Adolescent alcohol use and adult alcohol disorders: A two-part random-effects model with diagnostic outcomes

Shelley A. Blozis, Betsy Feldman, Rand D. Conger

Abstract

Alcohol use is often analyzed by treating the behavior as a single dimension, such as focusing on frequency of use. Based on data from a longitudinal study, this report considers two distinct aspects of semi-continuous alcohol use data. A two-part random-effects model was used to evaluate change in the log-odds and frequency of use from about age 13 to about age 18 years. Change features were then related to the log-odds of later alcohol disorders. Results suggested differences in the two aspects of use over time and their relationships with later disorders. Most important for the purposes of this study, different methods of analyzing antecedents and consequences of alcohol use trajectories were shown to generate both similar and disparate findings.

Keywords: Adolescents; Adult alcohol use disorders; Trajectories; Growth models; Random effects model; Longitudinal research; Epidemiology; Methodology

1. Introduction

A recent review of the literature on alcohol-related research concluded that drinking behavior by adolescents and adults can have severe negative consequences for individuals and their communities (National Institute on Alcohol Abuse and Alcoholism, 2000). For example, the research demonstrates significant associations between the abuse of alcohol and traffic accidents, major health problems, violent behavior, and disruptions in family life. The estimated annual cost of drinking problems was estimated to be over $180 billion per year. Moreover, developmental trends in drinking behavior appear to follow an age-related sequence, with the frequency and quantity of alcohol use rising during the adolescent and early adult years and diminishing thereafter. This finding poses several major questions for the study of drinking behavior. For example, how can this adolescent and early adult increase in alcohol use be adequately described and explained and what developmental consequences does it have? The present study contributes new information regarding these questions. Specifically, we consider a recently proposed statistical technique expected to deal effectively with the typically observed skewness in alcohol use data and compare it with a more traditional approach.

1.1. Antecedents and consequences of drinking trajectories

Earlier research on trajectories of drinking behavior has tended to focus on the quantity of alcohol use and, in particular, on binge drinking, which is typically defined as four, five, or six drinks in a row on a single occasion (e.g., Hill et al., 2000; Muthén and Muthén, 2000; Tucker et al., 2003). For example, Hill et al. (2000) followed a cohort of children from an average of 10 years of age to an average of 21 years old. For each wave of assessment during this period they asked participants how often during the past 30 days they had consumed five or more drinks in a row on a single occasion. Using contemporary statistical procedures for identifying trajectory classes, they examined both factors that predicted membership in a specific trajectory class as well as several important consequences of class membership. This report by Hill and his colleagues is very similar to other research on developmental trends in binge drinking (e.g., Chassin et al., 2002; Muthén and Muthén, 2000; Oesterle et al.,...
The research on trajectories in binge drinking provides important insights into the antecedents and consequences of particular classes of binge drinkers. First, all of these studies identify three or more classes of binge drinkers, typically ranging from participants who rarely or never drink to excess to those who persistently and oftentimes increasingly engage in high levels of such behavior. Second, these investigations have found many significant predictors or antecedents of trajectory class membership. Two consistently significant predictors have been participant sex and age of drinking onset. That is, the results show that males compared to females are at greater risk for high and/or increasing trajectories of binge drinking and those participants who demonstrated early onset of binging also are at greater risk than those who start later for entering into the most problematic drinking trajectories. Third, these studies demonstrate agreement in showing that the highest rate trajectories of binge drinking predict alcohol disorders involving abuse and/or dependence at a later point in time.

Although the majority of recent reports on trajectories of alcohol use have focused on binge drinking, some studies have examined drinking frequency (e.g., Flory et al., 2004; Li et al., 2001). For example, in a study of a cohort of youth across the period from 11 to 21 years of age, on each measurement occasion Flory et al. (2004) asked respondents how many drinks they had altogether during the past 30 days. All reported drinks might have been consumed on a single day or on many separate days; thus, quantity of drinking on a single day could not be determined. The important lesson from these studies is that trajectories of drinking frequency are predicted by similar variables as trajectories of binge drinking and that trajectories of frequent drinking predict to alcohol abuse and dependence in the same fashion as trajectories of binge drinking. Indeed, across both types of studies it appears that any drinking trajectory that reflects higher levels of alcohol consumption than the infrequent or non-using trajectory is predicted by similar variables and has similar consequences. Although this observation is likely true for adolescents, we should note that it might not apply equally as well to adult drinking. For example, regular moderate drinking (e.g., a glass of wine with dinner each evening) by adults may actually be health promoting rather than harmful. Regular adolescent drinking, on the other hand, is less likely to occur in this type of normative context.

For adolescents, research suggests that it may be an above average level or increase in the probability of alcohol use that is being predicted or that effectively predicts later alcohol disorders. For this reason, approaches to modeling drinking trajectories that do not rely on the identification of unique trajectory classes may provide meaningful alternatives to this common statistical technique. We consider the positive and problematic aspects of several of the possible methods for analyzing drinking trajectories in the following section. While there are a variety of analytic procedures for assessing drinking behaviors, we limit our discussion to those that concern data that are described as having a high frequency of zeros with the remaining scores being continuously distributed.

### Table 1

<table>
<thead>
<tr>
<th>Method</th>
<th>Common data treatments</th>
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<tbody>
<tr>
<td>Latent class growth model</td>
<td>Treat as continuous or categorical (e.g., use vs. no-use)</td>
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<tr>
<td>Latent curve models</td>
<td>Treat as continuous</td>
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<tr>
<td>Finite mixture growth models</td>
<td>Treat as continuous or categorical (e.g., use vs. no-use)</td>
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<tr>
<td>Latent transition models</td>
<td>Treat as categorical (use vs. no-use)</td>
</tr>
<tr>
<td>Two-part random-effects models</td>
<td>Simultaneous treatment of use/no-use and remaining continuous distribution</td>
</tr>
</tbody>
</table>

### 1.2. Analytic methods for semi-continuous longitudinal drinking data

#### 1.2.1. Latent class growth models

Substance-use data are often described as semi-continuous, that is, non-normal with a high frequency of zeros representing non-use and the remaining values being continuously distributed and often positively skewed. Semi-continuous data present several challenges for the analysis of drinking trajectories and several methods have been proposed to address them (see Table 1). A popular approach for dealing with longitudinal, semi-continuous data is a latent class growth model that assumes individual differences in growth are due to two or more latent class trajectories (Nagin, 1999; Nagin and Tremblay, 2001). As discussed earlier, this is a very common approach to evaluating the antecedents and consequences of drinking trajectories. With this method, individual variation apart from that captured by the class curves is assumed to be due to error. Class membership is unobservable, but the approach yields estimated probabilities of membership for each individual. Associations between hypothesized antecedents and consequences of the different latent classes may then be studied by including in a model such variables as predictors or criterion measures. A benefit of the approach is that it may well describe data that are not normally distributed prior to allowing for the latent classes. Considering substance-use data, the assumption that observations are normal deviations about class curves may not be tenable, however, particularly when data are semi-continuous or change is systematic at the individual level within classes.

#### 1.2.2. Latent growth curve models

An alternative approach is a latent growth curve model that simultaneously characterizes the population curve as well as the curves of individuals (Laird and Ware, 1982; Singer and Willett, 2003). When change follows a linear trend, for example, a model that includes an intercept and linear time effect may well describe change in the response. Random effects (e.g., random intercept and slope) allow each individual to have a trajectory that may be unique from others as well as the population. That is, responses at the individual level are not simply random deviations about a mean curve but rather are assumed to vary systematically as functions of time, with the addition of the random error about the individual’s curve. The parameters are combinations of what is common to all individu-
als plus that which is unique to the individual. Given individual differences in change characteristics, it may also be possible to study the moderating effects of person-level (e.g., sex) or contextual (e.g., school size) variables on change features, or the effects of change features on outcome variables. For example, girls and boys may differ on average in their rates of change in alcohol use, or change in alcohol use may be predictive of a later alcohol disorder diagnosis. Several applications of the latent growth curve model to substance-use data have appeared in the literature (Andrews and Duncan, 1998; Chassin et al., 1996; Duncan et al., 1997; Duncan and Duncan, 1996). Under the model, responses are assumed to be normal, however, making the application of the model to semi-continuous substance-use data potentially problematic in some situations.

1.2.3. Finite mixture growth models. A random-effects model that assumes a normal mixture of distributions for the random effects at the second level (Verbeke and Lesaffre, 1996, Section 4.1) represents a combination of features from the latent class growth model and the latent growth curve model. Individual trajectories are assumed to be due in part to latent classes. Within classes, responses at the individual level are assumed to be systematic, such as a function of time and not simply due to random error about a mean curve. A finite growth mixture model may be useful when the distribution of an outcome variable is not normal when considered as a function of time (or other known covariates) but is normal when considered as both a function of time and the latent classes (and possibly known covariates). This model was later extended to allow the latent classes to influence a separate set of outcome variables (Muthén and Shedden, 1999). There have been a few applications of a latent growth mixture model to adolescent substance-use data (e.g., see Colder et al., 2001; Muthén and Muthén, 2000). Similar to the assumption underlying a latent curve model, responses are assumed to be normal, which may also present problems when applying a finite growth mixture model to semi-continuous substance-use data.

1.2.4. Latent transition models. The methods described above concern change in a continuous variable. An alternative approach is to consider behavioral changes as transitions between discrete states, such as the transition from no-use to the onset of substance use. Different methods have been proposed for dealing with data characterized by discrete states. One approach is a latent transition model in which there are presumably multiple states, such as a state of non-use, and where individuals may transition in and out of the different states over time (Langeheine, 1994). This approach allows, for example, one to consider group differences in latent states at different time points, making it possible to study group differences in substance-use onset (Lanza and Collins, 2002). Thus, while potentially useful in characterizing movement through a variety of states, a latent transition model was not developed for the study of the continuous aspect of behavioral data, such as drinking frequency.

This discussion indicates that the distribution of alcohol or other substance use in the population creates analytic problems for many standard techniques of evaluating change over time. For this reason, a few approaches that address the non-normality of substance-use data have been suggested. One approach is to add one to each response and then use a log transformation (e.g., Colder et al., 2001), although this approach may not lead to approximately normal distributions, particularly when there are large numbers of zeros. Another is to assume a censored-normal distribution in which the censoring is assumed to address the high frequency of zeros. This latter approach has been criticized, however, due to the inappropriate treatment of zeros (see Olsen and Schafer, 2001). That is, a censored distribution does not treat the zeros as valid data points, but rather, assumes the values are proxies for negative or missing values.

In many studies of substance use it may be important to include in a statistical model variables that may be related to substance-use behaviors. These may be variables that distinguish different populations under investigation or those that relate to the individuals directly. Common to the methods described above that were intended for continuous data is the assumption that if other variables are introduced into a model, such as to test for the moderating effects of person-level or contextual variables on substance-use behaviors, a covariate is related in the same manner to both the decision to engage in the behavior as well as the amount or frequency of use (Olsen and Schafer, 2001). When semi-continuous substance-use behaviors are analyzed directly, the distinction between the likelihood of engaging in the behavior and the quantity or frequency of consumption, as measured on a continuous scale, when engaged cannot be made. Similarly, when status of use (e.g., onset) alone is studied, this distinction again cannot be made.

1.3. The present study

In the following analyses we evaluate the effectiveness of a two-part random-effects model in describing longitudinal, semi-continuous alcohol use data as compared to a more traditional latent growth curve model. Based on multi-year data from a cohort of adolescents, we consider a two-part random-effects model to address questions regarding the description, antecedents, and consequences of drinking trajectories. The procedure handles both the distributional qualities of semi-continuous substance-use data and the possibility that covariates may differentially predict the probability that one engages in the behavior and the degree of engagement. A related approach is that proposed by Berk and Lachenbruch (2002) that combines a random-effects logistic model and a random-effects model for log-normal repeated measures that also assumes left censoring. Unlike a two-part random-effects model, the procedure does not make the explicit distinction between the two processes that may underlie the response variable.

In the present report, we investigate whether the two-part random-effects model both parsimoniously describes trajectories of drinking frequency and provides sensitive indicators of change that are predictable and consequential. Drawing on the research discussed earlier, we expected that being male and engaging in drinking at a young age would predict higher levels and increases in drinking behavior. We also expected that the growth parameters in the model would predict alcohol disorders
involving abuse and dependence. As a means of comparison, we also fit a latent growth curve model to the alcohol data. Unlike a two-part random-effects model that distinguishes between the two aspects of use, a latent growth curve model treats the response as a continuous measure. Thus, the comparison between these two approaches may be helpful in emphasizing potential benefits of a two-part random-effects model.

1.3.1. Two-part random-effects models. A two-part random-effects model is based on two subcomponents, each addressing a unique aspect of an observed behavior (Olsen and Schafer, 2001; Tooze et al., 2002). Part 1 is a model for an indicator variable that takes on the value of 1 if the individual engaged in the behavior, and 0 if not. The indicator variable is missing otherwise. Part 2 is a model for a continuous variable that is equal to the original observed response if the individual engaged in the behavior, and is missing otherwise. Thus, this part of the model concerns the response conditional on whether the individual engaged in the behavior or not. Considering the two aspects of the behavior longitudinally, change in each is modeled. In the present analysis, the first sub-model of change concerned the log-odds that an individual used alcohol and the second concerned change in the frequency of alcohol consumption when the individual reported some level of use. In a two-part random-effects model, the two model parts that characterize change in each variable are considered simultaneously. Specifically, characteristics of change in each may be allowed to covary, yielding correlations between change features corresponding to one aspect of a behavior with change features relating to the second aspect. In the context of the current study, for example, it was possible to consider the association between the log-odds that one used alcohol at a young age and change in the conditional frequency of use over the study period.

1.3.2. Assumptions concerning no-use. Underlying valid inference of a two-part random-effects model is an assumption that the single discrete value (e.g., zero) is random. For example, when zero represents the absence of a behavior, an observation of zero is assumed to be due to measurement error or an on-and-off engagement in the behavior. This assumption of random zeros is particularly relevant with regard to substance-use data because zero may represent abstinence from a behavior rather than a random observation of no-use as it would for someone who occasionally engages in the behavior. Non-random zeros are referred to as ‘structural zeros’ (Carlin et al., 2001). In practice, a possible situation is one in which the population contains a combination of individuals characterized by both zero types. One approach to dealing with structural zeros is to exclude cases based on personal, persistent reports of no-use. A potential problem with this approach, however, is that some individuals may be incorrectly identified as true abstainers. One strategy for dealing with this situation in the context of longitudinal data is to consider a finite mixture growth model in which a mixture distribution is assumed for the first part of the model corresponding to use versus no-use to allow for the two populations: one that represents a class of users and the other that represents a class of abstainers. A general description of this type of model is provided in Muthén and Shedden (1999) where a random-effects model is extended to include a mixture distribution for the random-effects at the second level. We applied this procedure to the two-part random-effects model to test the tenability of the assumption of random zeros.

2. Method

2.1. Participants

Data come from the Family Transitions Project, a longitudinal, community epidemiological study of 451 target youth from two-parent families. Data collection began when they were in the seventh (1989) grade and continued on an annual basis through the 1990s with a 90% retention rate through 1999 (see Table 2). The target youth, their parents, and a close-aged sibling participated in the study. Because of a very small minority population in rural Iowa, all participants are of European heritage. Participants in the study were originally recruited to examine the family and developmental effects of the economic downturn in agriculture of the 1980s; for that reason, they were recruited from eight rural counties in Iowa. The original sample of families was primarily lower middle- or middle-class. Additional details about the study can be found in Conger and Conger (2002), Conger et al. (1994), and Simons et al. (1996).

2.2. Variables

2.2.1. Adolescent alcohol use data. Alcohol use was first measured when participants were in the seventh grade and averaged 13.2 years old, with approximately annual follow-ups through tenth grade (average age 16.1 years) with an additional assessment during their senior year of high school (average age 18.1 years). Individuals were asked how often they had consumed beer, wine, or hard liquor during the previous year. Their responses were coded 0 for “never,” 1 for “less than weekly,” 2 for “one to two times per week,” and 3 for “three or more times per week.” Responses for two categories: (1) beer or wine and (2) hard liquor were summed to form one alcohol consumption frequency score, denoted here as yti, for the previous year, where t denotes the measurement occasion and i an individual. The resulting alcohol use variable had a possible range of 0–6. Of the total participants, 343 (76.1%) reported use at one or more times over the targeted period. To fit a two-part random-effects model, two variables were

Table 2
Description of study sample (data from 215 male and 236 female participants in the Family Transitions Project, 1989–1999)

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<tbody>
<tr>
<td>Sex (count)</td>
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<td>Male</td>
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<td>200</td>
<td>189</td>
<td>188</td>
<td>196</td>
<td>191</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>236</td>
<td>224</td>
<td>218</td>
<td>216</td>
<td>228</td>
<td>227</td>
<td>224</td>
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<td>407</td>
<td>404</td>
<td>424</td>
<td>424</td>
<td>421</td>
<td>405</td>
</tr>
<tr>
<td>Age (mean and S.D.)</td>
<td></td>
<td>13(0.42)</td>
<td>14 (0.42)</td>
<td>15 (0.43)</td>
<td>16 (0.43)</td>
<td>18 (0.42)</td>
<td>21 (0.42)</td>
<td>24 (0.43)</td>
</tr>
</tbody>
</table>

Note: For Age, the standard deviation (S.D.) is given in parentheses.
created that related to the observed measures of past-year alcohol use. The first was a dichotomous variable, denoted as \( m_0 \) that, for time \( t \), was equal to 1 if the individual used alcohol, equal to 0 if the individual did not use alcohol, and was missing otherwise. The second variable, denoted as \( m_1 \), was, for time \( t \), equal to the original alcohol use variable, \( y_{ti} \), if the individual used alcohol, and was missing otherwise.

2.2.2. Alcohol disorder diagnosis. In addition to reported alcohol use, lifetime and past-year alcohol disorders were assessed using the University of Michigan modification of the Composite International Diagnostic Interview (UM-CIDI, DSM-III-R; Kessler et al., 1994). This fully structured diagnostic interview generates estimates of DSM-III-R (American Psychiatric Association, 1987) psychiatric disorders in terms of both onsets and recurrences for adolescents and adults. World Health Organization field trials show that the CIDI possesses good reliability and validity (Wittchen, 1994). Past-year diagnoses of (a) dependence and (b) abuse without dependence were made twice, first when the target adolescents were on average 21.1 years old, and again approximately 2.5 years later. Two indicator variables were created to represent the alcohol disorder diagnostic categories: dependence (\( n = 64 \) had positive diagnoses at one or both occasions) and abuse without dependence (\( n = 68 \) had positive diagnoses at one or both occasions). The majority of participants (\( n = 289 \)) had no diagnosis and \( n = 30 \) were missing this information. It is worth noting that some individuals may have met criteria for either classification prior to assessment. Table 3 provides descriptive statistics for the alcohol use and disorder variables.

2.2.3. Background variables. Adolescent alcohol use measures were considered functions of age (measured to the nearest month) over a 6-year period. Sex (1 = male; 0 = female) was included to study possible sex differences in alcohol measures of alcohol use frequency when an individual was engaged in the behavior, with frequency centered to 0. The conditional frequency of use at time \( t \), \( m_{ti} \), was considered a linear function of \( \text{Age}_{ti} \), plus a random error term, \( \epsilon_{ti} \):

\[
m_{ti} = \beta_0 + \beta_1 \text{Age}_{ti} + \epsilon_{ti},
\]

where for individual \( i \), \( \beta_0 \) represented the expected conditional frequency of use at age 13 years. The coefficient \( \beta_1 \) represented the expected past-year linear change in frequency. The error, \( \epsilon_{ti} \), represented a random deviation of an observation from an individual’s fitted trajectory, and so, denoted the within-subject error.

The individual-level regression coefficients, \( \beta_0 \) and \( \beta_1 \), each represented the sum of a fixed coefficient (i.e., \( \beta_0 \) and \( \beta_1 \), respectively) common to all individuals, plus a random component (i.e., \( h_{0i} \) and \( h_{1i} \), respectively) unique to

Table 3
Description of study variables (data from 215 male and 236 female participants in the Family Transitions Project, 1989–1996)

<table>
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<tbody>
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<td>Alcohol use (counts)</td>
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<td>77</td>
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<tr>
<td>Female</td>
<td>62</td>
<td>81</td>
<td>96</td>
<td>124</td>
<td>147</td>
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<tr>
<td>Total</td>
<td>126</td>
<td>145</td>
<td>173</td>
<td>225</td>
<td>272</td>
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<tr>
<td>Alcohol use frequencies, if using (means, S.D.)</td>
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<tr>
<td>Male</td>
<td>0.59 (0.20)</td>
<td>0.68 (0.30)</td>
<td>0.84 (0.35)</td>
<td>0.87 (0.41)</td>
<td>1.1 (0.59)</td>
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<tr>
<td>Female</td>
<td>0.64 (0.39)</td>
<td>0.70 (0.37)</td>
<td>0.88 (0.51)</td>
<td>0.87 (0.44)</td>
<td>0.98 (0.57)</td>
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<tr>
<td>Overall</td>
<td>0.62 (0.30)</td>
<td>0.69 (0.34)</td>
<td>0.86 (0.44)</td>
<td>0.87 (0.43)</td>
<td>1.1 (0.58)</td>
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<td>Previous year alcohol abuse diagnosis in 1997 or 1999 (counts)</td>
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<td>Male</td>
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<td>33</td>
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<td>Female</td>
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<td>Total</td>
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</table>
the individual. The coefficients $\beta_0$ and $\beta_1\gamma_1$ were assumed to randomly deviate about their respective population values. The variances of the random intercept and slope characterized the degree of individual differences in the conditional frequency of use at age 13 and the rate of change in the frequency over the targeted period, respectively. The two random effects were also allowed to covary with one another.

In a combined model, the two model parts, that for the log-odds of use and that for the conditional frequency of use, were joined by allowing covariances between the random effects at the second level of each model part. Letting the sets of random effects for the two model parts be represented by $a_i = (a_{0i}, a_{1i})'$ and $b_i = (b_{0i}, b_{1i})'$, the random-effects at the second level were assumed to have a joint normal distribution:

$$
\begin{pmatrix}
a_i \\
b_i
\end{pmatrix} \sim N
\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Phi \right),
\Phi = \begin{pmatrix}
\Phi_{aa} & \Phi_{ab} \\
\Phi_{ba} & \Phi_{bb}
\end{pmatrix},
$$

where the expected values of the random-effects were zero, and the matrix $\Phi$ was a symmetric block covariance matrix. Block elements of the matrix correspond to the covariance matrix for the random-effects included in the logit model ($\Phi_{aa}$), the continuous response model ($\Phi_{bb}$), and the covariances between the random-effects of the two sub-models, ($\Phi_{ab}$). More specifically, the covariance matrix $\Phi_{aa}$ contained the variances of the individual log-odds of use at age 13 (i.e., $\text{var}(a_{0i})$) and the rate of change in log-odds of use over time (var($a_{1i}$)), in addition to the covariances between the two change characteristics (cov($a_{0i}$, $a_{1i}$)).

The covariance matrix $\Phi_{bb}$ contained the variances of the individual conditional frequency levels of use at age 13 (i.e., $\text{var}(b_{0i})$) and the rate of change in frequency of use over time (var($b_{1i}$)), in addition to the covariances between these two change characteristics (cov($b_{0i}$, $b_{1i}$)). The lower block of the matrix, $\Phi_{ab}$, was not a symmetric covariance matrix but rather contained the covariances between the random effects of the two model parts. This was a particularly interesting component of the model because it provided a means for testing the associations between characteristics of change (e.g., intercept and slope) in both the log-odds of use and the conditional frequency of use.

2.3.1.2. Using change characteristics of alcohol use to predict later alcohol disorders. With the two aspects of alcohol use each summarized by a model imposing linear trends on the mean and individual trajectories across the targeted period, the individual-level latent measures of log-odds of use and frequency of use at age 13, as well as the rates of change in each, could be considered predictors of later alcohol disorder diagnostic categories, with child’s sex as a covariate. Given that both disorder diagnostic measures were dichotomously scored, an appropriate model for their regression was a logistic model in which the log-odds of each diagnosis were taken as a criterion measure. The logit models for the diagnostic measures were

$$
\xi_d = \eta_0 + \eta_1\gamma_1 + \eta_2 \text{Miss}_s + \eta_3 \text{Sex}_s + \eta_4 \text{Age},
$$

and

$$
\xi_d = \xi_1 + \xi_2 \text{Miss}_s + \xi_3 \text{Sex}_s + \xi_4 \text{Age},
$$

where $\eta_{00}$ was the log-odds of use at age 13 for females, and $\xi_{00}$ and $\xi_{10}$ denote differences in the two effects between those with complete versus complete alcohol disorder data holding constant the effects of child’s sex. More specifically, $\eta_{00}$ is the conditional frequency of use at age 13 for females, and $\eta_{10}$ is the difference in the conditional frequency of use at age 13 between males and females. The coefficients $\eta_{10}$ and $\eta_{11}$ denote the annual change in use for females and the sex difference in the annual change between males and females, respectively. The errors for these regressions are denoted by $u_0$ and $u_1$, respectively.

With the addition of child’s sex and the pattern for missing alcohol disorder data, the random effects at the second level represented individual differences in the intercepts and slopes of each model part adjusted for sex differences and effects due to the missing data. Consequently, this changed the joint distributions of the random effects as follows:

$$
\begin{pmatrix}
r_i \\
s_i
\end{pmatrix} \sim N
\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Phi \right),
\Phi = \begin{pmatrix}
\Phi_{rr} & \Phi_{rs} \\
\Phi_{sr} & \Phi_{ss}
\end{pmatrix},
$$

That is, the matrices in $\Phi$ and $\Phi_{rs}$ represented the variance–covariance matrices for the random effects of the first and second parts of the model, respectively, after adjusting for the effects of child’s sex and missing data. The matrix $\Phi_{rr}$ was a non-symmetric covariance matrix for the random effects between the two model parts, also after taking into account sex differences and effects due to subject attrition.

2.3.1.1. Second-level regressions. At the second level of the first part of the model, the random intercept, $a_{0i}$, and linear age effect, $a_{1i}$, were regressed on child’s sex and the missing data pattern code, denoted by Miss,s, resulting in two level-2 regressions:

$$
a_{0i} = \gamma_{00} + \gamma_{01}\text{Sex}_s + \gamma_{02}\text{Miss}_s + r_{0i}
$$

and

$$
a_{1i} = \gamma_{10} + \gamma_{11}\text{Sex}_s + \gamma_{12}\text{Miss}_s + r_{1i},
$$

where $\gamma_{00}$ and $\gamma_{10}$ represented the fixed intercept and slope, respectively, for females who were not missing the alcohol disorder data, $\gamma_{01}$ and $\gamma_{11}$ denoted the differences in the two effects between males and females holding constant the effects of the missing data pattern, and $\gamma_{02}$ and $\gamma_{12}$ denoted differences in the two effects between those missing the alcohol disorder data and those who did not hold constant the effects of child’s sex. Specifically, $\gamma_{00}$ was the log-odds of use at age 13 for females with complete alcohol disorder data, $\gamma_{10}$ was the sex difference in the log-odds at age 13 between males and females holding constant the effect of the missing data pattern. The coefficient $\gamma_{10}$ was the annual change in log-odds for females with complete alcohol disorder data, and $\gamma_{11}$ was the sex difference in the annual change in log-odds between males and females holding constant the effect of the missing data pattern. The errors for these regressions are denoted by $r_{0i}$ and $r_{1i}$, respectively.

Similarly at the second level of the second part of the model, the intercept, $b_{0i}$, and linear age effect, $b_{1i}$, were regressed on child’s sex and the missing data pattern. Miss,s, resulting in two level-2 regressions:

$$
b_{0i} = \eta_{00} + \eta_{01}\text{Sex}_s + \eta_{02}\text{Miss}_s + s_{0i}
$$

and

$$
b_{1i} = \eta_{10} + \eta_{11}\text{Sex}_s + \eta_{12}\text{Miss}_s + s_{1i},
$$

where $\eta_{00}$ and $\eta_{10}$ represented the intercept and slope, respectively, for females with complete alcohol disorder data. $\eta_{01}$ and $\eta_{11}$ denote the differences in the two effects between males and females holding constant the effects of the missing data pattern, and $\eta_{02}$ and $\eta_{12}$ denote differences in the two effects between those with incomplete versus complete alcohol disorder data holding constant the effects of child’s sex.
Fig. 1. Structural model relating sex and latent change characteristics of alcohol use based on a two-part random-effects model to diagnostic outcomes.

relationships were specified between the observed conditional drinking frequencies and the corresponding change characteristics, and so they are not described here in detail. Additionally, covariances between the random coefficients of the two model parts are shown by the double-headed arched arrows between these effects.

2.3.2. Latent growth curve model. A latent growth curve model was also fitted to the alcohol use data. For this model, the original alcohol response variable was considered for analysis. Alcohol use frequency was considered a linear function of Age, plus a random error term, $\varepsilon$:

$$ y_{ti} = \beta_0 + \beta_1 \text{Age}_{ti} + \varepsilon_{ti}, $$

where for individual $i$, $\beta_0$ represented the expected frequency of use at age 13 years. The coefficient $\beta_1$ represents the expected past-year linear change in drinking frequency for the individual. The coefficients $\beta_0$ and $\beta_1$ were assumed to be sums of fixed effects (i.e., $\eta_0$ and $\eta_1$, respectively) and random effects ($b_0$ and $b_1$, respectively) and to deviate at random about the fixed effects, $\beta_0$ and $\beta_1$, respectively. The variances of the random intercept and slope characterized the degree of individual differences in the frequency of use at age 13 and the rate of change in frequency of use over the targeted period, respectively. The two random effects were also allowed to covary with one another. Finally, the error, $\varepsilon_{ti}$, represented a random deviation of an observation from an individual’s fitted trajectory at time $t$. Unlike the second part of the two-part random-effects model, inference from this model was not conditional on an individual’s status at a given occasion as one who used or did not use.

At the second level of the model, the individual-level coefficients were considered functions of child’s sex and the pattern of missing data relating to the alcohol disorder diagnoses, similar to the two-part random-effects model:

$$ \beta_{t} = \eta_{t0} + \eta_{t1}\text{Sex}_t + \eta_{t2}\text{Miss}_t + s_t, $$

where $\eta_{t0}$ and $\eta_{t0}$ represent the fixed intercept and slope, respectively, for females with complete alcohol disorder data, $\eta_{t0}$ and $\eta_{t1}$ denote sex differences in the two effects holding constant the effects of missing data, and $\eta_{t2}$ and $\eta_{t3}$ denote differences in the two effects for those with complete versus incomplete alcohol disorder data holding constant the effects of child’s sex. Given the coding for child’s sex and the missing data patterns, $\eta_{t0}$ was the expected frequency of use at age 13 for females with complete alcohol disorder data, and $\eta_{t1}$ was the expected difference in the frequency of use at age 13 between males and females. The coefficients $\eta_{t0}$ and $\eta_{t1}$ denoted the annual change in use for males and the sex difference in the change rate between males and females, respectively, holding constant the effects of the missing data pattern. The errors for these regressions are denoted by $s_{t0}$ and $s_{t1}$, respectively. Similar to the two-part random-effects model, these errors represent the random change characteristics adjusted for child’s sex and the missing data pattern. Thus, their variances and covariance are also adjusted for these effects.

2.3.2.1. Using change characteristics of adolescent alcohol use to predict later alcohol disorders. Similar to the two-part random-effects model, characteristics of change in drinking frequency could be treated as predictors of dichotomous measures of alcohol abuse and dependence diagnoses assessed when individuals were adults, adjusting for sex differences. As was done for the two-part random-effects model, a logistic regression model was used to predict the log-odds of each disorder from child’s sex and the latent characteristics of change in alcohol use during adolescence.

The full model is depicted in Fig. 2. As shown in Fig. 2, observed drinking frequencies (shown in boxes), are dependent on the random effects (shown in circles) that relate to the two change characteristics and are labeled in the figure.
as frequency of use at age 13 and linear change rate in frequency. The paths from the random effects to the observed frequencies represent fixed and known values. Specifically, the paths from the intercept (i.e., frequency at age 13) to the observed frequencies were fixed to 1 because the intercept represented a constant level of the outcome. The paths from the linear time effect to the observed frequencies were set equal to the age of the child at each occasion, with adjustments made due to centering Age. Thus, these path coefficients could vary between individuals.

2.3.3. Estimation. The two-part random-effects model that included the regression of the diagnostic outcome categories on alcohol-use change features, with child’s sex and the missing data patterns as covariates (as specified earlier), represented a combination of continuous and categorical response variables. Simultaneous estimation of the two model parts was carried out using Mplus version 4 (Muthén and Muthén, 1998–2006) with maximum likelihood estimation and robust standard errors. Estimation of the latent growth curve model that included dichotomous outcomes was also estimated using Mplus.

3. Results

3.1. Two-part random-effects model

In addition to testing a model that assumed linear growth in the two aspects of alcohol use, we also fitted quadratic growth curve models to both outcome variables and found no improvement in model fit. We, therefore, describe results for the linear growth models. Homogeneity of variance was assumed for the variances of the errors at the first level of the model for the continuous responses. Initial results suggested that the variance of the random intercept relating to the continuous part of the model (i.e., that relating to the conditional drinking frequency) was close to or equal to zero. This was reasonable given that there was little variation in the conditional drinking frequencies at the first measurement occasion. Thus, the variance of the random intercept was set equal to zero and the model re-estimated. The effects of the missing data pattern on the difference characteristics of change in both the log-odds and the conditional drinking frequencies were not statistically significant and so were dropped from the model.

For females at age 13, the estimated mean log-odds of use was $-1.99$ (S.E. = 0.284), corresponding to a predicted probability of 0.12. On average, change in the log-odds of use for females was estimated to be 0.774 (S.E. = 0.104), suggesting an overall increase. Males did not differ, however, from females in the expected log-odds at age 13 (estimated difference = −0.105, S.E. = 0.361) or the corresponding change rate (estimated difference = −0.068, S.E. = 0.124). For females, the estimated conditional frequency of use was 0.181 (S.E. = 0.043), suggesting an overall low mean level of consumption when females were using. The estimated annual increase in the conditional frequency of use over time for females was 0.110 (S.E. = 0.029), suggesting a slow but constant rate of increase from about age 13 to about age 18. Males did not differ from females in the expected change in the conditional frequencies (estimated difference = 0.041, S.E. = 0.024). Thus, based on these results, males and females have comparable mean trajectories for both the log-odds of use as well as the conditional frequencies of use.

With the exception of the variance relating to the random intercept of the growth model for the conditional frequency response, all the variances associated with the random effects, after adjusting for differences due to child’s sex, were statistically different from zero, as based on conducting deviance tests, suggesting individual differences in three of the four aspects of change in alcohol use. For these random effects whose variances were statistically different from zero, the estimated covariances were studied. The estimated covariance between the intercept and slope of the model relating to the log-odds of use suggested that higher log-odds of use at age 13 tended to be related to slower rates of increase in the log-odds through adolescence (estimated covariance = −0.697; corresponding correlation, $r = -0.13$). Between the two aspects of alcohol use, there was a positive association between the log-odds of use

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1. For model identification purposes in the logit model, the intercept is fixed to zero and the threshold for the binary variable is estimated. The intercept of the logit model is equal to the negative of the threshold parameter (Muthén and Muthén, 1998–2006, p. 436). The estimated threshold for this model was 2.03, which corresponds to an intercept of $-2.03$. The predicted probability of use at age 13 was $\exp(-2.03)/(1 + \exp(-2.03))$, or more simply, $1/(1 + \exp(2.03)) = 0.12$. 

---

**Fig. 2.** Structural model relating sex and latent change characteristics of alcohol use based on a latent growth curve model to diagnostic outcomes.
at age 13 and the rate of change in the conditional frequency of use (estimated covariance = 0.224; corresponding correlation, \( r = 0.45 \)), indicating faster rates of increase in the frequency of use for those who were most likely to use at age 13. The estimated 95\% confidence interval for the covariance between the change in the log-odds of use at age 13 and the change in the conditional frequency of use included zero as an interior point, suggesting no linear association between these aspects of alcohol use.

3.1.1. Evaluating the assumption of random zeros. To evaluate the assumption of random zeros, we first refitted the model described above after removing those individuals who reported no-use during the targeted period. A comparison of the fixed-effects estimates for the complete sample and the subset that excluded those who reported no-use during adolescence suggested a few but minor differences. First, the estimated difference in the log-odds of use for females at age 13 based on the complete sample and the subset was negligible, with an estimate of \(-1.80\) (S.E. = 0.235) based on the complete sample and an estimate of \(-1.26\) (S.E. = 0.271) based on the subset of cases when non-users were excluded. This log-odds of use corresponded to a probability of \(1/(1 + \exp(1.26)) \approx 0.22\). The estimated change in the log-odds of use for females was also negligible, from 0.707 (S.E. = 0.109) for the complete sample to 0.809 (S.E. = 0.109) for the sub-sample. Negligible differences resulted between frequency of use and change in frequency of use between the two samples, as might be expected given that frequency estimates based on the complete sample were conditional, based on observed use at each occasion. The lack of striking differences in model estimates when the two samples were compared suggests that the assumption of random zeros may be tenable.

Olsen and Schafer (2001) also suggested fitting a mixture distribution for the random effects at the second level to evaluate the tenability of the assumption of random zeros. Using a method described in Muthén and Shedden (1999), a mixture distribution for the random effects at the second level was considered that allowed for mean differences in the log-odds of use by assuming two latent classes. Specifically, a two-class mixture distribution for the random effects relating to the log-odds of use was fitted to the complete data set. The revised model did not improve the fit of the model. We therefore proceeded in our analyses assuming that the observed zeros were random across individuals and based the remaining analyses on the complete sample.

3.1.2. Predicting diagnostic outcomes from latent change characteristics of alcohol use during adolescence. As shown in Table 4, in predicting later alcohol disorders the results suggested a sex difference in the log-odds of a dependence diagnosis (estimated difference = 1.79, S.E. = 0.348) but not in the log-odds of an abuse diagnosis (estimated difference = 0.225, S.E. = 0.326), suggesting males were more likely to have a later dependence diagnosis but not an abuse diagnosis. A higher log-odds of drinking at Age 13 was related to a higher log-odds of a later dependence diagnosis (estimated effect = 0.359, S.E. = 0.115) but not to the log-odds of a later abuse diagnosis (estimated effect = 0.019, S.E. = 0.097). Change in the log-odds of use for those who were most likely to use at age 13. The estimated covariance between the change in the log-odds of use at age 13 and the change in the conditional frequency of use included zero as an interior point, suggesting no linear association between these aspects of alcohol use.

<table>
<thead>
<tr>
<th>Parameter estimates for predicting diagnostic outcomes from sex and latent change characteristics of alcohol use, based on a two-part random-effects model</th>
<th>Criterion</th>
<th>Predictor</th>
<th>( \alpha_{0j} )</th>
<th>( \alpha_{1j} )</th>
<th>( \beta_{0j} )</th>
<th>( \beta_{1j} )</th>
<th>( \beta_{2j} )</th>
</tr>
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<tbody>
<tr>
<td>Intercept</td>
<td>Sex</td>
<td>-1.80 (0.225)</td>
<td>-2.24, -1.35</td>
<td>-0.13 (0.13)</td>
<td>-0.274, 0.480</td>
<td>0.707 (0.086)</td>
<td>0.358, 0.836</td>
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<td></td>
<td></td>
<td>0.645 (0.060)</td>
<td>0.541, 0.750</td>
<td>0.198 (0.046)</td>
<td>0.108, 0.288</td>
<td>0.158 (0.020)</td>
<td>0.119, 0.197</td>
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<tr>
<td>Dependence</td>
<td>Abuse with dependence</td>
<td>0.198 (0.036)</td>
<td>0.140, 0.256</td>
<td>0.709 (0.078)</td>
<td>0.581, 0.836</td>
<td>0.709 (0.078)</td>
<td>0.581, 0.836</td>
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<td>Abuse without dependence</td>
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<tr>
<td>Notes: Estimates are maximum likelihood. Standard errors are in parentheses. Estimated 95% confidence intervals for parameters appear in brackets. The coefficients ( \alpha_{0j}, \alpha_{1j}, \beta_{0j}, \beta_{1j} ) and ( \beta_{2j} ) are the individual log-odds of use at age 13, annual change in log-odds, conditional frequency of use at age 13, and annual change in frequency, respectively. The estimated covariance ( \sigma_{ij} ) did not differ statistically from zero so was not considered a predictor of the diagnostic outcomes.</td>
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</table>
of drinking over time was not predictive of a dependence disorder (estimated effect = 0.339, S.E. = 0.401) but was of an abuse disorder (estimated effect = 1.45, S.E. = 0.639). Change in the conditional drinking frequency was not predictive of either a dependence diagnosis (estimated effect = 0.478, S.E. = 1.29) or an abuse diagnosis (estimated effect = 1.03, S.E. = 1.34).

3.2. Latent curve model

Similar to the two-part random-effects model, a quadratic latent growth model was considered and compared to a linear latent growth model, with no improvement in model fit for the more complex model. We, therefore, describe results for linear growth models. Homogeneity of variance was assumed for the variances of the errors at the first level of the model. Results are presented in Table 5. At 13 years, females had an estimated average drinking frequency of 0.347 (S.E. = 0.043), suggesting on average a low level of use. The estimated change rate for females was 0.192 (S.E. = 0.017), suggesting a slow increase through adolescence. The results suggested no sex differences in the mean drinking frequency at age 13 (estimated effect = −0.112, S.E. = 0.062) or in the change rate (estimated effect = 0.041, S.E. = 0.026), suggesting that on average, males and females had similar behavioral trajectories.

The variances of the random intercept (estimated variance = 0.143, S.E. = 0.047) and slope (estimated variance = 0.019, S.E. = 0.007), adjusted for sex differences, were statistically different from zero, as judged by deviance tests. These results suggested that individuals varied with regard to their frequency levels at age 13 and their rates of change over time. Additionally, a statistically significant and positive covariance between the two conditional random effects (estimated covariance = 0.035; corresponding correlation, r = 0.67) suggested that higher levels of use were related to faster rates of increase over time.

Males and females differed in the log-odds of a dependence diagnosis (estimated difference = 1.75, S.E. = 0.388) but not in the log-odds of an abuse diagnosis (estimated difference = −0.008, S.E. = 0.332), with results suggesting that males were more likely than females to have a dependence diagnosis but no difference when considering an abuse diagnosis. Additionally, change in the drinking frequency was predictive of the log-odds of a later dependence diagnosis (estimated effect = 2.40, S.E. = 1.22) as well as the log-odds of a later abuse diagnosis (estimated effect = 3.18, S.E. = 1.36), suggesting a tendency for increases in drinking frequencies to be related to both a later dependence diagnosis and a later abuse diagnosis. However, the 95% confidence interval relating to the effect of change in drinking frequency on the dependence diagnosis had a lower bound value that was quite close to zero.

3.3. Comparison of findings from the two analytic approaches

Results from the two-part random-effects model and the latent growth curve model suggest differences in how drinking behaviors during adolescence develop, as well as how features of change in these behaviors may be related to later alcohol disorder diagnoses. To begin, both approaches were consistent in suggesting no sex difference in drinking frequencies, whether conditional or not conditional of use, at age 13. Both approaches suggested an increase in drinking frequencies through adolescence, with the average change rates being comparable for males and females. The latent growth curve model suggested a tendency for increases in drinking frequencies at age 13 to increase the risk for both a dependence and an abuse diagnosis in adulthood, whereas under the two-part random-effects model, change in the log-odds and the log-odds of early use were predictive of a dependence diagnosis.

4. Discussion

In the present report we evaluated the utility of a two-part random-effects model for estimating developmental trajectories based on alcohol use data. Unlike most approaches to the analysis of longitudinal data that assume a behavior may be represented by a single dimension, a two-part random-effects model assumes a behavior has two components: the first concerning the likelihood that an individual will engage in a behavior, and the second concerning the magnitude or frequency of the behavior when the individual is engaged. This distinction between the two aspects of a behavior may be particularly important when the two differ in their patterns of change or when covariates have different relationships with the two aspects.

Substantively, the results based on a two-part random-effects model for the current sample were generally consistent with earlier research concerning alcohol use. For example, a higher likelihood of early drinking (at ages 13) increased risk for alcohol dependence by about age 23. This finding is consistent with earlier research on trajectories of both drinking frequency (e.g., Flory et al., 2004; Li et al., 2001) and binge drinking (e.g.,
Chassin et al., 2002; Hill et al., 2000). However, because binge drinking represents one aspect of clinical diagnoses for alcohol disorders, we would expect that the findings in terms of antecedents and consequences would likely be more robust if the tested analytic strategy were evaluated using trajectories of binge drinking. Unfortunately that measure is not available across the adolescent years in the present study. There were no differences between males and females in either the log-odds of use or the frequency of use when drinking at age 13, nor were there differences in the rates of change in either aspects of use during adolescence.

As a means of comparison, we also estimated a latent growth curve model in which alcohol use was represented by the original response variable (frequency of use) and corresponding change characteristics of use served as predictors of clinical diagnoses for alcohol disorders, with sex as a covariate. Thus, this analysis considered drinking frequency that was not conditional on whether or not an individual was using. The results suggested no sex differences in the trajectories of frequency of use, similar to the two-part random-effects in which drinking frequencies were considered conditional on whether an individual was using. Unlike the two-part random-effects model, it was the change in the frequency of use that was positively related to both a later dependence and abuse diagnosis.

Despite some of the appealing features of a two-part random-effects model as a method for dealing specifically with semi-continuous data, there may be limitations to the method. The first concerns the assumption of random zeros. This means that any observation equal to zero is assumed to represent a random process, such as an occasion in which a user reports no-use. In studies of adolescent drinking behaviors, it may seem more reasonable to assume that some individuals are true abstainers and thus the model would provide a misfit to the data to the extent that this was the case. Despite this, methods have been proposed to deal with such situations, including a method for incorporating a mixture distribution for the part of the model that relates to whether or not an individuals uses, as described earlier. A second point is that the model does not address the issue of substance-use onset. For example, age at onset may vary across individuals. Although we considered the likelihood of use at an early age of 13 years, some individuals did not report their first use of alcohol until they were older. In adolescent substance use research, onset may be an important feature in characterizing substance use development. A useful addition to the two-part random-effects model for studies where onset is an important feature of development may be an extension of the model to incorporate onset so that that aspect may also be studied and related to other model components. Finally, in the current application of the method, factors that may have been related to either the likelihood of using or the frequency of use were not incorporated into the model. For example, individual trajectories relating to the likelihood of use may be dependent on time-varying environmental factors, such as changes in peer groups or other stressors.

A variety of analytic methods are available for the analysis of substance-use data. Decisions about which ones may be considered for a given study should be dependent on the goals of the study, as well as data characteristics. As was shown here, different treatments of the data lead to both similar and different substantive interpretations, as may have been expected given that the measure of alcohol was treated quite differently by the two approaches considered. In fact, such results suggest that researchers need not be restricted to a single approach, given that the strategies taken are in line with the research questions.

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