Eye, A. Configural frequency analysis: Methods, models, and applications. Erlbaum; Mahwah, NJ: 2002.
- Wechsler H, Austin SB. Binge drinking: The five/four measure. *Journal of Studies on Alcohol* 1998;59:122–124. [PubMed: 9498324]
- Wechsler H, Isaac N. “Binge” drinkers at Massachusetts colleges. *Journal of the American Medical Association* 1992;267:2929–2931. [PubMed: 1583763]
- Wechsler H, Lee JE, Kuo M, Lee H. College binge drinking in the 1990s: A continuing problem: Results of the Harvard School of Public Health 1999 College Alcohol Study. *Journal of American College Health* 2000;48:199–210. [PubMed: 10778020]
- White HR, Johnson V, Buyske S. Parental modeling and parenting behavior effects on offspring alcohol and cigarette use: A growth curve analysis. *Journal of Substance Abuse* 2000;12:287–310. [PubMed: 11367605]
- White HR, Pandina RJ, Chen P. Developmental trajectories of cigarette use from early adolescence into young adulthood. *Drug and Alcohol Dependence* 2002;65:167–178. [PubMed: 11772478]
- Widiger TA, Clark LA. Toward *DSM–V* and the classification of psychopathology. *Psychological Bulletin* 2000;126:946–963. [PubMed: 11107884]
- Windle M. A longitudinal study of antisocial behaviors in early adolescence as predictors of late adolescent substance use: Gender and ethnic group differences. *Journal of Abnormal Psychology* 1990;99:86–91. [PubMed: 2307771]
- York JL, Hirsch JA. Drinking patterns and health status in smoking and nonsmoking alcoholics. *Alcoholism: Clinical and Experimental Research* 1995;19:666–673.
- Zacny, JP. Behavioral aspects of alcohol–tobacco interactions. In: Galanter, M., editor. Recent developments in alcoholism Vol. 8: Combined alcohol and other drug dependence. Plenum Press; New York: 1990. p. 205–219.
- Zucker, RA. In: Rivers, PC., editor. The four alcoholisms: A developmental account of the etiologic process; Nebraska Symposium on Motivation, 1986: Alcohol and addictive behavior; Lincoln: University of Nebraska Press; 1987. p. 27–83.
- Zucker, RA. Pathways to alcohol problems and alcoholism: A developmental account of the evidence for multiple alcoholisms and for contextual contributions to risk. In: Zucker, RA.; Howard, J.; Boyd, GM., editors. The development of alcohol problems: Exploring the biopsychosocial matrix of risk. National Institute on Alcohol Abuse and Alcoholism; Rockville, MD: 1994. p. 255–289. NIH Publication No. 94–3495
- Zucker, RA.; Fitzgerald, HE.; Moses, HD. Emergence of alcohol problems and the several alcoholisms: A developmental perspective on etiologic theory and life course trajectory. In: Cicchetti, D.; Cohen,

DJ., editors. Developmental psychopathology: Vol. 2. Risk, disorder, and adaptation. Wiley; New York: 1995. p. 677-711.

Figure 1.

Heavy drinking (top panel) and smoking (bottom panel) at Times 1–4 as a function of birth cohort. Item responses for heavy or binge drinking were 1 (*never drink*), 2 (*once*), 3 (*twice*), 4 (*3–5 times*), 5 (*6–9 times*), and 6 (*10 or more times*). Item responses for quantity of smoking were 1 (*not at all*), 2 (*less than 1 cigarette per day*), 3 (*1–5 cigarettes per day*), 4 (*about 1/2 pack per day*), 5 (*about one pack per day*), 6 (*about 1½ packs per day*), and 7 (*2 packs or more per day*)

Figure 2.

Mixture model for frequency of heavy drinking (left) and smoking quantity (right) at Times 1-4 weighted by estimated class probabilities. Akaike's (1987) Information Criterion = 577,348.87; Bayesian Information Criterion (Schwartz, 1978) = 578035.59; Entropy = .92.

Table 1
Year Assessed as a Function of Birth-Year Cohort and Age (at Time of Assessment)

Birth-year cohort	Age											
	18	19	20	21	22	23	24	25	26	27		
1955					77	79	81					
1956				77	78	79	80	81	82	83		
1957			77	78	79	80	81	82	83	84		
1958		77	78	79	80	81	82	83	84	85		
1959	77	78	79	80	81	82	83	84	85	86		
1960	78	79	80	81	82	83	84	85	86	87		
1961	79	80	81	82	83	84	85	86	87	88		
1962	80	81	82	83	84	85	86	87	88	89		
1963	81	82	83	84	85	86	87	88	89	90		
1964	82	83	84	85	86	87	88	89	90	91		
1965	83	84	85	86	87	88	89	90	91	92		
1966	84	85	86	87	88	89	90	91	92	93		
1967	85	86	87	88	89	90	91	92	93	94		
1968	86	87	88	89	90	91	92	93	94	95		
1969	87	88	89	90	91	92	93	94	95	96		
1970	88	89	90	91	92	93	94	95	96	97		
1971	89	90	91	92	93	94	95	96	97	98		
1972	90	91	92	93	94	95	96	97	98	99		
1973	91	92	93	94	95	96	97	98	99	00		
1974	92	93	94	95	96	97	98	99	00			
1975	93	94	95	96	97	98	99	00				
1976	94	95	96	97	98	99	00					
1977	95	96	97	98	99	00						
1978	96	97	98	99	00							

Note. In the table cells, years do not contain the first two digits (i.e., 19 for all except the year 2000).

Table 2
Goodness of Fit for the Dual Trajectory Comorbidity Model

No. classes	AIC	BIC	Entropy
2	612,134.79	612,536.77	.94
3	608,195.36	608,655.97	.89
4	596,215.21	596,751.19	.90
5	588,369.40	588,964.00	.93
6	581,357.43	581,993.90	.92
7	577,348.87	578,035.59	.92
8	573,837.80	574,583.14	.92

Note. AIC = Akaike's (1987) information criterion; BIC = Bayesian information criterion (Schwarz, 1978).

Table 3
Zero-Order Correlations Between Heavy Drinking, Smoking, and Predictors

Variable	Frequency of heavy drinking				Smoking quantity			
	Time 1	Time 2	Time 3	Time 4	Time 1	Time 2	Time 3	Time 4
Alcohol expectancies (N = 2,309)	.24	.19	.19	.18	.19	.17	.17	.15
Delinquency (N = 3,554)	.35	.25	.25	.24	.16	.12	.12	.10
Religiosity/conservatism (N = 15,092)	-.20	-.17	-.16	-.16	-.19	-.17	-.16	-.16
Parent education (N = 14,820)	.06	.08	.04	.04	-.09	-.07	-.09	-.10
Sex (% male) (N = 15,162)	.21	.25	.27	.27	-.02	-.00	.01	.01
Race (N = 15,162)								
% Black	-.11	-.11	-.09	-.08	-.08	-.07	-.06	-.05
% Other	-.01	-.01	.00	.00	.03	.03	.03	.03
% Hispanic	-.03	-.03	-.01	-.01	-.04	-.05	-.05	-.05
% Asian	-.05	-.04	-.04	-.04	-.04	-.04	-.04	-.03

Table 4
Means and Proportions of External Predictors Across Dual Trajectories of Heavy Drinking-Smoking Quantity

Predictor	Dual drinking-smoking						
	Nonsmoke, nondrink 57%	Chronic drink, chronic smoke 6%	Low drink, chronic smoke 9%	Chronic drink, low smoke 14%	Moderate drink, late onset smoke 4%	Moderate drink, dv ltd smoke 5%	Moderate drink, moderate smoke 6%
Alcohol expectancies (N = 2,309)	0.20 ^a	0.31 ^{b,c}	0.26 ^c	0.28 ^{b,c}	0.26 ^{b,c}	0.28 ^{b,c}	0.27 ^c
Delinquency (N = 3,554)	1.09 ^a	1.36 ^b	1.17 ^c	1.33 ^{a,b}	1.20 ^c	1.25 ^{b,c}	1.17 ^c
Religiosity (N = 15,092)	2.85 ^a	2.24 ^b	2.39 ^c	2.44 ^c	2.54 ^d	2.40 ^{c,d}	2.55 ^d
Parent education (N = 14,820)	3.65 ^a	3.56 ^a	3.31 ^b	3.89 ^c	3.57 ^a	3.64 ^a	3.68 ^a
Sex (% male) (N = 15,162)	61 ^a	40 ^b	65 ^{a,b}	30 ^c	48 ^d	62 ^{a,b}	70 ^b
Race (N = 15,162)							
% Black	12 ^a	2 ^b	3 ^b	2 ^b	8 ^c	3 ^b	12 ^a
% Other	3 ^a	5 ^a	5 ^a	2 ^b	4 ^{a,b}	3 ^{a,b}	4 ^a
% Hispanic	6 ^a	1 ^b	1 ^b	4 ^c	2 ^{b,c}	4 ^{c,d}	6 ^{a,d}
% Asian	3 ^a	1 ^b	0.2 ^b	1 ^b	1 ^{a,b}	1 ^{b,c}	2 ^{a,c}

Note. Means with that share at least one superscript (within a row) are not significantly different at $p < .001$ according to pairwise odds ratios taken from multinomial logistic regressions (with different reference groups). Means are presented for continuous variables (alcohol expectancies, delinquency, religiosity, and parent education); proportions are presented for categorical variables (sex, race). Although continuous variables were standardized for the multinomial logistic regressions, raw means are presented here. Variables were assessed at Time 1. Note that despite multiple start values, the models that included alcohol expectancies as a predictor failed to converge. The estimates shown herein are for models with the latent class part of the model constrained to equal the values in the full model (with no exogenous risk factors; see Footnote 10). dv ltd = developmentally limited.