Trajectories of alcohol-induced blackouts in adolescence: Early risk factors and alcohol use disorder outcomes in early adulthood

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Declarations of competing interest RB has received untied educational grants from Mundipharma and Indivior for studies relating to pharmaceutical opioids. MF has received untied educational grants from the Australian Government Department of Health, in addition to Mundipharma, Indivior, and Seqirus for post-marketing surveillance of pharmaceutical opioids. AP has received untied educational grants from Mundipharma and Seqirus for post-marketing surveillance of pharmaceutical opioids. These agencies had no role in study design, conduct and reporting, and funding was not for work reported here. All other authors have no conflicts of interest to declare.

Clinical Trial Registration ClinicalTrials.gov (NCT02280551)
Abstract

**Background and aims:** Experience of alcohol-induced memory blackouts in adolescence may be an important risk factor for later harms. This longitudinal study i) modelled trajectories of alcohol-related blackouts throughout adolescence, ii) explored early-adolescent predictors of blackout trajectories, and iii) examined the association between blackout trajectories and alcohol use disorder (AUD) symptoms.

**Design:** Longitudinal study in which data from six annual surveys of a longitudinal cohort of Australian adolescents were used to model latent class growth trajectories of blackouts, adjusting for alcohol consumption frequency and typical quantity. Regression models were used to determine whether parent, child, and peer factors at baseline (\(M_{\text{age}}=12.9\)) predicted profiles of blackout trajectory membership and whether blackout trajectories predicted meeting criteria for AUD in early adulthood (\(M_{\text{age}}=19.8\)).

**Setting and participants:** Australian adolescents (\(n=1821\); \(M_{\text{age}}=13.9\) years until \(M_{\text{age}}=18.8\) years).

**Measurements:** Alcohol-related blackouts, alcohol consumption frequency, typical consumption quantity, and DSM-5 AUD in early adulthood were all self-reported.

**Findings:** We identified a three-class solution: *delayed alcohol initiation, rare blackouts* (\(n=701\); 38.5%); *early initiation, rare blackouts* (\(n=869\); 47.7%); and *early initiation, increasing blackouts* (\(n=251\); 13.8%). Female sex was associated with increased risk of *early initiation, increasing blackouts* relative to *delayed initiation, rare blackouts* (RRR: 3.90; 99.5% CI: 1.96, 7.76) and relative to *early initiation, rare blackouts* (RRR: 2.89; 99.5% CI:
1.42, 5.87). Early initiation, rare blackouts (OR: 1.96; 99.5% CI: 1.17, 3.29) and early initiation, increasing blackouts (OR: 4.93; 99.5% CI: 2.32, 10.48) were each associated with increased odds of meeting criteria for AUD in early adulthood relative to delayed initiation, rare blackouts. Early initiation, increasing blackouts was associated with increased odds of meeting criteria for AUD in early adulthood relative to early initiation, rare blackouts (OR: 2.51; 99.5% CI: 1.18, 5.38).

**Conclusions:** Females in Australia appear to be at higher risk of adolescent alcohol-related blackouts independent of alcohol consumption levels and age of initiation. Alcohol-related blackouts may be associated with later alcohol use disorder.
**Background**

One of the most commonly reported negative consequences of alcohol use in youth is alcohol-induced memory blackouts (1, 2), caused by the interference of alcohol with neural function involved in the formation of long-term memories. Characterised by periods of anterograde amnesia whilst intoxicated (3), a person experiencing a blackout can engage in behaviours such as talking, walking, and driving, but are unable to later recall these actions. This phenomenon is thus entirely distinct from the loss of consciousness following very heavy drinking. Depending on the amount of alcohol consumed and the speed of consumption, blackouts can range from fragmentary, characterised by partial retrieval of events (facilitated by cues such as someone else recounting the event), to en bloc, involving complete and permanent memory loss of events that occurred while intoxicated (4). The occurrence of a blackout is indicative of significant acute cognitive impairment due to rapid intoxication, with the risk of experiencing blackouts increasing with the rate of increase in blood alcohol concentration (3).

Whilst it has been established that levels of alcohol consumption increase steeply from initiation in adolescence to early adulthood (5), it is unclear how the experience of alcohol-induced blackouts changes over time, and whether different patterns of experiencing blackouts are associated with differential risk of experiencing other alcohol-related harms. Blackouts in early adulthood have been associated with increased odds of future alcohol-related injury, sexual assault, and other harms after adjusting for factors such as alcohol use and trait sensation seeking (6-8). Studer, Gmel (9) found that blackouts at age 20 were associated with symptoms of alcohol dependence five years later, after controlling for alcohol
use and other risk factors for alcohol dependence. Thus, blackouts may serve as a useful proxy for harmful levels of alcohol consumption involving both individual differences in tolerance and speed of alcohol consumption.

Current attempts to examine the risk and protective factors for experiencing an alcohol-induced blackout have focused on people aged 18 years and older, and predominantly comprised cross-sectional studies (10). Whilst alcohol consumption and associated harms peak in early adulthood, one study using data from a longitudinal cohort representative of the British population reported that almost 30% of adolescents aged 15 years who had ever consumed at least one full serve of alcohol had experienced a blackout in the past year (11). Distinct trajectories of escalating blackouts have previously been identified through longitudinal research, with some consistent risk factors for frequent blackouts including female sex, other substance use, and peer influences (11-14). These studies examined cohorts of young people who have already initiated alcohol use (11, 13), and/or have been identified due to their problematic alcohol use (12, 14). It is currently unknown how the experience of blackouts may escalate differentially among adolescents from the time of drinking onset, and whether any such trajectories have subsequent consequences. Additionally, previous studies have not taken into account parenting and other familial factors shown to be associated with binge drinking in adolescence, such as parental supply of alcohol and alcohol-specific household rules (15-17). An examination of how child, parent, and family factors influence developmental trajectories of alcohol-induced blackouts, and whether these trajectories are associated with risk of experiencing clinically relevant harms, can aid in identifying high-risk individuals for targeted early intervention.
The aims of this study were to identify the typical trajectories of alcohol-induced blackouts in a longitudinal cohort of young Australians. Specifically, this study examined: i) trajectories of self-reported alcohol-induced blackouts from age 13 to 19 years, whilst adjusting for frequency and typical quantity of alcohol consumption; ii) sociodemographic factors at age 12 that predict the trajectory of alcohol-induced blackouts; and iii) associations between blackout trajectory and meeting Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV; 18) criteria for alcohol abuse, alcohol dependence, and Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5; 19) criteria for alcohol use disorder (AUD) based on self-reported symptoms at age 20 years.

**Methods**

**Participants and procedure**

This study used the Australian Parental Supply of Alcohol Longitudinal Study (APSALS; registered at ClinicalTrials.gov: NCT02280551) cohort of 1927 young people. Participants and one parent or guardian were recruited via an opt-in process in 2010 and 2011 from Grade 7 classes in Australian private/independent (49%), Catholic (12%), and government (39%) schools. Distribution of sex, household composition, racial background, and parental education in the cohort was comparable to the Australian population, although families with higher socioeconomic status were somewhat over-represented. For more details about the recruitment methods and cohort profile, see Aiken, Wadolowski (20). Online or paper hardcopy surveys were sent to participants each year. Parents were also surveyed until the 6th annual wave of data collection. To minimise reporting bias, surveys were sent separately to each adolescent and parent. APSALS was approved by the University of New...
Analyses for this study used eight annual waves of data collection (Wave 1 to Wave 8; 2010-11 to 2017-18; mean ages 12.9 years and 19.8 years, respectively), including data collected from parents at Wave 1. Of the 1927 adolescents recruited into the study, 73.4% (n=1415) completed the Wave 8 survey (see Supplementary Figure S1 for flowchart of cohort retention). Participants who had completed at least three annual surveys were included in the current analyses (n=1821). Reporting is in accordance with Strengthening the Reporting of Observational studies in Epidemiology (STROBE) guidelines (see Supplementary Table S1 for checklist).

Measures

Alcohol consumption.

Self-reported alcohol consumption frequency and typical quantity in each wave were measured using two items, each with eight possible responses: “In the last 12 months, how often have you had an alcoholic drink of any kind?” (never, less than once/month, once/month, 2-3 days/month, 1-2 days/week, 3-4 days/week, 5-6 days/week, every day) and “In the last 12 months, on a day you have an alcoholic drink, how many standard drinks do you usually have?” (none, a sip, 1-2 drinks, 3-4 drinks, 5-6 drinks, 7-10 drinks, 11-12 drinks, 13 or more drinks), where a standard drink is defined as 10g of alcohol (21).

Blackouts.

The alcohol-induced blackout measure consisted of a single item adapted from the School Health and Alcohol Harm Reduction Project (SHAHRP; 22): “In the last 12 months,
how many times have you been unable to remember what had happened while you had been drinking?” with six possible responses (never, once, twice, 3-4 times, 5-11 times, 12+ times). This item was recoded to consider whether participants had consumed any alcohol within the last 12 months (i.e. the original “never” category was separated into “did not drink” and “drank but never had a blackout”).

**Alcohol dependence, alcohol abuse, and alcohol use disorder.**

The alcohol abuse measure consisted of 4 items adapted from the Diagnostic Interview Schedule for Children Version IV (DISC-IV; 23) which corresponded to DSM-IV symptoms of alcohol abuse. The alcohol dependence measure consisted of 7 items, likewise adapted from the DISC-IV and corresponding to DSM-IV symptoms of alcohol dependence. The measure for AUD consisted of 11 items corresponding to DSM-5 symptoms of AUD. Three binary variables were coded for: i) meeting DSM-IV criteria for alcohol abuse (at least one of the four symptoms and have never met criteria for alcohol dependence); ii) meeting DSM-IV criteria for alcohol dependence (at least three of the seven symptoms); and iii) meeting DSM-5 criteria for AUD (at least two of the eleven symptoms).

**Wave 1 characteristics.**

Wave 1 predictors of alcohol-induced blackout trajectories were identified from a literature search (see Supplementary Table S2 for review of literature and details of measures used); these included: child variables (sex, externalising), peer variables (peer disapproval of tobacco/alcohol use, peer tobacco/alcohol use), parent variables (highest level of education, alcohol-specific rules, parental monitoring), and family variables (socioeconomic status, one/two parent household, alcohol accessibility at home without parental knowledge, family
history of alcohol problems, family conflict).

**Statistical analysis**

These analyses were not pre-registered and as such, results should be considered exploratory.

*Latent class growth analyses.*

Latent class growth analyses (1-4 classes) were performed using Mplus version 8 (24). This form of latent growth curve modelling identifies ‘classes’ or clusters of individuals where variance and covariance estimates in the growth factors of observed variables within each class are assumed to be zero (25) to identify meaningful homogenous subgroups that display similar patterns of growth. One set of growth parameters were specified within each class based on the number of blackouts (did not drink, drank but no blackouts, once, twice, 3-4 times, 5-11 times, 12+ times) experienced within each 12-month period from Wave 2 to Wave 7, adjusting for zero-standardised 12-month frequency multiplied by typical quantity of alcohol consumption. Akaike’s Information Criterion (AIC) and sample-size adjusted Bayesian Information Criterion (ssaBIC) were used to assess model fit, where lower values indicated better fit. The Lo-Mendell-Rubin adjusted log-likelihood ratio test (LMR-ALRT; 26) statistic was used to compare fit of a $k$ class model with a $k-1$ class model, where $p < 0.05$ indicated that the $k-1$ class model should be rejected for the $k$ class model. Average class classification probability was used as an index of classification quality, where values approaching 1 (range 0.0-1.0) indicated better differentiation of individuals between classes. Class composition of models was examined alongside fit statistics to determine the most parsimonious and theoretically meaningful class structure.
Accounting for latent class classification uncertainty.

As latent classes cannot be assigned to individuals with certainty, the modified three-step Bolck-Croon-Hagenaars estimation method (BCH; 27, 28, 29) was used to account for classification uncertainty in subsequent analyses: 1) a latent class growth model is estimated; 2) an expanded data file with one record for each latent class for each individual is created, with weights assigned to each record calculated from the inverse of a matrix containing the classification probabilities for most likely latent class membership by latent class (30); and 3) associations between latent class membership and other variables are estimated as a multiple group model using the BCH weighting variable.

Regression analyses.

Logistic regression analyses were performed using Stata version 16 (31), weighted by the BCH variable. Multivariate multinomial logistic regression analyses were used to predict trajectory class membership using Wave 1 characteristics as a predictor (presented as relative risk ratios [RRR] with 99.5% confidence intervals [CIs]). Adjusted binary logistic regression analyses were used to predict whether participants met DSM-IV criteria for alcohol dependence and alcohol abuse, in addition to DSM-5 criteria for AUD, at Wave 8 using class membership as a predictor (presented as odds ratios [OR] with 99.5% CIs). All Wave 1 characteristics described above were included in adjusted regression analyses as covariates.

Missing data.

A summary of missing data can be found in Supplementary Table S3. To reduce any potential bias introduced by missingness in the data, primary analyses were conducted on multiply imputed data. The data were imputed using Mplus version 8 (24) using an
unrestricted H1 model (32) as recommended by Asparouhov and Muthén (30). Based on the percentage of missing information, we used M = 20 imputations (33). Latent class growth analyses were then conducted on each imputed dataset. The resulting datasets containing BCH weights from each run were manually combined and imported into Stata as a multiply imputed dataset for regression analyses. Sensitivity analyses using complete-case data can be found in Supplementary Tables S4-S9 and Figure S2.

Results

Number of blackouts

Figure 1 shows the distribution of the number of 12-month alcohol-related blackouts from Wave 2 to Wave 7. In Wave 2, nearly 10% of adolescents who had consumed alcohol in the past 12 months reported experiencing at least one blackout. By Wave 7, over 47% of young people who had consumed alcohol had experienced at least one blackout. Of the young people who had experienced a blackout in Wave 7, around 14% had experienced five or more blackouts.

Trajectories of blackouts

Fit statistics for the 1- to 4-class latent class growth models are shown in Supplementary Table S10. Although the 4-class model had the smallest ssaBIC, the LMR-ALRT did not indicate improved model fit over the 3-class solution and the model contained a class with less than 10% of the full sample. Examination of the average latent class probabilities matrix showed that the classes were reasonably distinct, with average classification probabilities of 0.830 for Class 1, 0.765 for Class 2, and 0.749 for Class 3 (see Supplementary Table S11). Class composition supported selection of the 3-class model as
each class was of substantive size and showed distinct trajectories of blackouts. Probabilities of endorsing each category of the blackouts variable for each class are summarised in Figure 2. Class 1 was labelled as the delayed initiation, rare blackouts class (n=701; 38.5%), where the majority did not initiate alcohol use until age 17-18 years and less than 30% of the class members had ever experienced a blackout by age 18-19 years. Class 2 was labelled as the early initiation, rare blackouts class (n=869; 47.7%) where the majority had initiated alcohol use by age 14-15 years, with less than 50% ever experiencing a blackout by age 18-19 years. Class 3 was labelled the early initiation, increasing blackouts class (n=251; 13.8%) where the majority had initiated alcohol use by age 14-15 years, with around 97% having experienced a blackout by age 18-19 years. Latent class growth models using complete-case data similarly supported the 3-class solution, with similar average latent class probabilities and class profiles (Supplementary Tables S4, S5, and Figure S2).

Predictors of blackout trajectory

Results of the multivariate multinomial logistic regression models are presented in Table 1 (see Supplementary Table S12 for bivariate models). Wave 1 factors associated with increased risk of early initiation, increasing blackouts relative to delayed initiation, rare blackouts were: female sex (RRR: 3.90; 99.5% CI: 1.96, 7.76), having more peers who use substances (RRR: 1.37; 99.5% CI: 1.07, 1.77), and greater accessibility to alcohol at home (RRR: 1.12; 99.5% CI: 1.01, 1.25). Having more peers who use substances (RRR: 1.34; 99.5% CI: 1.06, 1.70) and greater accessibility to alcohol at home (RRR: 1.08; 99.5% CI: 1.01, 1.15) were each associated with increased risk of early initiation, rare blackouts relative to delayed initiation, rare blackouts. Female sex (RRR: 2.89; 99.5% CI: 1.42, 5.87)
was the only Wave 1 factor associated with early initiation, increasing blackouts relative to early initiation, rare blackouts. Multivariate multinomial logistic regression models using complete-case data showed a similar pattern of results (Supplementary Table S7).

**Blackout trajectory as predictor of meeting criteria for DSM-IV alcohol abuse and dependence, and DSM-5 AUD based on self-reported symptoms**

Results of the adjusted logistic regression models are presented in Table 2 (unadjusted models are presented in Supplementary Table S13). Early initiation, rare blackouts (OR: 1.96; 99.5% CI: 1.17, 3.29) and early initiation, increasing blackouts (OR: 4.93; 99.5% CI: 2.32, 10.48) were both associated with increased odds of meeting self-reported DSM-5 criteria for AUD at Wave 8 relative to delayed initiation, rare blackouts. Early initiation, increasing blackouts (OR: 2.51; 99.5% CI: 1.18, 5.38) was also associated with meeting self-reported DSM-5 criteria for AUD at Wave 8 relative to early initiation, rare blackouts. Early initiation, increasing blackouts was associated with meeting self-reported DSM-IV criteria for alcohol dependence at Wave 8 relative to delayed initiation, rare blackouts (OR: 3.07; 99.5% CI: 1.46, 6.48). Blackout trajectory was not associated with DSM-IV criteria for alcohol abuse. Adjusted logistic regression models using complete-case data showed that these results remained robust (Supplementary Table S9).

**Discussion**

Over eight annual waves of survey data on 1821 young people assessed from age 12 years to age 19 years, we identified three distinct classes of alcohol-related blackout trajectories after adjustment for drinking frequency and quantity, in addition to early-adolescent predictors of blackout trajectory and associated adulthood outcomes. The three
classes identified consisted of: 1) delayed initiation, rare blackouts; 2) early initiation, rare blackouts; and 3) early initiation, increasing blackouts.

Consistent with a recent study which found that blackouts predicted later alcohol dependence (9), our findings indicate that escalation of blackouts in adolescence is associated with three times the odds of meeting DSM-IV criteria for alcohol dependence. Our study also adds the novel finding that early alcohol initiation, in conjunction with increasing blackouts, is associated with alcohol dependence; this was not the case for alcohol initiation alone. We found no difference in meeting DSM-IV criteria for alcohol abuse between the three classes, with neither blackouts nor age of initiation being associated with meeting DSM-IV criteria for alcohol abuse in early adulthood. This is unsurprising given that the symptoms of DSM-IV alcohol abuse pertain to social and work conflicts arising from alcohol use, as opposed to DSM-IV alcohol dependence which includes symptoms that more closely relate to blackouts (e.g., drinking more than intended, drinking more due to increased tolerance, spending more time drinking). Additionally, we found that early initiation and increasing blackouts each independently contributed to increased risk of meeting self-reported DSM-5 criteria for AUD, with the joint effect having the largest effect size of nearly five times the odds of meeting criteria for AUD. Escalating blackouts in adolescence are an important indicator of clinically-relevant alcohol problems, and should be considered as part of a risk factor assessment by clinicians. Prevention and intervention strategies targeting alcohol-induced blackouts may reduce the risk of future alcohol problems and also reduce injury and associated healthcare costs (6, 34).

Among the young people in our sample who rarely experienced blackouts, no sex
differences were found between the delayed and early initiation classes, indicating that sex was associated with experience of blackouts, but not with age of initiation alone. Females had nearly three times the risk of males in experiencing increasing blackouts, which increased to nearly four times the risk when earlier age of initiation was considered. Studies have established that young adult females are at increased risk of experiencing blackouts compared to males after adjusting for levels of consumption (7, 35), an effect which is likely due to differences in metabolism and body composition (36). Our finding that female sex is associated with increased risk of increasing blackouts supplements those of Schuckit, Smith (11) and Schuckit, Smith (14), with an additional novel finding that sex is associated with blackout trajectory independent of alcohol consumption levels and age of alcohol initiation. That is, adolescent females had increased risk of experiencing increasing blackouts compared to adolescent males at equivalent levels of consumption, but females were not more likely than males to start drinking at an earlier age. Although young people tend to understand the behavioural risk factors for alcohol-related blackouts, such as rapid consumption of alcohol, they have limited understanding of biological risk factors such as sex (37). As such, schools should consider educating students and caregivers about the biological risk factors for blackouts, in addition to blackouts themselves being a risk factor for future harm.

Young adolescents residing in households where alcohol was easier to access without parental knowledge had around 1.1 times the risk of early initiation to alcohol relative to delayed initiation, regardless of blackouts. Thus, accessibility of alcohol at home was weakly associated with the timing of alcohol initiation, but not necessarily blackouts. Having more peers who used substances at age 12-years was associated with over 1.3 times the risk of
early alcohol initiation relative to delayed initiation, regardless of blackouts. Our results suggest that peer substance use at age 12-years was only associated with the timing of alcohol use initiation, not with blackout experiences. This is consistent with previous longitudinal research regarding self-selection of alcohol-using peers in adolescents who drink (38), albeit with a small effect size. Contrary to other studies on blackout trajectories (11, 13), we found no evidence to suggest that peer substance use was linked to increasing blackouts as the peer effect was only present between the delayed and early initiation groups. A notable difference between our study and the aforementioned is that the blackout trajectories identified in our study adjusted for same-year alcohol consumption, whereas the trajectories reported by Merrill, Treloar (13) and Schuckit, Smith (11) were unadjusted. It is possible that the presence of substance-using peers drove increases in alcohol consumption (38) and thus blackouts increased due to escalation in drinking levels. Future research examining blackouts should note that analyses that do not adjust for alcohol consumption may instead be capturing patterns of heavy drinking rather than blackouts.

**Strengths and limitations**

To our knowledge, this is the first study to examine alcohol-related blackout trajectories from pre-exposure in early adolescence to early adulthood, whilst also adjusting for frequency and typical quantity of alcohol use. The blackout trajectory classes we identified are comparable to that of previous studies on older adolescents (11) and young adults (13, 14), where increasing blackout and moderate/low blackout groups have also been identified. Our study builds on this prior work by including age of alcohol initiation into the models, given its established association with later alcohol use and disorder (39, 40).
Notably, this study identified two distinct blackout trajectories (i.e., rare and increasing) in adolescents who initiated alcohol in early adolescence, suggesting that increasing blackouts are not necessarily linked to age of initiation. Additional strengths include a large sample size (1821 young people), high retention rate over 8 years (73.4%), repeated 12-month follow-up assessments, and consideration of child, parent, and peer covariates associated with adolescent alcohol use.

There are several limitations to note. Entropy for the chosen 3-class model was 0.586, which may indicate poor delineation of latent classes. However, the classes were theoretically meaningful and classification probabilities ranged from 0.749 to 0.830, meaning that classes were reasonably distinguished and comparable to the blackouts trajectory model chosen by a similar study (11). Our results may not be generalisable at the population level as participants were recruited using an opt-in process rather than randomly sampling from the population. Although our cohort does have similar levels of alcohol use and demographic profile to Australian population, families with low socioeconomic status are somewhat underrepresented due to a lower proportion of government schools participating (20).

Additionally, as alcohol consumption is often underestimated at higher levels of consumption (41), and given that blackouts are characterised by memory loss (3), our retrospective measures of alcohol use and blackouts may have resulted in underestimation of these variables. The extent to which other substance use may have also contributed to alcohol-related blackouts is also unclear, as we did not explicitly ask participants about other substance use in the context of blackouts. Finally, the measure we used for blackouts does not distinguish between en bloc and fragmentary blackouts. Given that en bloc blackouts are
more commonly associated with poly-substance use and are perceived more negatively than fragmentary blackouts (4, 42), we suggest that future studies measure both types of blackouts.

Conclusion

This longitudinal study shows that there are heterogenous patterns of experiencing alcohol-related blackouts across adolescence. Caregivers and educators of adolescents should note that females are particularly at risk of experiencing increasing blackouts which, in turn, places them at increased risk of alcohol-related harms in early adulthood. Although not part of a formal diagnosis of AUD, clinicians may wish to consider the experience of blackouts in adolescents as a risk factor for future clinical problems related to alcohol use independent of alcohol consumption frequency, typical quantity, and age of initiation.
Funding

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Figure 1. Proportion of sample that reported self-reported blackouts in the past 12 months by follow-up wave.


**Figure 2.** Probabilities of endorsing different numbers of blackouts for each class, 3-class solution.
Delayed initiation, rare blackouts (n = 701)

Early initiation, rare blackouts (n = 869)

Early initiation, increasing blackouts (n = 251)
Table 1. Multivariate multinomial logistic regression predicting latent class membership using baseline characteristics.

<table>
<thead>
<tr>
<th>Reference: Delayed initiation, rare blackouts</th>
<th>Reference: Early initiation, rare blackouts</th>
<th>Reference: Early initiation, increasing blackouts</th>
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<tr>
<td></td>
<td>Early initiation, rare blackouts</td>
<td>Early initiation, increasing blackouts</td>
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<tr>
<td>RRR 99.5% CI</td>
<td>RRR 99.5% CI</td>
<td>RRR 99.5% CI</td>
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<tr>
<td>Female sex</td>
<td>1.35 (0.82, 2.22)</td>
<td>3.90 (1.96, 7.76)</td>
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<td>Child externalising</td>
<td>1.02 (0.99, 1.04)</td>
<td>1.00 (0.97, 1.04)</td>
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<td>Peer disapproval of substance use</td>
<td>1.04 (0.91, 1.19)</td>
<td>0.98 (0.81, 1.17)</td>
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<td>Peer substance use</td>
<td>1.34 (1.06, 1.70)</td>
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<td>Parent education (Reference: High school or less)</td>
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<td>Diploma</td>
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<td>University</td>
<td>0.97 (0.53, 1.76)</td>
<td>1.40 (0.63, 3.14)</td>
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<td>Alcohol specific household rules</td>
<td>0.82 (0.47, 1.40)</td>
<td>0.84 (0.49, 1.44)</td>
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<td>Parental monitoring</td>
<td>0.94 (0.85, 1.04)</td>
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<td>Single parent household</td>
<td>1.73 (0.87, 3.43)</td>
<td>1.40 (0.58, 3.38)</td>
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<td>Accessibility of alcohol at home</td>
<td>1.08 (1.01, 1.15)</td>
<td>1.12 (1.01, 1.25)</td>
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<tr>
<td>Family history of alcohol problems</td>
<td>1.02 (0.63, 1.65)</td>
<td>0.98 (0.52, 1.85)</td>
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<tr>
<td>Family conflict</td>
<td>1.18 (0.89, 1.57)</td>
<td>1.16 (0.81, 1.68)</td>
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Table 2. Adjusted logistic regression predicting meeting criteria for DSM-IV alcohol dependence and abuse, and DSM-5 AUD by latent class.

<table>
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<tr>
<th>Class</th>
<th>Dependence</th>
<th>99.5% CI</th>
<th>Abuse</th>
<th>99.5% CI</th>
<th>AUD</th>
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<tbody>
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<td></td>
</tr>
<tr>
<td>Early initiation, rare blackouts</td>
<td>1.58</td>
<td>(0.82, 3.04)</td>
<td>0.98</td>
<td>(0.15, 44.52)</td>
<td>1.96</td>
<td>(1.17, 3.29)</td>
</tr>
<tr>
<td>Early initiation, increasing blackouts</td>
<td>3.07</td>
<td>(1.46, 6.48)</td>
<td>0.38</td>
<td>(0.03, 5.70)</td>
<td>4.93</td>
<td>(2.32, 10.48)</td>
</tr>
<tr>
<td>Reference: Early initiation, rare blackouts</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early initiation, increasing blackouts</td>
<td>1.95</td>
<td>(1.00, 3.80)</td>
<td>0.39</td>
<td>(0.02, 6.87)</td>
<td>2.51</td>
<td>(1.18, 5.38)</td>
</tr>
</tbody>
</table>

Note. Analyses adjust for all Wave 1 covariates.
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