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Predicting Drinking Onset with Discrete-Time Survival Analysis in Offspring from the San Diego Prospective Study

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Abstract

Previous research has shown that an early onset of drinking is associated with a range of problematic drinking outcomes in adulthood. However, earlier drinking is also linked to additional characteristics that themselves predict alcohol problems including male gender, a family history (FH) of alcoholism, age, race, parental alcoholism, depression symptoms, prior drug use, and conduct problems. This study tested the relationship between the age of first drink (AFD) and a range of risk factors that predict the onset of alcohol use. Participants were offspring from the San Diego Prospective Study (SDPS) who were at least 15 years old at the time of their most recent interview (n=147). Discrete-time survival analysis (DTSA) was used to relate multiple characteristics to the hazard function of alcohol onset across a relevant age range. The results demonstrated the predicted relationships to AFD for conduct problems, male gender, prior marijuana use, and a FH of alcoholism, even when these characteristics were estimated together. Furthermore, an interaction occurred such that offspring with both conduct problems and marijuana use were at substantially higher risk for alcohol use onset during this time period than would be predicted from the effect of these two risk factors alone. However, age at interview, ethnicity, parent education, and depressive symptoms did not predict the pattern of onset of drinking. Implications for future research and prevention efforts are discussed.

Keywords

drinking onset; conduct problems; survival analysis; family history

1. Introduction

Most men and women in the United States have ever consumed alcohol, with the usual onset of drinking between ages of 14 and 16, a figure that is slightly later in girls than boys (Johnson & Mott, 2001; Sartor et al., 2006; York, 2004). While results vary with the population and methods used, about 5% to 10% of adolescents have had their first drink by age 12; 25% to 35% have consumed alcohol by age 13 or 14 (i.e., in the 8th grade); 45% to 60% by age 15 or 16 (10th grade); and as many as 75% by the time of graduation from high school (Donovan et al., 2004; Faden, 2006; Faden & Fay, 2004; Johnson et al., 2008).

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An earlier age of onset of drinking has been reported to correlate with subsequent higher levels of alcohol consumption, a greater risk for alcohol problems, and a faster progression between the first use and the development of alcohol abuse or dependence (also known as alcohol use disorders, or AUDs) (Dawson et al., 2008; DeWit et al., 2000; Grant & Dawson, 1998; Hingson et al., 2008; York, 2004). However, not all studies agree that it is the earlier age of first drink (AFD) that is the key variable predicting future alcohol problems, with some reports raising the possibility that earlier drinking and higher risks for alcohol and other substance-related problems might each reflect a third characteristic, such as conduct problems, that is the major predictor of the alcohol outcomes (King & Chassin, 2007; Labouvie et al., 1997; Labouvie & White, 2002; McGue et al., 2001b; Prescott & Kendler et al., 1999). Such pre-existing conduct problems are themselves associated with an almost 2.5-fold increased probability of an earlier onset of drinking, and these same characteristics predict a 3-fold or higher risk for alcohol-related problems (Assanangkornchai et al., 2002; Kamarajan et al., 2006; McGue et al., 2001a, 2001b; Slutske et al., 1998; Tarter et al., 2004).

Additional phenomena may also relate to both an earlier AFD and a greater future risk for heavy drinking and alcohol-related problems. These include male gender, prior substance use, as well as correlates of conduct problems such as fights, school problems, and related phenomena (Hingson et al., 2006; Rhee et al., 2003; Slutske et al., 1998; Young et al., 2000). Other studies have also raised the possibility of the importance to both AFD and future alcohol problems for childhood histories of depression and anxiety, heavier drinking among peers and a family history (FH) of AUDs (Kuperman et al., 2005; McGue et al., 2001a; Sartor et al., 2006; Schuckit et al., 2009a). One aspect of FH, genetic factors, may be a more robust contributor toward heavier drinking than toward AFD itself (Hingson et al., 2006; Rhee et al., 2003).

Thus, the relationship between an earlier AFD and future alcohol related problems is complex, and is best understood through prospective studies that consider multiple predictors of the onset of drinking (Kuperman et al., 2005; Sartor et al., 2006; Warner et al., 2007). The current analyses evaluate the relations between AFD and several additional characteristics that by themselves are likely to be associated with future heavy drinking and alcohol problems, using offspring from the San Diego Prospective Study (SDPS). The central hypothesis is that the relationship to AFD for factors that also enhance the risk for later alcohol problems (gender, FH of AUDs, conduct problems, prior substance use and depression symptoms) is robust enough that all these characteristics must be considered along with AFD in predicting alcohol outcomes. In addition, we are also interested in determining whether interaction effects exist among the set of predictors of AFD.

2. Methods

2.1 Participants

The subjects are offspring of probands from the SDPS who, to optimize the number of subjects likely to have experience with alcohol, were at least 15 years old at the time of their most recent interview. The fathers of these offspring had originally been selected between 1978 and 1988 as 18- to 25-year-old drinking Caucasian men who responded to questionnaires mailed to randomly chosen students and non-academic staff at the University of California, San Diego (Schuckit & Gold, 1988; Schuckit & Smith, 2000). All 453 original subjects were evaluated using an initial baseline interview and followed up at 10 years (T10 with 99% retention), 15 years (T15 with 97%), 20 years (T20 with 95% retention) and during the ongoing 25 year followup (Schuckit & Smith, 1996, 2000). As subjects married and had children, these relatives entered the protocol, with participation rates that also exceeded 95% at the 20-year follow-up.

At both the T20 and the ongoing T25 followups of these families, offspring age 12 or older were evaluated using questions from the adolescent version of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) instrument developed by the Collaborative Study on the Genetics of Alcoholism (COGA) (Bucholz et al., 1994; Hesselbrock et al., 1999). These included questions on alcohol, drug, and mental health histories. Overall, the SSAGA has a 1-week reliability of .86 for alcohol-related items and similar validities compared with a second standard interview (Bucholz et al., 1994; Hesselbrock et al., 1999; Kuperman et al., 2001a). Note that in order to use data gathered at the age closest to the onset of drinking, the offspring data used in these analyses were from their first interview after drinking occurred (T20 or T25).

2.2 Measures

Family History (FH) of Alcoholism—Diagnoses for the biological parents of the offspring (i.e. the proband and his spouse) were assessed for DSM-III-R alcohol abuse and dependence from an interview similar to the SSAGA (American Psychiatric Association, 1987). FH was coded as a dichotomous variable where 23% of the offspring had at least one parent meeting criteria for an alcohol use disorder as determined from personal interviews with parents as part of the SDPS protocol.

Sex—The dichotomous variable indicating male offspring.

Age—The continuous variable of chronological age at time of interview.

Ethnicity—The dichotomous variable indicating both parents Caucasian.

Parent Education—The continuous variable reflecting the highest level of education, measured in years, completed by either parent.

Depression Symptoms—The number of depressive symptoms endorsed on the SSAGA.

Conduct Problems—The number of DSM-III-R conduct disorder criteria endorsed on the SSAGA.

Prior use of Tobacco/Marijuana/Illicit Drugs—Self-report data on the age of onset of any lifetime use for these substances was used to determine whether other substance use preceded the onset of alcohol use, which was coded as a dichotomous variable indicating those with substance use that preceded alcohol use. Note that marijuana (THC) and all other illicit drugs were separate categories.

Onset of Alcohol Use—The primary outcome of interest was the actual age at which the offspring reported first having a drink of alcohol (more than a sip).

2.3 Data Structure

In order to estimate the DTSA models in a latent variable framework, the raw data were prepared as outlined by Muthen & Masyn (2005). Since there was considerable age heterogeneity at each measurement wave, the hazard/survival curves were modeled as a function of the age of first drink, rather than of measurement occasion. A total of 12 binary time-specific event indicators were constructed to reflect yearly intervals between the youngest reported age of alcohol onset (12 years) and the oldest reported age of alcohol onset (22 years) for this subsample. The subject was considered to have experienced the event of interest at the age they reported drinking at least one full drink of alcohol for the first time, designated by a “1” score on the binary time-specific event indicators for the corresponding age; prior ages were scored “0” to reflect no event, and subsequent ages were treated as missing due to having

experienced the event. Right-censoring occurred when a subject did not report alcohol onset by their most recent assessment. Such subjects would have “0” scores for all ages that included up to their age at most recent interview, with any subsequent ages being considered missing with a score of “99”. The inclusion of younger subjects (15–17 years old) who have lifetime abstinence provides valuable information about how the predictor variables relate to abstinence at these earlier ages, but their inclusion also affects the form of the hazard function in Figure 2.

2.4 Data Analytic Approach

The relations between baseline covariates and the outcome of alcohol onset were evaluated with a series of discrete-time survival analyses (DTSA) conducted using a latent hazard function representing the event time distribution. The discrete-time hazard is the conditional probability that an adolescent will have an alcohol onset in a time period, given that they did not report alcohol use in an earlier time period (Singer & Willett, 2003). The DTSA model has been described as “a number of logistic regressions fitting the incremental probability of survival” (page 185; Asparouhov et al., 2006), which informs both whether and when an event of interest (here, alcohol onset) occurs. A related indicator of event occurrence is the survival function, which is the sample’s cumulative probability of not reporting alcohol use over time, and which is expressed as a function of the hazard function (Muthen & Masyn, 2005). This survival function has been shown to approximate the Cox regression model used in traditional continuous time survival models, and is preferred when the data are categorical and the number of categories is less than 20 (Asparouhov et al., 2006).

Conventional DTSA can be estimated within the general latent variable framework that corresponds to a single-class latent class analysis with binary time-specific event indicators (Muthen & Masyn, 2005). The first step was to fit an unconditional survival model that included only the 12 binary time-specific event indicators for the occurrence of alcohol onset across adolescence. The constant hazard assumption was then evaluated by comparing the unconditional survival model, which allowed the hazard rate to vary across time, to a model that constrained the hazard rate to equality across ages using a likelihood-ratio test (LRT) based on the model deviance statistics.

The next step evaluated the proportionality assumption for each of the covariates to determine whether the covariate effects were identical across all time points. Proportionality was assessed separately for each covariate by comparing a model with time-varying covariate effects to a model that constrained the covariate effects to equality across time. If the unconstrained covariate model fit better than the constrained model (based on LRTs), the covariate violated the proportionality assumption.

A full multivariable model including all the covariates was then estimated such that the latent hazard function with its 12 binary time-specific event indicators was regressed on the set of time-invariant covariates (all factor loadings fixed to 1.0 to reflect proportional covariate effects across time), and any covariate that violated the proportionality assumption had freely estimated regression paths on the binary time-specific event indicators to allow for time-varying effects. The results of this model allowed interpretation for each individual covariate effect on alcohol onset after adjusting for all other covariates.

Follow-up analyses evaluated a series of statistical interactions in the DTSA model to determine whether the interaction effects exist. Following the procedure of Singer & Willett (2003), these analyses were restricted to examine interactions among only those risk factors shown to be significant from the previous models. These interactions were tested by creating a cross-product term and adding it to a model that included the main effects of the covariates comprising the interaction. This model was compared to a model excluding the cross-product

variable using a LRT to determine whether the inclusion of each interaction significantly improved overall model fit. All models were run in Mplus v5.1 (Muthen & Muthen, 1998–2007). In order to increase confidence in the final maximum likelihood values, automatically generated starting values with random perturbations (100 random sets of starting values with 30 full optimizations) were used for all models described below.

3. Results

Table 1 reports descriptive statistics for all key variables generated from the 147 offspring of the original SDPS probands who were at least 15 years old when interviewed at the most recent assessment. About a quarter of the sample had a parent who ever met criteria for an AUD, the majority of offspring had two Caucasian parents, and most had at least one parent with a bachelor's degree. About half of the offspring were male with a mean age of about 18 years at the time of interview. The average number of depression symptoms ever experienced was .23 (4% endorsed any symptoms), and the average number of conduct problems was 1.29 (28% reported one such problem and 30% two or more). There were low levels of use of tobacco (7%), marijuana (15%) and other illicit drugs (8%) that preceded drinking onset.

In this sample, 68% reported using at least one standard drink of alcohol by the time of their most recent interview. The mean age of alcohol onset was 15.8 years ($sd=2.13$) for those offspring with lifetime alcohol use. Figure 1 shows the number of offspring reporting alcohol onset for each age: 34% of 15-year olds, 41% of 16-year olds, and 74% of 17-year-olds reported lifetime alcohol use; only one offspring in the 18-to-26 range reported never drinking alcohol.

3.1 Unconditional Survival Model

The unconditional hazard function in Figure 2 illustrates a gradual decrease in survival rates of alcohol onset during adolescence, with a steep decline from 14–18 years. Approximately 32% of offspring reported not having tried alcohol by the time of their most recent interview, and this is indicated in Figure 2 as a plateau after age 21. This graph, therefore, reflects the inclusion of younger subjects in the sample, a step incorporated into DTSA to allow for the use of covariate data from younger abstainers and drinkers to predict the pattern of drinking onset. However, the result is a hazard function that, reflecting the entire sample of subjects, never indicates directly that at older ages (e.g., 21, 22, etc.), all individuals at that age are drinkers. The latter requires observation of Figure 1 where the proportions of abstainers and drinkers at each age are listed. Of course, if the data are restricted to only the 100 offspring who reported a lifetime use of alcohol, the hazard function would demonstrate a steeper decline that extended to 0% abstainers in the early 20s.

The constant baseline hazard assumption was next evaluated to assess whether the hazard rate varied significantly across this time frame (i.e. across ages). The unconditional hazard model (with time-varying hazard rates) was found to significantly improve fit compared to a model that constrained the hazard rate to equality ($\chi^2 = 77.55$, $df=9$, $p<.001$). Thus, the constant baseline hazard assumption was rejected and the hazard function was allowed to vary across age in all models.

3.2 Univariate Covariate Effects

The evaluation of whether the effects of predictors on drinking onset were equivalent across all time points for each of the covariates (i.e. FH, conduct problems, prior drug use, etc.) revealed that no covariate resulted in a significantly better model fit when allowed to vary across time (analyses not shown). Therefore, the effect of each predictor was estimated to be constant, or proportional, across time.

The parameter estimates (“Est” in the table which represent the log hazard odds) and corresponding hazard odds ratios (with 95% confidence interval) for the univariate effects of each covariate on the hazard probabilities of drinking onset are listed in Table 2 under the Univariate Effects heading. Three predictors were significant in the expected direction: having an alcoholic parent was associated with drinking onset, and higher levels of conduct problems and prior use of marijuana were both associated with alcohol use onset across this age range. Offspring gender was only marginally significant such that males were more likely than females to have alcohol onset ($p=.07$).

3.3 Full Multivariable Survival Model

Next, a simultaneous evaluation of all predictors in a full multivariable survival model was carried out as shown regarding parameter estimates (log hazard odds) and hazard odds ratios (hOR) in Table 2 under the Full Model heading. After adjusting for all other covariates, the FH of AUDs, conduct problems, and prior marijuana use remained significant predictors of alcohol onset, and male gender became statistically significant. Interpreting the significant hazard odds ratios in the full model, offspring with an FH of alcoholism ($\text{hOR}=2.01$), males ($\text{hOR}=1.79$), more conduct problems ($\text{hOR}=1.28$), and prior marijuana use ($\text{hOR}=2.72$) all corresponded to higher hazard probabilities of lifetime alcohol use across the age range examined here.

3.4 Interaction Effects among Risk Factors

A set of follow-up analyses were conducted to determine whether there were interactions among the predictors. Recognizing the large number of potential interactions that might be tested, and to avoid Type I error with this modest sample size, an *a priori* decision was made to focus only on the significant covariate effects from the full model. A total of six 2-way interactions were created from the significant covariates of FH of AUDs, offspring gender, conduct problems, and prior THC use. One interaction yielded a significant likelihood ratio test (LRT) when a model with the cross-product and constituent main effects was compared to a model excluding the cross-product: Conduct problems by prior THC use ($\chi^2 = 4.92$, $\text{df}=1$, $p=.03$; $b=.39$, $p=.006$). Thus, allowing for this interaction effect improved model fit and there was evidence to reject the linear additivity assumption for these covariates.

To better visualize this interaction effect, a series of tables (not shown) of the onset of alcohol use from the earliest age of drinking onset in this sample (12 years) to age 22 was created using dichotomized subgroups of the main effect variables. For these analyses, prior THC use was still treated as a binary category (15% yes vs. 85% no), while conduct problems were dichotomized based on <2 problems (70%) vs. $2+$ problems (30%). The cutoff of two conduct problems was selected because 28% of the sample reported one conduct problem, most commonly fights (33%), skipping school (28%), and stayed out/sneaked out of the house at night (27%), any one of which alone may only be marginally indicative of a significant behavior problem. For this interaction, four subgroups were created from the variables described above, with the survival function then calculated for each subgroup individually and graphed over time.

Figure 3 describes the onset of alcohol use over time by the four combinations of conduct problems and prior THC use described earlier. As expected, the survival rate (i.e. not having drank alcohol) was highest for offspring with no prior THC use and <2 conduct problems (39% of subgroup). The survival rate decreased to $\sim 30\%$ for the subgroups with one of these risk factors present. Most notably, the 12 offspring with both prior THC use and at least 2 conduct problems all had an alcohol onset by age 16, potentially indicating that the effect of THC use on the hazard function of drinking onset occurred primarily in offspring with conduct problems. The rates of alcohol use onset by age 22 across groups were significantly different ($F(3,143)$

=3.04, $p=.03$), and post-hoc comparisons using the Games-Howell procedure (due to unequal group sizes) found significant group contrasts between the highest risk group and the two lowest risk groups only. While one potential reason for these group differences could be age heterogeneity across the subgroups, post-hoc analyses found that these groups were not significantly different in terms of offspring age at time of interview (Welch $F(3)=1.41$, $p=.26$).

4. Discussion

The current study offers support for our primary hypotheses that the age of first drink is associated with characteristics that themselves predict future alcohol problems. This is one of the few studies to prospectively evaluate multiple risk factors that are likely to impact on how the AFD performs in predicting subsequent alcohol problems. The data demonstrated that several risk factors operated relatively independently of each other in predicting the AFD (e.g. sex, FH of AUDS, prior marijuana use, and conduct problems) and all need to be considered together when evaluating whether AFD predicts later alcohol problems and AUDs.

For example, the relationship between AFD and alcohol outcomes should always be evaluated in the context of conduct problems, as these clearly predicted the occurrence of drinking onset across adolescence in offspring from the ongoing San Diego Prospective Study. Additional studies support a strong relationship between such problems early in life and later alcohol problems (i.e. Slutske et al., 1998; McGue et al., 2006). In the current data, the relationship of conduct problems to AFD remained significant even after controlling for other robust risk factors including the FH of AUDs, male gender, and prior substance use. Also, prior THC use related to AFD even when considered in the context of conduct problems. Furthermore, in these analyses, offspring with both conduct problems and THC use were at greater risk for experiencing alcohol use onset during this developmental period than would have been predicted from either characteristic alone. Therefore, this interaction adds important information to the current literature.

The pattern of alcohol onset in the survival curve (Figure 2) illustrates close approximations of the rates obtained by large-scale epidemiological studies such as Monitoring the Future (MTF) (Johnston et al., 2008). The mean age of alcohol onset for those who reported drinking in the current sample was ~16 years which roughly corresponds to the 58% lifetime alcohol use rate reported for 10th graders in the 2008 MTF report. In addition, the probability for alcohol onset increased with age, and only one subject in the 18+ age range denied ever trying alcohol. In viewing these results shown in Figure 2, it is important to remember that DTSA includes offspring who have not yet passed through the full age of risk for drinking onset. This allows us to examine how predictors relate to drinking onset at each age for all subjects regardless of their lifetime alcohol use. However, the inclusion of these younger respondents impacts the slope of the survival curve to plateau at about age 21 across time.

The finding that lifetime conduct problems, FH of AUDs, prior THC use, and male gender were each individually associated with drinking onset is also consistent with previous research (Kuperman et al., 2001a, 2001b; McGue et al., 2001a, 2001b; Prescott & Kendler, 1999). This further highlights the importance of controlling for these robust risk factors when examining the relation between AFD and drinking problems later in life. Therefore, it may be premature to conclude that a delay in the AFD by itself could decrease risk for development of AUDs (Grant & Dawson, 1998). While this is a logical step to consider, the approach should be tested in future longitudinal studies to provide greater support for such causal inferences. Continued follow-ups in the San Diego Prospective Study will allow for further testing of whether AFD is a unique predictor of alcohol-related outcomes in early adulthood. Such information will also be critical to test whether AFD mediates the effects of the risk factors identified here

(conduct problems, FH, THC use, and male gender) on a range of substance use outcomes in early adulthood.

The significant interaction between conduct problems and THC use identified a high-risk group of adolescents who all reported drinking by age 16 and who may be at especially high risk for future alcohol and drug problems. This finding is consistent with the notion of a potentiator model in which one risk factor increases the probability of an outcome especially when it occurs in the presence of another risk factor (Kupersmidt et al., 1995). As such, future prevention efforts may benefit from targeting multiple problem behaviors in adolescence to minimize impact on future substance use. It should be noted that conduct problems were assessed lifetime and not prior to drinking onset, therefore interpretations of temporal causality between the two behaviors are limited. However, related traits such as impulsivity and disinhibition are usually seen early in life and likely precede the AFD (Slutske et al., 1998; Kuperman et al., 2001a).

Some hypothesized predictors of drinking onset did not perform as expected in this sample. Regarding prior reports of the relation of race to an earlier AFD (Dawson et al., 2008), the SDPS is largely Caucasian and this demographic breakdown may have hindered finding any racial/ethnic differences. As for lower parent education as a risk factor, the current sample was also fairly homogenous, with most families having parents with higher education reflective of the original selection criteria. Finally, these subjects reported low rates of depressive symptoms, tobacco use, and other illicit drug use which may have reflected their relatively young age. Significant effects of these characteristics may emerge with increased sample size and the availability of an older age range in future data collections. This issue may be especially relevant to the performance of tobacco use as a predictor of drinking onset, as <10% of this sample had previous smoking experience. The relatively low rate of smoking might also reflect the relatively high education levels of the families in the sample.

In interpreting the current results, several caveats should be noted. First, the sample was relatively small which may have obscured finding additional characteristics with small main effects and interactions. A related issue was that not all possible interactions were tested due to the *a priori* decision to limit such evaluations to covariates with significant main effect in order to minimize problems with Type I error, a step that may have increased Type II error resulting in unidentified significant interaction effects. Third, as noted above, the unique attributes of the SDPS generates caution regarding generalizing these findings to other populations. Finally, a larger sample of subjects would have greater power to detect smaller effect sizes that were not detected in the current study. Furthermore, information on additional predictors of substance use (e.g., drinking among peers, alcohol expectancies) was not available in the younger group reported here since such items were not added to the protocol until the most recent assessments of these offspring and their families.

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Figure 1.

The number of offspring of each age in this sample, and the percent reporting lifetime alcohol use (black=yes, white=no) within each age category (n=147).

Figure 2.
Percent never drank alcohol across age for the total sample of offspring (n=147).

Figure 3.

Percent never drank alcohol across age by prior THC/conduct problem subgroups. Groups were created by prior THC categories (yes/no) and a split of conduct problems (<2 problems vs. 2 + problems).

Table 1

Descriptive Data for Study Variables (n=147).

	Mean/%	Std Dev	Range
Family history (FH) of alcoholism	23%	-	-
Male	46%	-	-
Caucasian	88%	-	-
Age at interview	17.7	2.65	15–26
Parent education (# years)	16.8	1.78	12–20
Depression symptoms (max lifetime)	0.2	1.21	0–9
Conduct problems (max lifetime)	1.3	1.59	0–7
Tobacco use before alcohol use	7%	-	-
THC use before alcohol use	15%	-	-
Illicit drug use before alcohol use	8%	-	-

Table 2

Predicting the Hazard Function of Offspring Drinking Onset (n=147).

	Univariate Effects		Full Model	
	Est (SE)	OR (95% CI) ^a	Est (SE)	OR (95% CI) ^b
FH of alcoholism	.82 (.25) ^{**}	2.27 (1.39–3.71)	.70 (.26) ^{**}	2.01 (1.21–3.35)
Male	.43 (.24) [†]	1.54 (.96–2.46)	.58 (.27) [*]	1.79 (1.05–3.03)
Caucasian	-.39 (.36)	.68 (.33–1.37)	-.47 (.41)	.63 (.28–1.40)
Age at interview	-.09 (.06)	.91 (.81–1.03)	-.07 (.07)	.93 (.81–1.07)
Parent education	-.07 (.08)	.93 (.80–1.09)	-.07 (.08)	.93 (.80–1.09)
Depression symptoms	-.01 (.06)	.99 (.88–1.11)	.02 (.08)	1.02 (.87–1.19)
Conduct problems	.28 (.07) ^{***}	1.32 (1.15–1.52)	.25 (.06) ^{***}	1.28 (1.14–1.44)
Prior tobacco use	-.22 (.44)	.80 (.34–1.90)	.53 (.76)	1.70 (.38–7.54)
Prior THC use	.79 (.36) [*]	2.20 (1.09–4.46)	1.00 (.32) ^{**}	2.72 (1.45–5.09)
Prior illicit drug use	-.26 (.39)	.77 (.36–1.66)	-.87 (.71)	.42 (.10–1.68)

[†] p<.10.^{*} p<.05.^{**} p<.01.^{***} p<.001.

Note. Est= log odds of drinking onset; SE= standard error; t= Est/SE (t-statistic); OR= odds ratio; CI= confidence interval