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Early Onset Drinking Predicts Greater Level But Not Growth of Alcohol-Induced Blackouts Beyond the Effect of Binge Drinking During Emerging Adulthood

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Abstract

Background—Early onset drinking is associated with later heavy drinking and related consequences. Early drinking onset and binge drinking are also independently associated with blackouts, which are periods of amnesia for events during a drinking episode. The objective of this study was to examine how early onset drinking relates to changes in the frequency of experiencing blackouts across 3 years controlling for year-specific binge drinking.

Methods—Participants (N = 1,145; 67.9% female) from a 6-year, longitudinal study are included in these analyses. Measures of self-reported age at drinking onset included ages at first drink, first high, and first drunk, which were used to create a latent early onset drinking factor. Frequency of binge drinking and blackouts were assessed annually during Years 4 to 6.

Results—Overall, 69.2% of participants reported experiencing blackouts. After controlling for year-specific binge drinking, a growth curve model indicated that early onset drinkers reported more frequent blackouts at Year 4. There were, however, no significant effects of acceleration or deceleration in the frequency of blackouts across the 3 years. Early onset drinkers continued to experience more frequent blackouts compared with those who initiated alcohol use later, despite decreases in binge drinking over time.

Conclusions—Early onset drinkers reported more frequent blackouts across all 3 years, indicating that early alcohol initiation predisposes those individuals to continue to experience more frequent blackouts, despite a decrease in their binge drinking. This may be due to various factors, such as altered hippocampal development and functioning resulting from early alcohol exposure.

Keywords

Alcohol; Blackout; Early Onset Drinking; Binge Drinking; College Students

DESPITE THE LEGAL drinking age being 21 in the United States, many individuals initiate alcohol use at an earlier age. This is problematic as a young age at first drink leads to myriad consequences. For instance, those who consumed their first drink before age 15 were

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more likely to experience negative neurological, physical, and psychological consequences, including alcohol-induced blackouts (Jennison and Johnson, 1994), hangovers, and needing greater amounts of alcohol to achieve the same intoxicated feeling (LaBrie et al., 2008) compared with those who consumed their first drink after age 15. A young age at first drink is also associated with the development of alcohol dependence (Dawson et al., 2008; Hingson et al., 2003, 2006).

Whereas early onset drinking is often conceptualized as first drink, an early age of first becoming intoxicated may also be an important developmental marker. Whereas first drink could mean taking a sip of alcohol, first intoxication corresponds to the first drinking episode in which an individual consumed enough alcohol to feel subjectively intoxicated. Similar to a young age at first drink, those with a first subjective intoxication before age 14 were more likely to develop alcohol dependence compared with those who had a first subjective intoxication at or after age 21 (Hingson et al., 2006). As such, a young age at first drink and a young age at first intoxication appear to predispose one to similar alcohol-related consequences.

Binge drinking, often characterized as consuming 4 or more drinks for women and 5 or more drinks for men on 1 occasion, is also associated with many of the same consequences as early onset drinking, including alcohol use disorders and alcohol-induced blackouts (White and Hingson, 2014). In addition, blackouts are most likely to occur during binge drinking episodes because blackouts typically result from consuming large amounts of alcohol, often in a short period of time (Jennison and Johnson, 1994; White, 2003; White et al., 2004).

Alcohol-induced blackouts are periods of amnesia for all or some events during a drinking episode (Hartzler and Fromme, 2003; Wetherill and Fromme, 2011; White, 2003). Individuals who experience blackouts are unable to recall events, conversations, or even their own actions that occurred during the blackout. Blackouts are thought to result from a failure to transfer information from short-term memory into long-term memory (White, 2003); however, only about half of drinkers will experience blackouts (White et al., 2002).

The fact that some drinkers experience blackouts whereas others who consume similar amounts of alcohol do not suggests that individual differences in biologic or environmental factors likely contribute to the experience of blackouts. Indeed, there is evidence to suggest a genetic basis for susceptibility toward experiencing blackouts (Nelson et al., 2004), including having a family history of problematic alcohol use (Jennison and Johnson, 1994; LaBrie et al., 2011; Marino and Fromme, 2015). Individuals with a history of blacking out also show differential brain activation when intoxicated (Wetherill et al., 2012), demonstrating impaired contextual and episodic memory recall (Hartzler and Fromme, 2003; Wetherill and Fromme, 2011; Wetherill et al., 2012). Further, those with a history of blackouts who binge drink show altered neurochemistry in the anterior cingulate cortex, exhibiting lower gamma-aminobutyric acid (GABA) and glutamate levels as measured by magnetic resonance spectroscopy (Silveri et al., 2014). Consequently, blackouts are unique neurobiologic phenomena which warrant further study.

Addressing Gaps in the Literature

Despite evidence that an early age at drinking onset and binge drinking are both associated with experiencing blackouts (Jennison and Johnson, 1994; White and Hingson, 2014), possible changes in the occurrence of blackouts across time for early onset drinkers have yet to be examined. It is not known, for example, whether early exposure to alcohol might sensitize the brain and predispose one to experiencing alcohol-induced blackouts after accounting for levels of binge drinking over time. Further, it is not known whether changes in binge drinking translate to changes in the frequency of blacking out and how early onset drinking may affect these changes.

Consequently, the objective of this study was to examine how early onset drinking may influence both the level and changes in the experience of alcohol-induced blackouts across time. By controlling for year-specific binge drinking, we separated the influence of early onset drinking on blackouts from the environmental predisposing factor of heavy drinking. Thus, we examined a full model that accounts for early onset drinking, binge drinking, and alcohol-induced blackouts to provide explanatory clarity in a multivariate framework in a nonclinical, diverse sample.

Materials and Methods

Participants and Procedure

Participants were part of an incoming class of first-time college freshmen who were recruited during the summer before they enrolled at a large state university in the Southwestern United States. These data come from the 6-year, longitudinal arm of the study which examined alcohol use and behavioral risks during the transition from high school through college. Initially, 6,391 students were invited to participate, and 4,832 indicated an interest in the study and met the inclusion criterion of being unmarried. Among these, 3,046 participants were randomized to be assessed longitudinally, and the remainder was assigned to assessment conditions not pertinent to the current study. The final longitudinal sample included 2,245 who provided informed consent and completed the baseline survey (for further description of these samples, see Corbin et al., 2008).

There were 10 assessments over 6 years: baseline (high school), biannually during Years 1 to 3, and annually during Years 4 to 6. Participants included in these analyses (N = 1,145) must have completed the Year 4 survey and reported consuming their first drink by this wave of data collection. Overall, 67.9% were female, and 62.2% were Caucasian, 20.1% were Asian, 3.9% were Black, and 13.8% reported other ethnicities. Average age at Year 4 was 21.8 years (SD = 0.3).

The study received institutional review board approval, and all participants provided informed consent. They were contacted by email to complete Web-based surveys. Participants were compensated $40 for each survey during Years 4 to 6.

Measures

Early Onset Drinking—Early onset drinking was a latent factor that was regressed onto age at first drink, first high, and first drunk to capture both objective and subjective indices of early onset drinking. In the measurement model, which demonstrated perfect fit, all the 3 indicators were statistically significant: first drink ($b = 1.444$, $p < 0.001$), first high ($b = 1.819$, $p < 0.001$), and first drunk ($b = 1.638$, $p < 0.001$). Participants were asked to report how old they were when they "took your first drink on your own" (first *drink*), further specifying that this excluded drinking from a parent's glass or drinking as part of a religious ceremony. In addition, participants were asked to report how old they were when they "first got high or light-headed after drinking alcohol" (first *high*) and "first got drunk after drinking alcohol" (first *drunk*). Response options on a 9-point Likert-type scale were as follows: never, <9, 10 to 12, 13 to 15, 16, 17, 18, 19, and >20. Ages were coded as 9, 11, 14, 16, 17, 18, 19, and 20. While all participants included in these analyses reported consuming their first drink, some reported never feeling high or drunk. Those individuals were coded at the oldest age. For the growth curve analysis, ages were reverse-coded to test whether younger ages at drinking onset were positively associated with experiencing blackouts. We used age at first drink, first high, and first drunk reported at Year 4, which was the first-time participants provided these data, to create our latent factor of drinking onset.

Binge Drinking—Using items adapted from Wechsler and Isaac (1992), participants provided an open-ended response to the question: "during the past 3 months, how many times did you have 4/5 [women/men] or more drinks in 1 sitting?"

Alcohol-Induced Blackouts—One question asked participants to indicate the frequency with which they "had difficulty remembering things you said or did or events that happened while you were drinking" during the past 3 months. The response format was a 5-point Likert-type scale, where 1 = never, 2 = some of the time, 3 = half of the time, 4 = most of the time, and 5 = always. To describe the differences between those who did and did not report experiencing blackouts, we dichotomized this measure across all the 3 years as yes/no for experiencing any blackouts. For our main analysis (growth curve model), we used the Likert coding as a measure of frequency of experiencing blackouts.

Covariates and Baseline Characteristics

Demographics: Sex and race were assessed at the baseline survey. They were used in the bivariate analyses by attrition and blackout status. Sex was also entered as a covariate in the growth curve analysis.

Baseline Alcohol Use: To compare the sample used in the current study with those from the final longitudinal sample who were excluded from these analyses, we assessed 2 facets of alcohol use at baseline. Binge drinking was measured in the same manner as previously described. Alcohol-induced blackouts at baseline were taken from the Rutgers Alcohol Problem Index (White and Labouvie, 1989). This item asked respondents whether they "suddenly found yourself in a place that you could not remember getting to." It was dichotomized as yes/no for experiencing blackouts. Whereas this is a narrow definition of blackouts, we did not add our expanded definition of blackouts until the Year 4 survey.

Statistical Analysis

We used SPSS version 18 (SPSS Inc., Chicago, IL) to examine the bivariate associations between demographic and baseline alcohol-use characteristics between those who were included versus excluded from our analyses to determine whether our sample was representative of the overall sample from which our data are drawn. In addition, we performed bivariate analyses between those who did and did not report experiencing any alcohol-induced blackouts by comparing these groups on demographic and alcohol-use characteristics. Chi-square tests were used to examine categorical variables, and 2-tailed *t*-tests were used to examine continuous variables.

Next, using Mplus version 7 (Muthén and Muthén, 1998), we used growth curve modeling (McArdle and Nesselroade, 2003) to estimate the effect of early onset drinking on the growth parameters of experiencing alcohol-induced blackouts across the 3 years. This particular type of structural equation modeling estimates 3 latent, unobserved factors: intercept *(I)*, linear slope *(S)*, and quadratic slope *(Q)* to measure change over repeated assessments (i.e., time). These 3 latent factors estimate the mean level of the outcome variable at the initial time point or where the sample starts *(I)*, the linear growth across all the assessments *(S)*, and any nonlinear (i.e., quadratic) growth across all the assessments *(Q)*. In contrast to the linear slope, the quadratic slope can account for acceleration or deceleration across time.

Results

Attrition Analyses

In total, 3,046 individuals were randomized to be assessed longitudinally. Of these, 2,245 participants completed the baseline survey and comprised the final longitudinal sample. Those who completed the first survey were more likely to be female and lighter drinkers with no racial/ethnic difference compared with those who did not complete the first survey (see Corbin et al., 2008).

The current study includes 1,145 (51.0%) of the original longitudinal sample of 2,245. Inclusion in these analyses required participants to have completed the Year 4 survey and consumed their first drink by this wave of data collection. As shown in Table 1, those included in the current analyses were more likely to be women ($p < 0.001$) and Caucasian ($p = 0.006$) compared with those who were excluded. The 2 groups did not differ in age ($p = 0.068$), number of binge drinking episodes in the past 3 months at baseline ($p = 0.819$), or alcohol-induced blackouts at baseline ($p = 0.471$). Given this, we concluded that the sample for our analyses was representative of the overall sample in terms of alcohol use and related consequences.

Participant Characteristics

All participants had consumed their first drink by Year 4 ($N = 1,145$), but 8.2% reported that they had never felt high after drinking, and 12.2% reported that they had never felt drunk. Overall, 69.2% reported blackouts during Years 4 to 6. Finally, the average number of binge

drinking episodes decreased over the 3-year period (Year 4: $M = 5.00$ [SD = 7.9], Year 5: $M = 3.9$ [SD = 6.8], Year 6: $M = 3.3$ [SD = 6.4]).

As shown in Table 2, there were significant bivariate differences among those who did and did not report experiencing any blackouts across the 3 years. For these descriptive purposes only, alcohol-induced blackouts were dichotomized as yes/no for experiencing any blackouts during Years 4 to 6. Those who reported blackouts were more likely to be Caucasian ($p = 0.005$), had their first drink at a younger age ($p < 0.001$), reported feeling subjectively intoxicated (i.e., high and drunk) for the first time at a younger age ($p < 0.001$), and reported binge drinking more often ($p < 0.001$) compared with those who did not report blackouts.

Growth Curve Analysis

Using maximum-likelihood robust estimation, the growth curve analysis measured the effect of early onset drinking on the growth parameters of a continuous measure of alcohol-induced blackouts across the 3 years, controlling for the effect of sex on blackouts (see Fig. 1). Because binge drinking, our time-varying covariate, was positively skewed, it was treated as a count variable in the model, and as such, we specified a negative binomial distribution. Consequently, traditional model fit statistics are unavailable, but the Akaike information criteria (AIC) and the Bayesian information criteria (BIC) (Akaike, 1987; Sclove, 1987) statistics were calculated for our final model (AIC = 34,709.511 and BIC = 34,860.806).

The early onset latent factor was just identified and, thus, demonstrated perfect fit. We conducted a Wald test to determine whether the effect of binge drinking could be constrained to be equal across time. A significant result ($p = 0.038$) indicated that the effect of binge drinking on blackouts did change over time and, thus, could not be constrained to be equal. Results suggested that the effect of binge drinking on blackouts decreased over time (see Fig. 1), which is consistent with the decrease in the number of binge drinking episodes, despite a stable level of blackouts. Further, because women are more likely to experience blackouts, we included sex as a covariate on the growth parameters of blackouts (i.e., *I, S, Q*). The only significant sex effect was on the intercept, indicating that women were more likely to show a higher level of experiencing blackouts at Year 4 ($b = -0.088$, 95% confidence interval (CI) [−0.170, −0.005], $p = 0.037$). We also tested whether there was a significant time-varying interaction between binge drinking and sex on blackouts; however, we found no significant binge drinking by sex interactions on blackouts across time.

As shown in Fig. 2, early onset drinkers are experiencing more frequent blackouts at Year 4 ($b = 0.124$, 95% CI [0.078, 0.170], $p < 0.001$). When examining the linear slope, there was no significant increase or decrease in blackouts by early onset drinking ($b = 0.037$, 95% CI [−0.057, 0.131], $p = 0.443$). Further, based on the quadratic slope, there was no significant acceleration or deceleration in the frequency of blackouts across time by early onset drinking ($b = -0.007$, 95% CI [−0.055, 0.041], $p = 0.770$). As such, early onset drinkers continued to experience greater levels of blackouts over time, but they did not demonstrate differential growth or change in reported blackouts, despite the fact that the effect of binge drinking on blackouts decreased across the 3 years.

Discussion

This study examined the effect of early onset drinking on the growth parameters of alcohol-induced blackouts across 3 years using a latent factor of age at drinking onset (i.e., age at first drink, first high, and first drunk). Findings indicate that those who started drinking earlier were more likely to show greater levels of blackouts compared with those who initiated alcohol use later. Analyses also indicated that there was no significant acceleration or deceleration in blackouts across time, demonstrating that early onset drinkers continued to experience frequent blackouts even though the average number of binge drinking episodes decreased. Overall, these findings indicate that there are likely unique neurobiologic and possibly genetic factors contributing to the experience of blackouts that are stemming from early alcohol initiation above and beyond those explained by time-varying patterns of binge drinking.

Adolescence is an important period for the final phases of neural maturation (Biagi et al., 2007). During this time, heavy alcohol use can affect the development of brain regions and interfere with cognitive functioning. For instance, individuals who consumed their first drink before age 14 demonstrated neurocognitive deficits and neurodegeneration in brain regions responsible for learning and memory (Zeigler et al., 2005). Further, binge drinking adolescents have impaired visuospatial memory and attention, as well as abnormal brain activation signaling neurocognitive deficits (Squeglia et al., 2009, 2012). As such, initiating alcohol use during adolescence when the brain is more susceptible to the damaging pharmacological effects of alcohol causes the brain to be more vulnerable to the effects of alcohol later in life compared with alcohol exposure during adulthood (Israel et al., 2015). This includes being more susceptible to alcohol-induced hippocampal dysregulation, which leads to memory deficits (Silveri, 2012). For instance, adolescent rats exposed to binge drinking demonstrated poorer working memory than rats exposed to binge drinking during adulthood (White et al., 2000), possibly due to hippocampal neurotoxicity or *N*-methyl-D-aspartate (NMDA) receptor hyperexcitability resulting from heavy alcohol use. Overall, the effect of early alcohol use appears to alter neural functioning, in particular memory.

In turn, the detrimental effect of early onset drinking on neural functioning may be related to the neuroanatomical development that occurs during adolescence, especially in brain regions responsible for memory, which could have lasting negative effects into adulthood. Indeed, animal models have shown that binge drinking during adolescence alters the synaptic structure of the hippocampus, causing functional abnormalities (Risher et al., 2015), as well as producing alterations in GABA receptor subtype expression in the hippocampus (Centanni et al., 2014). Further, similar detrimental effects have been found in human studies, including hippocampal atrophy in adolescents who binge drink frequently (Welch et al., 2013). Given the integral part the hippocampus plays in memory formation, processing, and retrieval, these enduring developmental and structural changes due to early alcohol use may cause the brain to be more vulnerable to the pharmacological effects of alcohol on memory during emerging adulthood. This may then explain why early alcohol use increases susceptibility to experiencing the neurobiologic phenomena of alcohol-induced blackouts and why early onset drinkers continue to experience more frequent blackouts over time despite a decrease in binge drinking, one powerful risk factor for blackouts.

Future Directions

Genetic factors, such as alterations in GABA, NMDA receptors, and brain-derived neurotrophic factors (all known to influence cognition, memory, and potentially blackouts) (Nelson et al., 2004), may help further explain why initiating alcohol use at a young age is associated with the experience of blackouts. We are currently collecting DNA to look at the genetic underpinnings of alcohol-induced blackouts. Future studies might also examine whether genetic factors predict differences in developmental trajectories of blackouts. For example, Schuckit and colleagues (2015) have identified trajectory classes of experiencing any blackouts (yes/no) from middle to late adolescence, but it is unknown whether genetic factors can predict class membership in such a model. In addition, other potential early environmental factors that are associated with problematic drinking and alcohol-related consequences, such as childhood adversity or trauma (Carlson et al., 2015; Smith et al., 2014), can be explored as possible gene × environment interactions that increase the likelihood of experiencing blackouts for early onset drinkers.

Limitations

Our study has several limitations, including the survey methodology, which precludes any conclusions about causality. In particular, participants self-reported the age at which they began drinking, felt high/light-headed for the first time, and felt drunk for the first time at Year 4 of the larger 6-year longitudinal study. Further, participants also self-reported the number of times that they binge drank and whether they experienced blackouts during a 3-month period at each annual assessment. As retrospective measures, they may be biased by the passing of time or inaccurate recall; however, the collection of multiple waves of data increase confidence in the reported experiences. Last, these data were gathered from a college sample, limiting the generalizability to the broader population.

Conclusions

Despite the aforementioned caveats, these findings come from a longitudinal study over 3 years with a large, ethnically diverse sample, which strengthens our findings by allowing us to prospectively examine early onset drinking, binge drinking, and alcohol-induced blackouts over time. We also took a relatively novel approach to the assessment of early onset drinking using a latent construct derived from age at first drink, first high, and first drunk. This allowed us to examine early onset drinking with both objective (first drink) and subjective (first high, first drunk) measures.

Our findings indicate that early onset drinkers reported more frequent blackouts compared with those who initiated alcohol use later. It is noteworthy, and a unique contribution to the extant literature, that early onset drinkers continued to experience more frequent blackouts over time despite a decrease in their binge drinking. Thus, early onset drinking may be a marker of an underlying vulnerability toward experiencing alcohol-related consequences, specifically blackouts, which are not entirely dependent on binge drinking.

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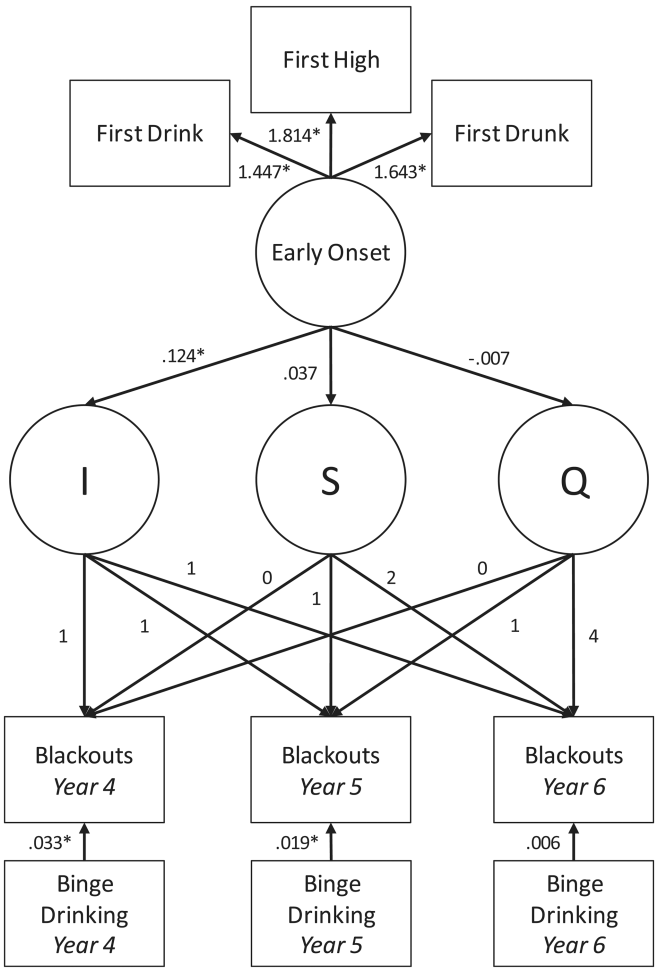

Fig. 1.

Growth curve model for alcohol-induced blackouts across the 3 years. * indicates a statistically significant path at $p < 0.001$. All paths are unstandardized estimates. Covariate included in the model was sex, which was regressed onto the intercept *(I)*, slope *(S)*, and quadratic slope *(Q)* of alcohol-induced blackouts.

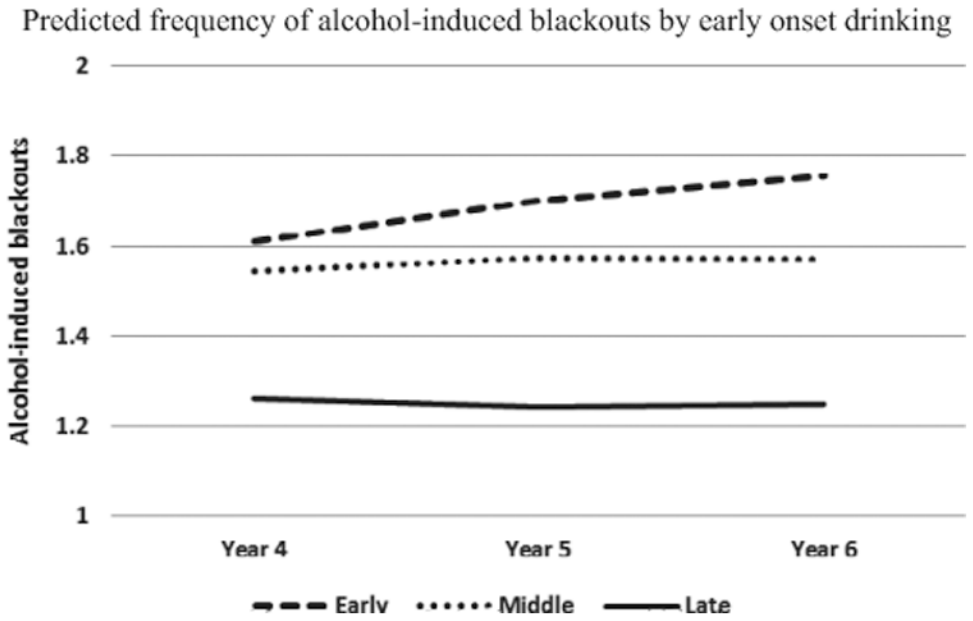

Fig. 2.

Predicted frequency of alcohol-induced blackouts by early onset drinking. Covariate included in the model was sex; time-varying covariates included in the model were year-specific binge drinking. Frequency of alcohol-induced blackouts is based on a Likert scale (1 to 5). Early, Middle, and Late categories represent −1 standard deviation (SD), the mean, and +1 SD of the latent Early Onset factor, respectively.

Table 1

Attrition Analyses: Bivariate Comparisons of Participants' Baseline Characteristics by Inclusion in the Current Study

Participant characteristics (*N* = 2,245)	Included [a] (*n* = 1,145)	Excluded [a] (*n* = 1,100)	*p*-Value
Demographics			
Female	776 (67.8%)	569 (51.6%)	<0.001
Age	18.4 (0.3)	18.4 (0.4)	0.068
Caucasian	712 (62.2%)	622 (56.4%)	0.006
Alcohol use			
Number of binge drinking episodes in the past 3 months	2.1 (5.8)	2.2 (5.0)	0.819
Alcohol-induced blackouts [b]	89 (7.8%)	95 (8.7%)	0.471

[a] The 2,245 who completed the baseline survey comprised the full longitudinal sample. Bivariate comparisons were made between those from that sample who were included versus excluded from the current study.

[b] Based on the Rutgers Alcohol Problem Index blackout item.

n (%) or mean (SD). Comparisons all made at the baseline survey.

Table 2

Bivariate Analyses of Participants' Demographic Characteristics and Alcohol Use by the Experience of Any Blackouts During Years 4 to 6

Participant characteristics (*N* = 1,145)	Blackouts Yes (*n* = 792)	Blackouts No (*n* = 353)	*p*-Value
Demographics			
Female	534 (67.4%)	243 (68.8%)	0.636
Age at Year 4	21.8 (0.3)	21.7 (0.4)	0.158
Caucasian	514 (64.9%)	198 (56.1%)	0.005
Alcohol use			
Age at first *drink*	16.0 (2.5)	17.3 (2.6)	<0.001
Age at first *high*[a]	16.8 (2.1)	18.0 (2.1)	<0.001
Age at first *drunk*[a]	17.1 (2.1)	18.1 (1.9)	<0.001
Year 4 binge drinking[b]	6.8 (8.7)	1.0 (2.5)	<0.001
Year 5 binge drinking[b]	5.5 (7.8)	1.0 (2.6)	<0.001
Year 6 binge drinking[b]	4.7 (7.4)	0.8 (2.6)	<0.001

[a]Among only those reporting an age at first high or first drunk.

[b]Number of binge drinking episodes in the past 3 months at each year. *n*(%) or mean (SD).