UC San Diego

UC San Diego Previously Published Works

Title

The Relationship between Early Alcohol Use Behaviors and Adolescent Pubertal and Psychosocial Development: A Latent Growth Analysis

Permalink https://escholarship.org/uc/item/5t46001n

Journal Substance Use & Misuse, 56(6)

ISSN

1082-6084

Authors

May, AC Aguinaldo, LD Tan, R <u>et al.</u>

Publication Date 2021-05-12

DOI 10.1080/10826084.2021.1899231

Peer reviewed



HHS Public Access

Author manuscript Subst Use Misuse. Author manuscript; available in PMC 2022 March 23.

Published in final edited form as:

Subst Use Misuse. 2021; 56(6): 861-870. doi:10.1080/10826084.2021.1899231.

The Relationship between Early Alcohol Use Behaviors and Adolescent Pubertal and Psychosocial Development: A Latent Growth Analysis

A.C. May^{a,b,*}, L.D. Aguinaldo^b, R. Tan^b, K.E. Courtney^b, J. Jacobus^{a,b}

^aSan Diego State University/University of California, San Diego Joint Doctoral Program in Clinical Psychology, San Diego, CA, USA

^bDepartment of Psychiatry, University of California, San Diego, San Diego, CA, USA

Abstract

Objective: The present longitudinal study examines how age of alcohol initiation and regular use (weekly drinking for 6 months) relates to adolescent physiological development, social behaviors, psychological functioning, and substance use patterns.

Method: Data are drawn from a prospective sample of 295 youth (42% female) who completed a 15-year longitudinal study. The current investigation uses data collected at 4 timepoints from ages 12-19.

Results: Latent growth modeling revealed earlier age of alcohol initiation is associated with 1) a more advanced stage of pubertal development, more self-reported dating experience, and greater externalizing behaviors at ages 12-13 (study entry); 2) a slower rate of change in pubertal development; and 3) greater rate of increase in externalizing and internalizing symptoms from ages 12 to 19. These relationships were not moderated by gender.

Conclusion: Early alcohol initiation appears to be associated with early onset pubertal development and dating behaviors. Over time, early alcohol use behaviors may delay pubertal development while exacerbating psychological risk behaviors (i.e., externalizing and internalizing behaviors). These findings suggest the importance of delaying alcohol initiation and may be beneficial for improving existing adolescent substance use prevention efforts.

Keywords

adolescence; alcohol initiation; regular alcohol use; pubertal development status; dating; externalizing; internalizing

Introduction

Adolescence is a time of significant cognitive, emotional, and physical growth (Choudhury et al., 2006; Chulani & Gordon, 2014; Steinberg, 2005; Yurgelun-Todd, 2007). During this

^{*}Corresponding Author: J. Jacobus, Department of Psychiatry, University of California, San Diego, 9500 Gilman Drive, San Diego, CA, 92093, jjacobus@health.ucsd.edu.

The Authors declare that there is no conflict of interest.

period of development, exposure to common environmental risk factors (e.g., alcohol, drugs, substance-using peer groups) can influence an individual's developmental and mental health trajectory (Danzo et al., 2017; Feldstein Ewing et al., 2014; Spear, 2000, 2018; Yurasek et al., 2019). According to *Monitoring the Future*, an annual survey conducted by the National Institute of Drug Abuse (NIDA; Johnston et al., 2019), alcohol consumption is common among adolescents with approximately 52% of 12th graders reporting alcohol use in the past year. Alarmingly, 19% of 8th graders report having consumed alcohol within the past year and 8% of 8th graders report consuming alcohol in the past month (Miech et al., 2019). Given the high rates of alcohol use during this developmentally significant period, it is critical to discover the potential adverse psychological and social outcomes associated with this use.

Early life indicators of future substance use problems include frequency and severity of use (Duncan et al., 1997; Moss et al., 2014), presence of diagnostic criteria for abuse or dependence (Hanson et al., 2011; Moss et al., 2014), substance use treatment status (Aarons et al., 1999), and age of initiation (Donovan & Molina, 2011; Hingson et al., 2006). Age of *any* alcohol initiation is one of the more commonly investigated risk factors for the development of heavy substance use and related use disorders (Morean et al., 2014) with early findings suggesting that youth who begin drinking alcohol before age 14 are 1.78 times more likely to develop an alcohol use disorder in young adulthood (ages 19-26; Hingson et al., 2006); however, subsequent findings have been inconsistent.

A less frequently studied characteristic of alcohol use, albeit potentially important marker of progression to problematic use and alcohol use disorder, is the age at which one begins to drink regularly. Despite the potential significance of this construct, the definition of regular drinking has varied across studies. For example, in an original research study, Sartor and colleagues (2016) defined age of onset for regular drinking as drinking at least once a month for 6 consecutive months or at least once a week for 8 consecutive weeks, compared to the requirement of drinking a "few times" a month or more as used by Poelen and colleagues (2007). These differences in measurement likely cloud our understanding of the implications of regular drinking onset in adolescence. Despite these variations in definition, multiple quantitative studies have found early onset of regular alcohol use to be a risk factor for future use problems (Grant et al., 2006; Guttmannova et al., 2011). More specifically, initiation of regular alcohol use, defined as three or more times per month, at any age before 21 was found to be associated with an increased rate of dependence in adulthood, while individuals who initiated regular alcohol drinking by the age of 14 did not appear to be at any greater risk for alcohol dependence in adulthood than those who initiated regular use at an older age but still before turning 21 (Guttmannova et al., 2011). For our study, age of drinking regularly is operationalized as weekly drinking for 6 months which aligns with the lowest threshold of the National Institute of Alcohol Abuse and Alcoholism and US Department of Health and Human Services' definition of regular moderate drinking (up to one drink per day each week, on average; U.S. Department of Health and Human Services, 2015).

Early initiation of alcohol use may impact other domains important to healthy development during adolescence. Various original research studies have demonstrated a reciprocal

relationship between substance use, emotional distress, and association with deviant peers (Boyd et al., 2018; Deas, 2006; Malmberg et al., 2013). For example, early alcohol use may lead to increased socialization with older peers and exposure to more adult-like social scenarios at a younger age. This may further increase the consumption of alcohol and accelerate the initiation of other potentially risky behaviors such as sexual intercourse and/or the use of additional substances (Calvert et al., 2010; Stueve & O'Donnell, 2005). Relatedly, externalizing behaviors, such as oppositional, aggressive, impulsive, disruptive, and rulebreaking behaviors, have been found to significantly predict alcohol use disorder in adulthood (Farmer et al., 2016). In contrast, alcohol use at age 12 has been found to predict future internalizing symptoms, such as those associated with depression, anxiety, rumination, and distress, during adolescence (Jun et al., 2015). Research has also suggested a reciprocal relationship between alcohol consumption and physiological factors such as pubertal development; early alcohol consumption may delay pubertal onset (Dees et al., 2017) and stage of pubertal development at the time of drinking initiation may affect the number of drinks consumed per month in young adulthood (Blomeyer et al., 2013). Taken together, these findings suggest that early adolescent alcohol use may have significant physiological, psychological, and social consequences for youth prior to entering young adulthood.

The present longitudinal study aims to examine how age of any alcohol initiation and regular use initiation relates to adolescent development from ages 12-19, including physiological development, social behaviors, psychological functioning, and substance use patterns. Specifically, the study aimed to examine the effects of age of alcohol initiation and regular alcohol use within a prospective sample of 295 youth assessed at four independent time points. It was hypothesized that an earlier age of any alcohol use initiation and earlier age of regular use initiation would be associated with 1) more advanced pubertal development, more dating experience, and greater externalizing and internalizing behaviors, from ages 12-13 (Time 1, study entry) and 2) greater change in their physiological development, dating experiences, internalizing and externalizing behaviors, and number of drinking days over time (from Time 1, ages 12-13 to Time 4, ages 18-19). Additional exploratory analyses were conducted to determine whether these relationships differed between males and females. This is the first study to examine the effect of early alcohol initiation and regular use across multiple domains of adolescent development within a large prospective sample followed over the course of 8 years.

Methods

Participants

This study used a prospective sample of 295 youth from a completed 15-year longitudinal project (Courtney et al., 2018; Infante et al., 2019). Data for the present analyses were collected at ages 12-13 (Time 1), 14-15 (Time 2), 16-17 (Time 3), and 18-19 (Time 4). At study enrollment, youth were largely substance-use naïve, reporting little to no experience with alcohol or other drugs (average lifetime alcohol use episodes = 1.82 [SD = 9.29, Median = 0]). Youth were recruited from San Diego area schools, and the study protocol was executed in accordance with the standards approved by the University of California, San

Diego Human Research Protections Program. Informed consent and assent, eligibility criteria information, and background information were obtained from the adolescent and a biological parent at Time 1 and each subsequent time point as appropriate. Participants who had significant experience with alcohol or drugs and any history of a neurological or Axis I disorder were excluded (Squeglia et al., 2017).

Procedures

At Time 1, a comprehensive clinical and substance use interview was administered. Interviews were conducted annually to obtain more accurate information on age of initiation, age of regular use initiation, and cumulative use for alcohol and other substances. At study enrollment, all participants were considered control participants with fewer than 11 lifetime drinking occasions, and no more than one drink consumed per occasion (97% of the sample endorsed no prior experience with alcohol). Self-report questionnaires were also administered at each time point to gather information regarding pubertal development status, dating experience, and internalizing/externalizing behaviors.

Measures

Substance use measures—The Customary Drinking and Drug Use Record (CDDR; Brown et al., 1998) was administered to the youth at each time point to obtain information on the pattern and severity of alcohol and drug use. The CDDR gathers information regarding age of onset, quantity and frequency of lifetime and recent (past year) use, withdrawal/hangover symptoms, and substance use disorder criteria. The primary alcohol use variables, age of alcohol initiation (i.e., age at which the youth had their first full drink of alcohol) and age of regular alcohol use (weekly drinking for 6 months), were assessed at each time point using the CDDR and composited with previous time points to ensure there were no inconsistencies in the database.

Background information—Trained research assistants conducted demographic interviews to obtain information about the participant's gender, age, race, grade in school/ education status, and dating status (response options: no, never dated; no, not now; yes, only in group activities; yes, rarely [once every few months]; yes, occasionally [every couple of weeks]; yes, regularly [every week]; yes, steady boyfriend/girlfriend).

Pubertal development—The Pubertal Development Scale (PDS; Petersen et al., 1988) is a 5-item self-report measure administered to youth to assess pubertal development and is customized for each gender. Items ask participants to self-assess growth, body hair, and skin changes. Males are asked about facial hair, growth, and voice changes, and females are asked about breast development and menarche. Items are rated on a 4-point Likert scale ranging from 1 ("has not yet begun") to 4 ("seems completed"), and the total score is calculated by taking an average of the 5 items. Higher values indicate more advanced pubertal development. The PDS has been shown to be both reliable and valid (Petersen et al., 1988), and scores from the PDS correlate well with physicians' Tanner ratings (.61-.67), as well as parents' and other examiners' Tanner ratings (.75-.87; Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987).

Internalizing and externalizing behaviors—Measures of internalizing and externalizing behaviors were obtained using the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2004), the Youth Self Report (YSR; Achenbach & Rescorla, 2004), and Adult Self Report (ASR; Achenbach & Rescorla, 2003). Parents completed the CBCL for youth ages 12-14, while youth ages 15-18 completed the YSR independently and the ASR upon turning age 19. This well validated assessment system has shown excellent reliability and validity between assessment instruments and cross-informant agreement (e.g., child/parent) on reports of externalizing behaviors show large effect sizes (Achenbach, McConaughy, & Howell, 1987; Achenbach & Rescorla, 2001).

Data Analysis

Gender differences in demographic variables were examined in the R statistical program (R Core Team, 2014) using the furniture package (Barrett & Brignone, 2017). Continuous variables were examined using t-tests, and categorical variables were examined by chi-square tests of independence. Correlations were also conducted between all variables of interest to identify any potential sources of collinearity (Figure 1).

Two series of five latent growth models (LGM) were fit to the data using MPlus 7.3 (Muthén & Muthén, 2017) to examine the relationship between each predictor of interest (ages of onset for alcohol initiation or regular use) and five outcome variables (self-reported pubertal development status, dating experience, externalizing and internalizing symptoms, average number of drinking days per year) measured from ages 12-19 (Time 1-4; See Table 1). The Benjamini-Hochberg (1995) procedure was applied within each experimental family (i.e., age of onset for alcohol initiation and age of onset for regular use of alcohol) to reduce the false discovery rate and protect against type I errors. This adjustment was conducted in the R statistical program (R Core Team, 2014) using 'p.adjust' (Wright, 1992). Unadjusted p-values are reported for each model with indication of whether or not the adjusted p-value fell below an alpha level of .05 and was therefore deemed to be significant.

In LGMs, repeated measures of data indicate latent variables that describe mean trajectories over time (Duncan & Duncan, 2004). The loadings of the outcome variables (OVs) were set to 0 at Time 1, 1 at Time 2, 2 at Time 3, and 3 at Time 4 to model growth over time. Therefore, the intercept of each model represents the estimate of the OV at Time 1, while the slope represents the corresponding change in the OV for a 1-unit change in the predictor variable (PV). Good model fit was based on the following cut-offs for each fit statistic: chi-square p-value > .05, comparative fit index (CFI) .90, Tucker Lewis index (TLI) .95, and root-mean-square error of approximation (RMSEA) < .08 (Hooper et al., 2008). Race, ethnicity, and socioeconomic status were examined as potential covariate; however, they yielded no significant results and were therefore removed from the analyses. Gender was included as a covariate in all models allowing for a non-hypothesis driven examination of gender differences on the developmental OVs. The interaction of gender and each PV (initiation and regular use) was also examined; however, there were no significant results for this variable (ps > .05), thus the interaction term was removed and will not be reported.

For models examining age of alcohol initiation, missing data are due to participants not coming in for an assessment during the specified age range. The models examining age of

regular use include n=105 participants, only those who initiated regular drinking during the study period. The 'finalfit' R package (Harrison et al., 2020) was used to determine if any of the developmental OVs systematically differed as a function of either of the PVs (i.e., age of drinking initiation, age of regular drinking) or gender. The results indicated that there was no significant pattern of missingness for externalizing symptoms, internalizing symptoms, or average number of drinking days. However, the results indicated that significantly more males than females had missing data for the PDS (p = .010) and dating experience (p = .007) variables at Time 2, suggesting that the data were not missing completely at random. Given the presence of missing data, full information maximum likelihood with robust standard errors was used, which allows for the presence of missing data while taking the clustering of cases into account (Muthén & Muthén, 2017).

Results

Participants

Descriptive statistics for demographics variables, PVs, and OVs, are shown in Tables 2 and 3. At time 1, participants (N = 295) were, on average, 12.74 years old (SD = 0.73), with 42% of the sample being female and 70.8% of the sample being White (see Table 2). The mean age of drinking initiation was 15.97 years old (SD = 2.08), and the mean age of regular drinking was 18.58 years old (SD = 1.87). Significantly fewer females than males were White (p=.001). Females reported significantly greater pubertal development at each time point (all ps<.001). Males and females also significantly differed in their report of dating experience at Time 4 (p=.021; Table 3) as females reported more dating behaviors with a steady partner.

Latent Growth Models

Age of Drinking Initiation—A series of five models were fit to the data for age of drinking initiation (see Table 4 for results from all age of drinking initiation models).

Self-reported Pubertal development.—A quadratic model was found to best fit the pubertal development data (Model 1). Model 1 demonstrated sufficient fit ($\chi^2(7, N = 258) = 11.62, p = .11$; CFI = .98, TLI = .96; RMSEA = .05 with 90% CI [.00-.10]). Age of drinking initiation was a significant predictor of the intercept ($\beta = -0.04, p = .017$) and of the linear slope ($\beta = 0.02, p = .015$), indicating that youth who initiated drinking alcohol at a younger age reported a higher level of pubertal development at ages 12-13 and a slower linear rate of change in pubertal development over time. Gender was also a significant predictor of the intercept ($\beta = 0.75, p < .001$) and linear slope ($\beta = -0.14, p < .001$), indicating that females were at a higher level of pubertal development than males at ages 12-13, and that females also experience a slower rate of change in pubertal status over time than males.

Dating Experience.—A linear model was found to best fit the dating experience data. Model 3 ($\chi^2(9, N = 259) = 10.31, p = .33$; CFI = .97, TLI = .95; RMSEA = 0.02 with 90% CI [.00-.07]) indicated that youth who began drinking alcohol at a younger age, reported significantly more dating experience ($\beta = -0.21, p < .001$) at ages 12-13 (Time 1).

Psychological Symptoms.—Like the pubertal development outcome, a quadratic model provided the best fit for externalizing and internalizing symptoms. Model 5 had sufficient model fit ($\chi^2(7, N = 254) = 9.81, p = .20$; CFI = .99, TLI = .97; RMSEA = 0.04 with 90% CI [.00-.09]). The results suggest that youth who initiated drinking alcohol at a younger age have significantly more externalizing symptoms ($\beta = -0.65, p = .02$) at ages 12-13 (Time 1) and a greater rate of linear change in externalizing symptoms ($\beta = -0.38, p = .007$) from ages 12 to 19 (i.e., more externalizing symptomatology over time).

Model 7, examining the relationship between age of drinking initiation and internalizing symptoms, had sufficient fit ($\chi^2(7, N = 254) = 12.26, p = .09$; CFI = .96, TLI = .93; RMSEA = 0.05 with 90% CI [.00-.10]). The results suggest that youth who initiated drinking at a younger age experienced a greater rate of linear change in internalizing symptoms across time ($\beta = -0.47, p = .002$; i.e., more internalizing symptomatology over time).

Average Drinking Days.—A quadratic model provided the best overall model fit for this variable. Model 9 had acceptable model fit ($\chi^2(7, N = 259) = 9.37, p = .23$; CFI = .99, TLI = .99; RMSEA = 0.04 with 90% CI [.00-.09]), however, there were no significant findings.

Age of Onset for Regular Drinking—The same series of five models were fit to the data for age of initiation of regular drinking (see Table 5 for results from all age of onset of regular drinking models).

Self-reported Pubertal development.—Model 2, examining age of onset for regular drinking, demonstrated sufficient fit (χ^2 (7, N = 189) = 13.74, *p* = .06; CFI = .96, TLI = .92; RMSEA = .07 with 90% CI [.00-.12]) The results suggest that youth who began drinking regularly at a younger age had a slower linear rate of change in their pubertal development (β = 0.02, *p* = .02), however, this was result was not significant following the Benjamini-Hochberg adjustment (*p* >.05). Females reported higher pubertal status (β = 0.611, *p* < .001) than males at ages 12-13 (Time 1), but also a slower rate of pubertal development than males over time (β = -0.10, *p* = .012).

Dating Experience.—Model 4 ($\chi^2(9, N = 190) = 7.65, p = .57$; CFI = 1.00, TLI = 1.00; RMSEA = 0.00 with 90% CI [.00-.07]) indicated that youth who began drinking regularly at a younger age also reported greater experience with dating ($\beta = -0.14, p = .03$) at ages 12-13 (Time 1) but this finding was not significant following the Benjamini-Hochberg adjustment (p>.05).

Psychological Symptoms.—Model 6 examined externalizing symptoms in relation to age of onset for regular drinking. This model had acceptable fit ($\chi^2(77, N = 187) = 5.31, p = .62$; CFI = 1.00, TLI = 1.00; RMSEA = 0.00 with 90% CI [.00-.07]). Results suggest that youth who began drinking alcohol regularly at a younger age experience a greater rate of linear change in externalizing symptoms ($\beta = -0.48, p = .04$) from ages 12-19 (i.e., more externalizing symptomatology over time) but this result did not remain significant following the Benjamini-Hochberg adjustment (p > .05).

Model 8, examining the relationship between age of regular drinking initiation and internalizing symptoms, fell below the required thresholds on all indices of fit, so the predictors of interest were not interpreted.

Average Drinking Days.—Model 10, examining average number of drinking days per year in relation to age of initiation for regular drinking did not meet the required thresholds on all indices of fit and the predictors of interest were not interpreted.

Discussion

This prospective study sought to examine the relationship between early alcohol behaviors and adolescent development among 295 youth from 12 to 19 years old using Latent Growth Modeling. Of interest was whether youth who report initiation of any alcohol use and initiation of regular alcohol use at a younger age experience differences in physiological development, social behaviors, psychological functioning, and substance use behaviors.

In the present study, the initiation of *any* alcohol use was found to be associated with 1) a more advanced stage of pubertal development at ages 12-13; 2) a slower rate of change in pubertal development from ages 12-19; 3) more self-reported dating experience at 12-13; 4) more externalizing symptoms at ages 12-13; 5) a greater rate of increasing externalizing symptoms from ages 12-19; and 6) a greater rate of increasing internalizing symptoms from ages 12-19. Regarding initiation of *regular* alcohol use, there was some evidence that earlier age of regular use onset is associated with 1) a slower rate of change in pubertal status from ages 12-19; 2) more dating experience at ages 12-13; and 3) a greater rate of increasing externalizing symptoms from ages 12-19. Importantly, however, these results did not survive correction for multiple comparisons suggesting there is only modest support for these findings. Consistent with the literature, there was also a significant difference in the onset and rate of pubertal development between males and females; however, none of the findings above were found to be significantly moderated by gender.

Overall, the present findings suggest that early onset alcohol use is associated with advanced pubertal development, more experience with dating, and increased externalizing behaviors at a younger age. The early timing hypothesis is one possible explanation for the relationship demonstrated by these findings (Marklein et al., 2009). This hypothesis posits that females who mature earlier are at greater risk for substance use because they are more likely to socialize with older peers, thereby increasing their exposure to opportunities to use substances (Caspi & Moffitt, 1991). In line with this hypothesis, the present study found early pubertal development and greater experience with dating at a younger age to be associated with an earlier age of onset for alcohol initiation. Importantly, the present results suggest that this relationship is true for male adolescents as well (Costello et al., 2007), as gender was not found to moderate this effect. This is an important finding given that current substance use prevention efforts may target early developing females more so than males (Graber et al., 2004).

Given that research has demonstrated that youth who begin drinking alcohol at a young age are more likely to develop an alcohol use disorder in young adulthood (Hingson et al.,

2006), early identification of young alcohol users is key. Age of onset for regular drinking, defined as drinking one time per week for 6 months or more, was found to be associated with greater dating experience at ages 12-13. This finding also aligns with the early timing hypothesis, by demonstrating that youth who begin dating at a younger age not only drink alcohol earlier, but they also drink with more frequency at a younger age. The knowledge of these relationships may be useful for the development of better evidence-based screening measures and substance use prevention programs by increasing the ability to identify youth at the greatest risk for early substance use. For example, selective interventions that are targeted to individuals identified as high risk for developing substance-related problems may be better positioned to prevent the uptake of alcohol use during adolescence (Teesson et al., 2017). Improving our knowledge of risk factors for early-life alcohol use problems could allow for primary and secondary prevention approaches to more accurately identify youth most at risk and intervene within smaller, targeted groups of students. The use of selective and more tailored approaches could better prevent the development of severe alcohol use disorders and reduce financial costs and allow for more effective treatment of more youth at the greatest risk for health outcomes.

The results of the present study also support an effect of early drinking initiation on developmental trajectories of increasing externalizing and internalizing behaviors throughout adolescence. These findings are consistent with previous research demonstrating high rates of comorbid mental health problems in substance-using adolescents (Fleming et al., 2008). Interestingly, a younger age at the time of *any* alcohol initiation was found to be associated with a slower rate of change in pubertal development from ages 12-19. The slower rate of change may be related to an earlier peak in pubertal development at ages 12-13; however, research has demonstrated that early alcohol consumption may delay pubertal onset (Dees et al., 2017) and thus, may also slow the progression of pubertal development over time. In sum, the current findings suggest that early alcohol consumption may impact ongoing development, which could potentially result in emotional and physical-health related consequences.

A strength of the present study is the examination of alcohol initiation effects on multiple domains of adolescent development including physiological, social, and psychological markers. Research on adolescent alcohol use often focuses on only one domain of development; however, these domains are most likely interdependent and by examining multiple domains a more comprehensive understanding of the complicated relationship between early alcohol use and adolescent development is afforded. This study is further strengthened by its large sample size and longitudinal study design, allowing for the examination of change in developmental effects over time. Additionally, examination of the age at which youth begin to drink *regularly (as compared to first starting drinking),* is a strength of the study despite the more limited findings. Age of regular drinking onset is a less-researched yet potentially clinically important construct (Guttmannova et al., 2011). Overall, the findings provide a better characterization of the age-related effects of these different early-life drinking patterns on physiological and emotional outcomes.

Despite several strengths, this study is not without limitations. First, the participants were predominantly non-users at Time 1 and were not recruited based on any risk factors for

potential substance abuse, resulting in low levels of heavy alcohol use by the end of the study. Although these results may not apply to heavy users or treatment-seekers, they do provide insights into what may be considered typical alcohol use during adolescence. The demographics of the sample may also limit generalizability as all youth were recruited in the San Diego area resulting in a predominantly Non-Hispanic White sample. However, race, ethnicity, and socioeconomic status were examined as potential covariates initially, with no significant effects observed, and were therefore removed from the analyses. Large studies, such as the Adolescent Brain Cognitive Development (ABCD) Study (Jernigan et al., 2018), will allow for the future examination of these variables within a demographically representative sample of the United States to better determine if these relationships differ as a function of race, ethnicity, or socioeconomic status. An additional limitation is greater missing data at Time 4 when youth were 18-19 years old. However, this issue was addressed by using the robust maximum likelihood (MLR) estimator in MPlus. In comparison to the default maximum likelihood estimator, MLR is better suited to handle missing nonnormal data, resulting in fewer Type I errors overall and more accurate standard error estimates (Maydeu-Olivares, 2017). Given that the models met required thresholds for adequate model fit despite missing data, we believe these findings make a meaningful contribution to the literature.

Overall, the results of the present study highlight a relationship between early alcohol use behaviors and psychological and physiological development. Early alcohol initiation appears to be associated with early onset pubertal development and dating behaviors. Over time, early alcohol use behaviors may delay pubertal development while exacerbating psychological risk behaviors. These findings contribute to our understanding of the effects of adolescent alcohol use on important developmental outcomes and could be used to improve existing adolescent substance use prevention efforts.

Acknowledgments

This work was funded by National Institute on Alcohol Abuse and Alcoholism grants R01 AA013419, T32AA013525, and F31AA027169 and National Institute on Drug Abuse grants U01 DA041089 and R21 DA047953, and the California Tobacco-Related Disease Research Grants Program Office of the University of California Grants 580264 and T30IP0962.

References

- Aarons GA, Brown SA, Coe MT, Myers MG, Garland AF, Ezzet-Lofstram R, Hazen AL, & Hough RL (1999). Adolescent alcohol and drug abuse and health. Journal of Adolescent Health. 10.1016/ S1054-139X(99)00006-3
- Achenbach TM, & Rescorla LA (2004). Manual for the ASEBA School-Age Forms & Profiles. Burlington, VT: University of Vermont, Research Center for Children. In University of Vermont, Research Center for Children, Youth, & Families. 10.1002/gps.2459
- Achenbach T, McConaughy S, & Howell C (1987). Child/Adolescent Behavioral and Emotional Problems: Implications of Cross-Informant Correlations for Situational Specificity. In Psychological Bulletin. 10.1037/0033-2909.101.2.213
- Achenbach TM, & Rescorla L. a. (2003). Manual for the ASEBA Adult Forms & Profiles. English.
- Achenbach Thomas M., & Rescorla L (2001). Manual for the ASEBA school-age forms & profiles: Child behavior checklist for ages 6-18, teacher's report form, youth self-report. In An integrated system of multi-informant assessment.

Barrett T, & Brignone E (2017). Furniture for Quantitative Scientists.

- Benjamini Y, & Hochberg Y (1995). Controlling the False Discovery Rate: A Practical and Powerful Approach to Multiple Testing. Journal of the Royal Statistical Society: Series B (Methodological). 10.1111/j.2517-6161.1995.tb02031.x
- Blomeyer D, Friemel CM, Buchmann AF, Banaschewski T, Laucht M, & Schneider M (2013). Impact of Pubertal Stage at First Drink on Adult Drinking Behavior. Alcoholism: Clinical and Experimental Research, 37(10). 10.1111/acer.12154
- Boyd SJ, Sceeles EM, Tapert SF, Brown SA, & Nagel BJ (2018). Reciprocal relations between positive alcohol expectancies and peer use on adolescent drinking: An accelerated autoregressive cross-lagged model using the NCANDA sample. Psychology of Addictive Behaviors. 10.1037/ adb0000371
- Brooks-Gunn J, Warren MP, Rosso J, & Gargiulo J (1987). Validity of self-report measures of girls' pubertal status. Child Development. 10.1111/j.1467-8624.1987.tb01423.x
- Brown SA, Myers MG, Lippke L, Tapert SF, Stewart DG, & Vik PW (1998). Psychometric evaluation of the Customary Drinking and Drug Use Record (CDDR): a measure of adolescent alcohol and drug involvement. Journal of Studies on Alcohol, 59(4), 427–438. 10.15288/jsa.1998.59.427 [PubMed: 9647425]
- Calvert WJ, Bucholz KK, & Steger-May K (2010). Early drinking and its association with adolescents' participation in risky behaviors. Journal of the American Psychiatric Nurses Association. 10.1177/1078390310374356
- Caspi A, & Moffitt TE (1991). Individual Differences Are Accentuated During Periods of Social Change: The Sample Case of Girls at Puberty. Journal of Personality and Social Psychology. 10.1037/0022-3514.61.1.157
- Choudhury S, Blakemore SJ, & Charman T (2006). Social cognitive development during adolescence. Social Cognitive and Affective Neuroscience. 10.1093/scan/nsl024
- Chulani VL, & Gordon LP (2014). Adolescent Growth and Development. In Primary Care Clinics in Office Practice. 10.1016/j.pop.2014.05.002
- Costello EJ, Sung M, Worthman C, & Angold A (2007). Pubertal maturation and the development of alcohol use and abuse. Drug and Alcohol Dependence. 10.1016/j.drugalcdep.2006.12.009
- Courtney KE, Worley M, Castro N, & Tapert SF (2018). The effects of alcohol hangover on future drinking behavior and the development of alcohol problems. Addictive Behaviors. 10.1016/ j.addbeh.2017.11.040
- Danzo S, Connell AM, & Stormshak EA (2017). Associations between alcohol-use and depression symptoms in adolescence: Examining gender differences and pathways over time. Journal of Adolescence. 10.1016/j.adolescence.2017.01.007
- Deas D (2006). Adolescent substance abuse and psychiatric comorbidities. In Journal of Clinical Psychiatry. 10.4088/jcp.0706e02
- Dees WL, Hiney JK, & Srivastava VK (2017). Alcohol and Puberty. In Alcohol research[ISP]: current reviews (Vol. 38, Issue 2, pp. 277–282). National Institute on Alcohol Abuse and Alcoholism.
- Donovan JE, & Molina BSG (2011). Childhood Risk Factors for Early-Onset Drinking*. Journal of Studies on Alcohol and Drugs, 72(5), 741–751. 10.15288/jsad.2011.72.741 [PubMed: 21906502]
- Duncan S, Alpert A, Duncan TE, & Hops H (1997). Adolescent alcohol use development and young adult outcomes. Drug and Alcohol Dependence. 10.1016/S0376-8716(97)00137-3
- Duncan TE, & Duncan SC (2004). An introduction to latent growth curve modeling. Behavior Therapy. 10.1016/S0005-7894(04)80042-X
- Farmer RF, Gau JM, Seeley JR, Kosty DB, Sher KJ, & Lewinsohn PM (2016). Internalizing and externalizing disorders as predictors of alcohol use disorder onset during three developmental periods. Drug and Alcohol Dependence. 10.1016/j.drugalcdep.2016.04.021
- Feldstein Ewing SW, Sakhardande A, & Blakemore SJ (2014). The effect of alcohol consumption on the adolescent brain: A systematic review of MRI and fMRI studies of alcohol-using youth. NeuroImage: Clinical. 10.1016/j.nicl.2014.06.011
- Fleming CB, Mason WA, Mazza JJ, Abbott RD, & Catalano RF (2008). Latent Growth Modeling of the Relationship Between Depressive Symptoms and Substance Use During Adolescence. Psychology of Addictive Behaviors. 10.1037/0893-164X.22.2.186

- Graber JA, Seeley JR, Brooks-Gunn J, & Lewinsohn PM (2004). Is pubertal timing associated with psychopathology in young adulthood? Journal of the American Academy of Child and Adolescent Psychiatry. 10.1097/01.chi.0000120022.14101.11
- Grant JD, Scherrer JF, Lynskey MT, Lyons MJ, Eisen SA, Tsuang MT, True WR, & Bucholz KK (2006). Adolescent alcohol use is a risk factor for adult alcohol and drug dependence: Evidence from a twin design. Psychological Medicine. 10.1017/S0033291705006045
- Guttmannova K, Bailey JA, Hill KG, Lee JO, Hawkins JD, Woods ML, & Catalano RF (2011). Sensitive Periods for Adolescent Alcohol Use Initiation: Predicting the Lifetime Occurrence and Chronicity of Alcohol Problems in Adulthood*. Journal of Studies on Alcohol and Drugs. 10.1177/108482239600800317

Hanson KL, Medina KL, Padula CB, Tapert SF, & Brown SA (2011). Impact of Adolescent Alcohol and Drug Use on Neuropsychological Functioning in Young Adulthood:10-year Outcomes. Journal of Child and Adolescent Substance Abuse. 10.1080/1067828X.2011.555272

Harrison E, Drake T, & Ots R (2020). finalfit: Quickly Create Elegant Regression Results Tables and Plots when Modelling. In R package version 1.0.1. https://cran.r-project.org/package=finalfit

- Hingson RW, Heeren T, & Winter MR (2006). Age at Drinking Onset and Alcohol Dependence. Archives of Pediatrics & Adolescent Medicine, 160(7), 739. 10.1001/archpedi.160.7.739 [PubMed: 16818840]
- Hooper D, Coughlan J, & Mullen MR (2008). Structural equation modelling: Guidelines for determining model fit. Electronic Journal of Business Research Methods. 10.21427/D79B73
- Infante MA, Nguyen-Louie TT, Worley M, Courtney KE, Coronado C, & Jacobus J (2019). Neuropsychological Trajectories Associated with Adolescent Alcohol and Cannabis Use: A Prospective 14-Year Study. Journal of the International Neuropsychological Society. 10.1017/ S1355617719001395
- Jernigan TL, Brown SA, & Dowling GJ (2018). The Adolescent Brain Cognitive Development Study. In Journal of Research on Adolescence. 10.1111/jora.12374
- Johnston LD, Miech RA, O'Malley PM, Bachman JG, Schulenberg JE, & Patrick ME (2019). Monitoring the Future national survey results on drug use 1975-2018: Overview, key findings on adolescent drug use.
- Jun HJ, Sacco P, Bright CL, & Camlin EAS (2015). Relations among internalizing and externalizing symptoms and drinking frequency during adolescence. Substance Use and Misuse. 10.3109/10826084.2015.1058826
- Malmberg M, Kleinjan M, Overbeek G, Vermulst AA, Lammers J, & Engels RCME (2013). Are there reciprocal relationships between substance use risk personality profiles and alcohol or tobacco use in early adolescence? Addictive Behaviors. 10.1016/j.addbeh.2013.08.003
- Marklein E, Negriff S, & Dorn LD (2009). Pubertal timing, friend smoking, and substance use in adolescent girls. Prevention Science. 10.1007/s11121-008-0120-y
- Maydeu-Olivares A (2017). Maximum Likelihood Estimation of Structural Equation Models for Continuous Data: Standard Errors and Goodness of Fit. Structural Equation Modeling. 10.1080/10705511.2016.1269606
- Miech RA, Schulenberg JE, Johnston LD, Bachman JG, O'Malley PM, & Patrick ME (2019). National Adolescent Drug Trends in 2019: Findings Released.
- Morean ME, Kong G, Camenga DR, Cavallo DA, Connell C, & Krishnan-Sarin S (2014). First Drink to First Drunk: Age of Onset and Delay to Intoxication Are Associated with Adolescent Alcohol Use and Binge Drinking. Alcoholism: Clinical and Experimental Research, 38(10), 2615–2621. 10.1111/acer.12526
- Moss HB, Chen CM, & Yi H ye. (2014). Early adolescent patterns of alcohol, cigarettes, and marijuana polysubstance use and young adult substance use outcomes in a nationally representative sample. Drug and Alcohol Dependence. 10.1016/j.drugalcdep.2013.12.011
- Muthén LK, & Muthén BO (2017). Mplus User's Guide. Eighth Edition. Los Angeles, CA: Muthén & Muthén. 10.1111/j.1600-0447.2011.01711.x
- Petersen AC, Crockett L, Richards M, & Boxer A (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence. 10.1007/BF01537962

- Poelen EAP, Scholte RHJ, Willemsen G, Boomsma DI, & Engels RCME (2007). Drinking by parents, siblings, and friends as predictors of regular alcohol use in adolescents and young adults: A longitudinal twin-family study. Alcohol and Alcoholism. 10.1093/alcalc/agm042
- R Core Team. (2014). R Core Team (2014). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL Http://Www.R-Project.org/.
- Sartor CE, Jackson KM, McCutcheon VV, Duncan AE, Grant JD, Werner KB, & Bucholz KK (2016). Progression from First Drink, First Intoxication, and Regular Drinking to Alcohol Use Disorder: A Comparison of African American and European American Youth. Alcoholism: Clinical and Experimental Research. 10.1111/acer.13113
- Spear LP (2000). The adolescent brain and age-related behavioral manifestations. In *Neuroscience and Biobehavioral Reviews* (Vol. 24, Issue 4, pp. 417–463). Pergamon. 10.1016/ S0149-7634(00)00014-2
- Spear LP (2018). Effects of adolescent alcohol consumption on the brain and behaviour. In Nature Reviews Neuroscience. 10.1038/nrn.2018.10
- Squeglia LM, Ball TM, Jacobus J, Brumback T, McKenna BS, Nguyen-Louie TT, Sorg SF, Paulus MP, & Tapert SF (2017). Neural predictors of initiating alcohol use during adolescence. American Journal of Psychiatry. 10.1176/appi.ajp.2016.15121587
- Steinberg L (2005). Cognitive and affective development in adolescence. In Trends in Cognitive Sciences. 10.1016/j.tics.2004.12.005
- Stueve A, & O'Donnell LN (2005). Early alcohol initiation and subsequent sexual and alcohol risk behaviors among urban youths. American Journal of Public Health. 10.2105/AJPH.2003.026567
- Teesson M, Newton NC, Slade T, Carragher N, Barrett EL, Champion KE, Kelly EV, Nair NK, Stapinski LA, & Conrod PJ (2017). Combined universal and selective prevention for adolescent alcohol use: a cluster randomized controlled trial. Psychological Medicine. 10.1017/ S0033291717000198
- U.S. Department of Health and Human Services. (2015). 2015 2020 Dietary Guidelines for Americans. 2015 2020 Dietary Guidelines for Americans (8th Edition).
- Wright SP (1992). Adjusted P-Values for Simultaneous Inference. Biometrics. 10.2307/2532694
- Yurasek AM, Brick L, Nestor B, Hernandez L, Graves H, & Spirito A (2019). The Effects of Parent, Sibling and Peer Substance Use on Adolescent Drinking Behaviors. Journal of Child and Family Studies. 10.1007/s10826-018-1251-9
- Yurgelun-Todd D (2007). Emotional and cognitive changes during adolescence. In Current Opinion in Neurobiology. 10.1016/j.conb.2007.03.009

May et al.



Figure 1.

Correlations between examined variables. Darker colors represent stronger correlations between variables (blue colors = positive correlations, red colors = negative correlations). Note: PDS = pubertal development status; T1 = Time 1; T2 = Time 2; T3 = Time 3; T4 = Time 4.

May et al.



Figure 2.

Visual representation of missing data by participant for all variables.

Table 1.

List of Latent Growth Models

	Independent Variable	Dependent Variable
Model 1	Initiation	Pubertal Development
Model 2	Regular Use	Pubertal Development
Model 3	Initiation	Dating Experience
Model 4	Regular Use	Dating Experience
Model 5	Initiation	Externalizing Symptoms
Model 6	Regular Use	Externalizing Symptoms
Model 7	Initiation	Internalizing Symptoms
Model 8	Regular Use	Internalizing Symptoms
Model 9	Initiation	Days of Alcohol Use/Year
Model 10	Regular Use	Days of Alcohol Use/Year

Table 2.

Sample Demographics

	Entire Sample n=295	Males n = 171	Females n = 124	Significance <i>p</i> -value
Age				
Time 1	12.74 (0.44)	12.78 (0.42)	12.70 (0.46)	0.209
Time 2	14.53 (0.50)	14.49 (0.50)	14.59 (0.50)	0.171
Time 3	16.81 (0.39)	16.78 (0.41)	16.85 (0.36)	0.220
Time 4	18.45 (0.50)	18.47 (0.50)	18.43 (0.50)	0.717
Race				
White	209 (70.85%)	134 (78.36%)	75 (60.48%)	0.001
Non-white	86 (29.15%)	37 (21.64%)	49 (39.52%)	
Missing	0 (0.00%)	0 (0.00%)	0 (0.00%)	
Ethnicity				
Hispanic	52 (17.63%)	26 (15.2%)	26 (20.97%)	0.308
Non-Hispanic	236 (80.00%)	139 (81.29%)	97 (78.23%)	
Missing	7 (2.37%)	6 (3.51%)	1 (0.81%)	

Table 3.

Means and standard deviations or percentages of developmental variables of interest across the time points.

Age of Drinking Mill Drinking Initiation Regular Drinking Pubertal Developme Time 1 Time 2 Time 4 Externalizing Sympte Time 3 Time 4 Time 4 Time 1 Time 2 Time 3 Time 4 Time 3 Time 4 Time 3 Time 4 Time 1 Time 2 Time 3	lestones 15.97 (2.08) 18.58 (1.87) ent Status 2.40 (0.69) 3.05 (0.58) 3.51 (0.40) 3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	15.78 (2.00) 18.64 (1.93) 2.12 (0.54) 2.81 (0.52) 3.35 (0.39) 3.50 (0.41) 42.75 (8.80)	16.24 (2.17) 18.49 (1.78) 2.74 (0.69) 3.46 (0.45) 3.73 (0.32) 3.85 (0.25)	0.080 0.597 <.001 <.001 <.001
Drinking Initiation Regular Drinking Pubertal Developme Time 1 Time 2 Time 3 Time 4 Externalizing Sympt Time 1 Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 1 Time 2 Time 2	15.97 (2.08) 18.58 (1.87) ent Status 2.40 (0.69) 3.05 (0.58) 3.51 (0.40) 3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	15.78 (2.00) 18.64 (1.93) 2.12 (0.54) 2.81 (0.52) 3.35 (0.39) 3.50 (0.41) 42.75 (8.80)	16.24 (2.17) 18.49 (1.78) 2.74 (0.69) 3.46 (0.45) 3.73 (0.32) 3.85 (0.25)	0.080 0.597 <.001 <.001 <.001
Regular Drinking Pubertal Developme Time 1 Time 2 Time 3 Time 4 Externalizing Sympt Time 1 Time 3 Time 4 Internalizing Sympt Time 1 Time 1 Time 1 Time 2 Time 2 Time 3 Tim	18.58 (1.87) ent Status 2.40 (0.69) 3.05 (0.58) 3.51 (0.40) 3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	18.64 (1.93) 2.12 (0.54) 2.81 (0.52) 3.35 (0.39) 3.50 (0.41) 42.75 (8.80)	18.49 (1.78) 2.74 (0.69) 3.46 (0.45) 3.73 (0.32) 3.85 (0.25)	0.597 <.001 <.001 <.001
Pubertal Developme Time 1 Time 2 Time 3 Time 4 Externalizing Sympt Time 1 Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 1 Time 2 Time 1 Time 2 Time 3	ent Status 2.40 (0.69) 3.05 (0.58) 3.51 (0.40) 3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	2.12 (0.54) 2.81 (0.52) 3.35 (0.39) 3.50 (0.41) 42.75 (8.80)	2.74 (0.69) 3.46 (0.45) 3.73 (0.32) 3.85 (0.25)	<.001 <.001 <.001 <.001
Time 1 Time 2 Time 3 Time 4 Externalizing Sympt Time 1 Time 3 Time 4 Internalizing Sympt Time 1 Time 1 Time 2 Time 2 Time 3	2.40 (0.69) 3.05 (0.58) 3.51 (0.40) 3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	2.12 (0.54) 2.81 (0.52) 3.35 (0.39) 3.50 (0.41) 42.75 (8.80)	2.74 (0.69) 3.46 (0.45) 3.73 (0.32) 3.85 (0.25)	<.001 <.001 <.001 <.001 <.001
Time 2 Time 3 Time 4 Externalizing Sympt Time 1 Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 2 Time 2 Time 2 Time 2	3.05 (0.58) 3.51 (0.40) 3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	2.81 (0.52) 3.35 (0.39) 3.50 (0.41) 42.75 (8.80)	3.46 (0.45) 3.73 (0.32) 3.85 (0.25)	<.001 <.001 <.001
Time 3 Time 4 Externalizing Sympt Time 1 Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 2 Time 2 Time 2 Time 2	3.51 (0.40) 3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	3.35 (0.39) 3.50 (0.41) 42.75 (8.80)	3.73 (0.32) 3.85 (0.25)	<.001 <.001
Time 4 Externalizing Sympt Time 1 Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 2 Time 2 Time 2 Time 2	3.65 (0.39) toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	3.50 (0.41) 42.75 (8.80)	3.85 (0.25)	<.001
Externalizing Sympt Time 1 Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 2 Time 2 Time 3	toms 42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	42.75 (8.80)		
Time 1 Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 2 Time 2	42.18 (8.27) 41.64 (8.10) 40.44 (8.06)	42.75 (8.80)		
Time 2 Time 3 Time 4 Internalizing Sympt Time 1 Time 2 Time 2	41.64 (8.10) 40.44 (8.06)		41.48 (7.56)	0.280
Time 3 Time 4 Internalizing Sympt Time 1 Time 2 Time 3	40.44 (8.06)	42.18 (7.95)	40.79 (8.30)	0.217
Time 4 Internalizing Sympt Time 1 Time 2 Time 2		40.89 (8.02)	39.88 (8.12)	0.350
Internalizing Sympt Time 1 Time 2	43.29 (9.80)	42.76 (8.01)	44.07 (12.07)	0.588
Time 1 Time 2	oms			
Time 2	42.86 (8.48)	43.18 (8.97)	42.47 (7.87)	0.554
Time 3	42.55 (8.76)	43.39 (8.63)	41.24 (8.86)	0.076
Time 5	41.39 (8.56)	41.44 (8.43)	41.33 (8.75)	0.926
Time 4	42.02 (9.47)	41.07 (8.94)	43.56 (10.27)	0.304
Alcohol Use (Averag	e Drinking Days/Year)			
Time 1	0.58 (3.93)	0.53 (3.19)	0.64 (4.78)	0.814
Time 2	1.76 (8.10)	1.50 (5.40)	2.12 (10.80)	0.526
Time 3	12.42 (27.26)	12.14 (23.79)	12.81 (31.61)	0.847
Time 4	43.81 (51.33)	42.75 (52.69)	45.26 (49.59)	0.684
Dating				
Time 1				0.215
Never	105 (35.59%)	48 (28.07%)	57 (45.97%)	
Not now	55 (18.64%)	36 (21.05%)	19 (15.32%)	
Group dates only	14 (4.75%)	10 (5.85%)	4 (3.23%)	
Rarely	15 (5.08%)	9 (5.26%)	6 (4.84%)	
Occasionally	9 (3.05%)	6 (3.51%)	3 (2.42%)	
Regularly	2 (0.68%)	1 (0.58%)	1 (0.81%)	
Steady partner	6 (2.03%)	3 (1.75%)	3 (2.42%)	
Missing	89 (30.17%)	58 (33.92%)	31 (25.00%)	
Time 2				0.084
Never		33 (19.3%)	27 (21.77%)	
Not now	60 (20.34%)		· · · · · · /	
Group dates only	60 (20.34%) 59 (20.00%)	36 (21.05%)	23 (18.55%)	

	Entire Sample n=295	Males n = 171	Females n = 124	Significance <i>p</i> -value
Rarely	19 (6.44%)	15 (8.77%)	4 (3.23%)	
Occasionally	16 (5.42%)	15 (8.77%)	1 (0.81%)	
Regularly	6 (2.03%)	4 (2.34%)	2 (1.61%)	
Steady partner	25 (8.47%)	14 (8.19%)	11 (8.87%)	
Missing	97 (32.88%)	45 (26.32%)	52 (41.94%)	
Time 3				0.059
Never	18 (6.10%)	7 (4.09%)	11 (8.87%)	
Not now	78 (26.44%)	46 (26.9%)	32 (25.81%)	
Group dates only	5 (1.69%)	0 (0.00%)	5 (4.03%)	
Rarely	17 (5.76%)	13 (7.6%)	4 (3.23%)	
Occasionally	26 (8.81%)	14 (8.19%)	12 (9.68%)	
Regularly	6 (2.03%)	4 (2.34%)	2 (1.61%)	
Steady partner	53 (17.97%)	31 (18.13%)	22 (17.74%)	
Missing	92 (31.19%)	56 (32.75%)	36 (29.03%)	
Time 4				0.021
Never	8 (2.71%)	2 (1.17%)	6 (4.84%)	
Not now	45 (15.25%)	30 (17.54%)	15 (12.10%)	
Group dates only	2 (0.68%)	0 (0.00%)	2 (1.61%)	
Rarely	9 (3.05%)	5 (2.92%)	4 (3.23%)	
Occasionally	13 (4.41%)	11 (6.43%)	2 (1.61%)	
Regularly	9 (3.05%)	7 (4.09%)	2 (1.61%)	
Steady partner	42 (14.24%)	20 (11.70%)	22 (17.74%)	
Missing	167 (56.61%)	96 (56.14%)	71 (57.26%)	

Author Manuscript

Author Manuscript

Table 4.

Parameter values for Age of Initiation of Alcohol Use Models.

Model	Parameter	Intercept	Slope
1	Pubertal Development	-0.04 **	0.02 **
3	Dating Experience	-0.21 **	.016
5	Externalizing Symptoms	-0.65 **	-0.38 **
7	Internalizing Symptoms	0.21	-0.47 **
9	Days of Alcohol Use/Year	-0.14	-0.31

** significant at alpha level of .05 following Benjamini-Hochberg correction

Table 5.

Parameter values for Age of Onset for Regular Alcohol Use Models.

Model	Parameter	Intercept	Slope
2	Pubertal Development	-0.04	0.02^+
4	Dating Experience	-0.14+	0.03
6	Externalizing Symptoms	-0.59	-0.48^{+}
8	Internalizing Symptoms		
10	Days of Alcohol Use/Year		

 $^{\prime}$ only significant at alpha level of .05 prior to Benjamini-Hochberg correction