Genetic and Environmental Influences on Developmental Trajectories of Adolescent Alcohol Use

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Abstract

Adolescent alcohol use demonstrates distinct developmental trajectories with different times of onset, levels, and rates of growth. Twin research on adolescent alcohol use has shown that genetic influences are consistent with a gradual growth of risks, whereas non-shared environmental influences are more consistent with an accumulation of risks over time. The current study investigated the relative contributions of genetic and environmental influences on shaping different developmental trajectories of alcohol use through adolescence. Self-reported past year alcohol use was collected from 877 Canadian twins (47.1% males) at age 13, 14, 15, and 17-year-old. Growth mixture models were fit to examine different developmental trajectories of alcohol use, and biometric liability threshold models were fit to investigate genetic and environmental influences on the liability of belonging to identified trajectories. Three trajectories were identified: low (15.1%), early onset (8.2%), and normative increasing (76.7%). Memberships in the low and early onset group were under genetic (27.6% and 34.7%), shared (42.4% and 21.5%), and non-shared environment influences (30.0% and 43.8%). Membership in the normative increasing group was under genetic (37.7%) and non-shared environment influences (62.3%). Non-shared environmental influences were significantly larger for the normative increasing trajectory than for the low trajectory. These findings provide a more refined picture of genetic and environmental influences in the development of alcohol use in subgroups of adolescents. Genetic and environmental influences both matter, but to different degrees in different trajectories. Future research should identify specific shared and non-shared environmental experiences that distinguish different trajectories.

Key words: adolescence; alcohol use; developmental trajectory; genetic influences; twin design

Introduction

Adolescent alcohol use remains a major public health problem, and is associated with various short- and long-term adverse consequences [1], such as depression [2], risky sexual behavior [3], antisocial behavior [4], and substance abuse and dependency later in life [5]. At the global level, alcohol use and alcohol-related problems (e.g., driving under the influence, injuries) bring tremendous societal burden and costs [6]. Therefore, it is imperative to better understand the risk and protective factors of adolescent alcohol use, as well as its underlying developmental processes, to achieve better intervention effectiveness.

Despite low prevalence in early adolescence, alcohol use steadily increases throughout adolescence [7–9]. For instance, the prevalence of any past year alcohol use at grade 8, 10, and 12 was 17.6%, 38.3%, and 55.6% respectively in the US [7]. This number was even higher in Québec province of Canada, increasing from 23.4% at grade 7 to 83.1% at grade 11 [8]. The substantial increase of alcohol use prevalence throughout adolescence consequently requires a developmental approach to study its onset and development [10, 11]. Particularly, although most adolescents who use alcohol do not develop problematic use in adulthood, about 15% of adolescents nevertheless meet diagnostic criteria for alcohol abuse by age 18 [12]. Furthermore, the majority of adults with alcohol use disorder usually started use during adolescence or even earlier. Early onset of alcohol use is thus one of the strongest predictors of later alcohol use-related problems and psychiatric disorders. For instance, individuals who used alcohol before age 15 are at an increased risk for substance dependence in adulthood, as well as STD infection, educational underachievement, and criminal conviction [9, 13, 14]. Moreover, a faster increase in adolescent alcohol use predicts more alcohol use disorder, antisocial social behavior, and risky sexual behavior in young adulthood [15]. These findings underscore the importance of examining the onset as well as the growth rates of alcohol use from an early age. Notably, person-centered longitudinal studies have revealed that adolescents follow distinct developmental trajectories of alcohol use characterized by different times of onset, levels, and rates of growth.

Many studies have examined developmental trajectories of adolescent alcohol use, and the number and types of trajectories identified vary over studies. This discrepancy is largely due to differences across studies in the specific measures (e.g., past year use vs. past month use, alcohol use vs. binge drinking) and sample characteristics of each study (e.g., age range, ethnicity composition, gender composition). Despite these differences, most studies nevertheless typically found two to five trajectories [see 16 for a comprehensive review], with three common trajectories consistently identified across studies using different measures and samples. Specifically, most studies have identified a trajectory with low level of alcohol use that remains stable

during adolescence. Another common trajectory typically starts at low level and increases steadily through adolescence, representing a pattern of relatively normative use. A third type of trajectory with a more problematic pattern but lower prevalence is characterized with early onset and quick escalation, and its level remains relatively high during adolescence [16–23]. Expectedly, the trajectory with early onset and high level is usually associated with worse alcohol-related outcomes in adulthood [16–18]. These findings highlight the importance of further identifying risk and protective factors and developmental mechanisms that steer adolescents along different trajectories.

Twin studies have shown that genetic, shared environmental (i.e., environmental experiences that make twins of a pair similar to each other), and non-shared environmental (i.e., environmental experiences that create differences between twins reared together) factors all contribute to the initiation and escalation of alcohol use during adolescence [24, 25], with a sample-size weighted average effect of 37%, 36%, and 27%, respectively [26]. Notably, genetic influences tend to be higher, whereas shared environmental influences tend to be lower, for alcohol frequency/quantity than for alcohol initiation [26, 27]. For instance, Maes et al. [28] found predominant genetic (74%) but non-significant shared environmental influences on current alcohol use among 13- to 16-year-old US twins. In contrast, shared environment predominantly (71%) explained lifetime alcohol use, with non-shared environment explained the remaining variance. Rose et al. [29] also found predominant shared environmental influences (76%) on alcohol use/abstinence at age 14 in Finnish twins. Similar results were also reported in other US [30, 31], Dutch [32, 33], Australian [34], and UK [27] adolescent samples.

Longitudinal twin studies have demonstrated that the magnitude of genetic influences increases, whereas shared environmental influences become less salient through adolescence in US [35-37], Dutch [38], and Finnish [39] samples. They have also investigated the dynamic patterns of genetic and environmental contributions to phenotypic continuity and change, revealing high genetic stability but low non-shared environmental stability [38, 40, 41], although some studies have also demonstrated genetic innovation [42]. Particularly, recent studies examining the rates of growth of adolescent alcohol use found that genetic influences are consistent with a gradual growth of risks, whereas non-shared environmental influences are more consistent with an accumulation of risks over time [43, 44]. Together, these findings suggest that a stable set of genetic factors largely contribute to the stability of alcohol use, with new genetic factors emerging over time, while non-shared environmental factors are largely time-specific and mostly contribute to the change of alcohol use [24].

The aforementioned longitudinal twin studies primarily focused on age-to-age change of genetic and environmental influences and/or genetic and environmental stability. The two studies that have examined the

rates of growth both assumed that adolescents follow similar curves [43, 44]. Given that person-centered longitudinal studies have shown that adolescents follow distinct developmental patterns of alcohol use [16–23], it is unclear to what extent genetic and environmental factors can differentially explain different developmental trajectories. No study, to our knowledge, has examined this question for adolescent alcohol use. However, twin studies on other externalizing phenotypes provided some evidence that genetic and environmental factors could differentially contribute to distinct developmental trajectories of externalizing behaviors. Barnes et al. [45] found 70% genetic influences on delinquents showing early-onset persistent trajectory, as opposed to 35% on delinquents primarily showing antisocial behavior during adolescence. Zheng and Cleveland [46] reported genetic influences (31%) in male delinquents showing chronically moderate level of antisocial behavior, but not among those males showing a decreasing pattern (0%) from adolescence to young adulthood. Lastly, Fontaine et al. [47] showed significant shared environmental influences among girls showing a stable high level (75%) of callous-unemotional traits, but not among those girls showing stable low (8%) or decreasing level (26%) from age 7 to 12. Therefore, it seems reasonable to expect that genetic and environmental influences could also differ across different developmental trajectories of alcohol use during adolescence.

Using prospective longitudinal data from a sample of community-based Canadian adolescent twins followed from age 13 to 17 years, the primary aim of the present study was to elucidate the role of genetic, shared and non-shared environmental factors in explaining different developmental trajectories of alcohol use during adolescence. We expected to find at least the few trajectories commonly identified in previous studies: A trajectory with low use throughout adolescence, a trajectory that starts earlier with higher level and rate of growth, and a third trajectory that starts in adolescence and shows a normative increase. Given that these trajectories differ in their times of onset, levels, and rates of growth, and that genetic influences tend to be higher, whereas shared environmental influences tend to be lower, for alcohol frequency/quantity (i.e., levels) than for alcohol initiation (i.e., onset) [26–29], we expected that genetic and/or environmental influences could differ across different developmental trajectories of alcohol use during adolescence.

Methods

Sample and Procedure

The 421 twin pairs in the study (73 MZ males, 90 MZ females, 61 DZ males, 67 DZ females, 130 DZ opposite-sex) were part of a population-based sample of 662 twin pairs from the greater Montréal area recruited at birth between November 1995 and July 1998 [48]. Genetic marker analysis of eight to 10 highly polymorphous genetic markers was conducted to determine zygosity. If genetic material was insufficient or

unavailable, zygosity was also assessed at 18 months and again at age 9 based on physical resemblance using the Zygosity Questionnaire for Young Twins [49, 50]. Comparison between zygosity based on the similarity of genetic markers and physical resemblance showed a 94% correspondence rate, similar to previous studies [50, 51]. Eighty-four percent of the families were of European descent, 3% of African descent, 2% of Asian descent, and 2% were Native Americans. The remaining families (9%) did not provide ethnicity information. The demographic characteristics of the families were comparable to those of a sample of single births representative of the large urban centers in the Québec province when the children were 5 months old [52]. At the time of their children's birth, 95% of parents lived together; 44% of the twins were firstborn; 66% of mothers and 60% of fathers were between 25 and 34 years old; 17% of mothers and 14% of fathers had not finished high school; 28% of mothers and 27% of fathers held a university degree; 83% of the parents held an employment; 10% of the families received social welfare or unemployment insurance; 30% of the families had an annual income of less than $30,000, 44% had an annual total income between $30,000 and $59,999, and 27% had an annual total income of more than $60,000.

The sample was followed longitudinally in secondary school at grades 7, 8, 9, and 11 when the children were 13, 14, 15, and 17 years old, respectively. Overall average attrition was approximately 2% per year, such that 981 twins participated at least once in grades 7 through 11. To conduct trajectory analysis, only twins with at least two waves of data were included, rendering a total of 877 twins. Compared to those excluded families or lost due to attrition, families retained in the final analysis were more likely to be European descent, intact families (i.e., biological parents living together), and had higher annual total income. Data collection took place via personal interviews in the twins' home. Active written consent from the twins and their parents was obtained. The study and procedure were approved by the Institutional Review Board of the University of Québec in Montréal and the Saint-Justine Hospital Research Center.

Measures

Alcohol use. In grades 7, 8, 9, and 11, adolescent twins self-reported the frequency of their past year alcohol use on a 5-point scale, with potential responses ranging from 0 (I haven't consumed alcohol in the past 12 months), 1 (just once, to try), 2 (less than one time per month), 3 (about once a month), to 4 (one or two times a week or more). The question was "during the past 12 months, how frequently have you consumed/drank alcohol?" The questionnaire defined that one alcoholic drink is 4–5 oz of wine, or 10 oz beer, or 1–1.5 oz liquor, and that 0.5% beer does not count as alcohol.

Analytic Strategy

We first identified alcohol use trajectories using growth mixture modeling (GMM) [53–55] in Mplus 7.2 [56]. The statement CLUSTER = family was used to account for twins of a pair being nested in the same family. A maximum likelihood estimator with robust standard error was used in all analyses to account for non-independency of the data. Full-information maximum likelihood was used to handle missing data [57]. Both linear and quadratic slope were modeled and alcohol use was specified as an ordinal outcome. Starting from a 2-class model and for each subsequent model with one more class, multiple models (500) with randomly generated starting values were run to avoid local optima. A few model fit indices were used for model selection, with smaller values suggesting better fit: Akaike Information Criteria (AIC), Bayesian Information Criterion (BIC), and sample size-adjusted BIC (aBIC) [58]. Substantial interpretation and meaning of trajectories, parsimony, and consistency with previous studies were also considered. The Vuong-Lo-Mendell-Rubin (VLMR-LRT) and Lo-Mendell-Rubin adjusted likelihood ratio test (LMR-LRT) [59] were also used. A significant *p* value suggests that a model with *k* class fits the data better than the model with *k-1* classes. GMM provides the estimated portion of participants following each identified trajectory, as well as trajectory-specific estimated means of alcohol use at each time point, which are used for the interpretation, labeling, and plotting of the trajectories.

Next we examined genetic and environmental influences on membership in identified trajectories. Only MZ and DZ twins with valid data for both members of a pair were included in twin analyses, rendering an analyzed sample of 842 twins (421 pairs). Specifically, GMM produces a model estimated posterior probability that ranges between 0 to 1 for each participant for each trajectory after the best model is selected. The posterior probability for one trajectory represents the probability of belonging to this specific trajectory, or group membership, for each participant. Consistent with previous studies, this posterior probability was used as phenotype in twin analyses and we rescaled each posterior probability into five ordinal values to account for its high skewness (0–4) [46, 47]. Specifically, the posterior probability of zero was assigned an ordinalized value of zero, with the remaining non-zero posterior probabilities ordinalized into four equal groups using its three quartile points (e.g., any non-zero posterior probabilities falling below 25% were assigned a value of one, those between 50 and 75% were assigned a value three).

Separately for each trajectory, a biometric liability threshold model was fit to each ordinalized posterior probability. We allowed the thresholds to be different between girls and boys where necessary. This model assumes an underlying normally-distributed liability that represents each person's probability of belonging to each identified trajectory, and decomposes the variance of the latent liability into additive genetic (A), shared environmental (C), and non-shared environmental (E) component including measurement error [60]. For twins

of the same pair, the correlation between A is set to 1 for MZ twins, because they share all of their genes, and 0.5 for DZ twins, because they share on average half of their segregating genes. The correlation between C is set to 1, and to 0 between E for both MZ and DZ twins. If the MZ correlation is more than double that of DZ twins, non-additive (dominance) genetic influences (D) are indicated, which refer to interactions of alleles at the same locus or on different loci. Whenever dominant genetic influences were suggested by intra-pair correlations, an ADE mode was also fit. In this case, the correlation for D between twins is 1 for MZ twins and 0.25 for DZ twins.

All biometric models were conducted using package OpenMx 2.0 with raw data maximum likelihood estimation [61] in R 3.4.1 [62]. Parameter estimates, 95% confidence intervals, and model fit statistics were provided. Model goodness of fit was assessed with minus twice the log likelihood (-2LL). Difference in -2LL between a full model and a nested submodel (reduced model with fewer parameters) was assessed by χ^2 difference tests, with the degrees of freedom equal to the difference in the number of parameters estimated between the full and the reduced model. A non-significant χ^2 test favors the reduced model as a more parsimonious model. AIC was also computed, with a smaller value suggesting a better fit.

Results

Descriptive Statistics and Correlations

The level of past year alcohol use steadily increased over time from 0.30 at grade 7 to 2.14 at grade 11 (see Table 1). The same pattern of increase was observed for both males (from 0.35 to 2.20) and females (from 0.26 to 2.08). Through grade 7 to 11, females remained constantly at a slightly lower level than males. Alcohol use was moderately correlated over time, with *r*s ranging between 0.20–0.54. Except for alcohol use at grade 7, which was positively skewed as would be expected at this age, all other variables were generally normally distributed.

Developmental Trajectories of Adolescent Alcohol Use

As shown in Table 2, AIC decreased from 2- to 4-class model, although only slightly so from 3- to 4-class model. BIC increased from 2- to 4-class model. Sample size adjusted BIC decreased from 2- to 3-class model, and then increased from 3- to 4-class model. Both VLM-LRT and LMR-LRT *p* values were significant in the 2-class model, suggesting that 2-class model fit the data significantly better than 1-class model. However, neither *p* value was significant in either the 3- or 4-class model. At last, entropy for both 2- and 3-class model was acceptable (> 0.75), but not 4-class model (0.65). Since no fit index favored the 4-class model, which had a prevalence of only 5.4% for the smallest trajectory, models with more classes (e.g., 5-class) were not examined.

As fit indices and *p* values disagreed, we chose the 3-class model as the final model, due to the substantive meanings of the trajectories and their consistency with previous studies, as discussed below.

Figure 1 depicts the model-estimated trajectories of alcohol use throughout adolescence. The respective shapes of these trajectories - particularly their relative times of onset, rates of growth, as well as overall drinking levels – guided the interpretation and labeling of these three trajectories. As shown in Figure 1, the majority of twins (76.7%) showed a *normative increasing* trajectory (dashed line) that had a similar level of alcohol use (approximately 0.31, indicating less than once in the past year) to the overall sample (0.30) at age 13. However, their alcohol use steadily increased over time and reached a level of use around 2.40 at age 17, which is comparable to the overall sample level (2.14), indicating slightly less than once a month in the past year. About 15.1% of twins followed a *low* trajectory (solid line) that had low level of alcohol use (i.e., constantly lower than 0.30, indicating less than once in the past year) throughout adolescence without increase. This low trajectory demonstrated the lowest level of initial alcohol use at age 13, rate of growth, and overall level of use. Lastly, a small minority (8.2%) of twins followed an *early starter* trajectory (dotted line). They started using alcohol relatively early with a level of use around 0.50 at age 13 (less than once in the past year), remained at a higher level than the other two trajectories except at age 14, and rapidly increased to a very high level of use at 4.0 at age 17 (one to two times a week or more, as opposed to 2.14 in the overall sample). Adolescents of this trajectory thus represent the most problematic pattern of use, with the earliest time of onset, as well as the highest overall level of alcohol use and rate of growth among all three identified trajectories.

Genetic and Environmental Influences on Trajectory Membership

As shown in Table 3, for the low and early starter trajectories, the intra-pair correlation in MZ twins (0.70 and 0.56) was higher than that in DZ twins (0.56 and 0.39), suggesting additive genetic as well as shared and non-shared environmental influences for both trajectory memberships. For the normative increasing trajectory, however, the MZ intra-pair correlation (0.42) was more than twice that of DZ twins (0.12), suggesting dominant genetic effects. As shown in Table 4, for membership in the normative increasing trajectory, consistent with the intra-pair correlations, the ADE model provided a better fit (AIC = 995.49) than the ACE model (AIC = 997.03). However, the AE model provided a better fit (AIC = 995.03, $\Delta\chi^2 = 1.54$, *ns*) and similar broad heritability estimate, thus was chosen as the final model. It estimated substantial additive genetic (37.7%) and non-shared environmental influences (62.3%). For membership in the *low* trajectory, additive genetic, shared, and non-shared environmental influences were 27.6%, 42.4%, and 30.0%, respectively. Fixing either additive genetic or shared environmental factor to 0 led to a significantly worse fit ($\Delta\chi^2 = 4.17$ and 12.51, $p = .04$ and .00,

respectively). For membership in the early starter trajectory, additive genetic, shared, and non-shared environmental influences were 34.7%, 21.5%, and 43.8%, respectively. Fixing additive genetic or shared environmental factor to 0 did not significantly deteriorate model fit ($\Delta\chi^2$ = 2.38 and 1.42, *ns*, respectively). However, AIC actually increased when fixing additive genetic factor, and only decreased slightly (ΔAIC = 0.58) when fixing shared environmental factor. Considering the non-negligible estimates for each component in the full model, the ACE model was chosen as the final model. Notably, judging by their non-overlapping 95% confidence intervals, non-shared environmental influences were significantly larger for the normative increasing trajectory (95% CI: 0.495–0.765) than for the low trajectory (95% CI: 0.221–0.407).

Discussion

Adolescent alcohol use – particularly early onset and fast rates of growth - is associated with a wide variety of adverse consequences including psychiatric problems (e.g., depression, substance use disorder) [1–5, 12–15]. Therefore, adolescent alcohol use remains a major public health issue, with considerable social burden and costs [6]. A developmental approach to adolescent alcohol use is therefore essential to help understand its onset and escalation, and to provide information about risk and protective factors that could be targeted in interventions to delay onset and mitigate negative outcomes of alcohol use [10, 11]. Twin studies have demonstrated both genetic and environmental influences on the development of adolescent alcohol use [24, 25]. Particularly, recent studies have utilized autoregressive latent growth curve models to investigate genetic and environmental contributions to the rates of growth of adolescent alcohol use [43, 44]. However, previous longitudinal studies have shown that adolescents follow distinct developmental patterns of alcohol use that vary in their times of onset, levels, and rates of growth [16–23]. Therefore, the current study examined genetic and environmental contributions to different developmental trajectories of alcohol use.

Similar to previous studies [16–23], we found substantial inter-individual heterogeneity regarding the developmental trajectories of alcohol use during adolescence. As expected, we identified three trajectories commonly found in previous studies: the largest group with steadily increasing level of alcohol use, labeled normative increasing; a group with low and stable level throughout adolescence, labeled low; and a smallest group that started relatively early, increasing faster and maintained a higher level, labeled early onset.

Consistent with previous studies [26], genetic influences on the three trajectory memberships fell close to the sample-size weighted average effect of 37%. However, different from previous studies on other externalizing behaviors [45–47], genetic influences were largely similar to each other (27.6%–37.7%) across different trajectories, suggesting that genes influence adolescents' development of alcohol use to the same

degree despite their following different developmental patterns. It is important to note that the current findings did not indicate that one predetermined stable set of genes predict adolescents' developmental trajectories of alcohol use over multiple years. New genetic factors emerge over time [38, 40–42]. Instead, genetic (and environmental) influences on trajectory membership indicate that cumulative effects manifest throughout adolescence, which collectively push or drive youths into or away from different trajectories. Therefore, genetic influences on trajectory membership are better regarded as the cumulative magnitude of genetic predisposition to alcohol use expressed through different environmental experiences over development.

Membership in the low trajectory was influenced by both genetic and environmental factors. Shared environment appeared to play a larger role than the other two sources. Also, shared environmental influences appeared to be larger for the low trajectory membership than the other two trajectories. Notably, non-shared environmental influences for the low trajectory membership was significantly lower than that for the normative trajectory membership. Previous twin studies have demonstrated that non-shared environmental factors are largely time-specific and mostly contribute to change of alcohol use, whereas shared environmental influences, when present, typically contribute to stability [38, 40, 41]. The low trajectory was characterized by a stable level of alcohol use, especially compared to the other two trajectories, which showed substantial intra-individual mean changes across adolescence. The current findings are thus congruent with previous studies in suggesting that, for adolescents following developmental trajectories with little or no intra-individual changes (hence high stability), cumulative shared environmental influences would play a larger role. In contrast, non-shared environmental influences, which contribute primarily to change, could cumulatively lead adolescents to follow developmental trajectories divergent from the group average, demonstrating substantial intra-individual changes.

It is possible that some adolescents following the low trajectory may have never used alcohol, at least not until the last time they were followed (i.e., grade 11). Therefore, some adolescents of the low trajectory would be abstainers if they never used even after grade 11 or late-onset users if they started using after grade 11, the latter being more in accordance with the legal drinking age in Québec province (18 years old). The pattern of results for this low trajectory, relative to the other two trajectories where adolescents apparently had already initiated alcohol use but differed in the progression of their use frequency, is consistent with previous findings regarding alcohol initiation vs. frequency. Specifically, studies have shown that shared environmental influences tend to be lower for alcohol frequency/quantity than for alcohol initiation [26–29]. This finding highlights the importance of shared environmental factors in affecting the initiation of alcohol use throughout adolescence. Parental attitudes toward their children's alcohol use, parental supervision, family religion [11, 29, 63], and even

influences from broader contexts such as neighborhood and community norms on alcohol use and regulations [39, 64], possibly play a major role in delaying the initiation of alcohol use.

As the largest group in the sample, adolescents following the normative increasing trajectory had initiated their alcohol use during adolescence. This finding is concordant with studies showing that most adolescents will use alcohol at some point during middle to high school years [7–9]. However, adolescents following the normative increasing trajectory differed from adolescents following the early onset trajectory in that the latter started earlier, generally remained at a higher level of use, and increased at a faster rate than the former. Interestingly, membership in the normative increasing trajectory showed no shared environmental influences. This finding suggests that, besides genetic predisposition, the environmental factors that lead adolescents to follow the normative increasing trajectory are primarily person-specific experiences that distinguish family members from each other. In contrast, the relatively more problematic early onset trajectory was under moderate shared environmental influences. Similar shared environmental factors that influence membership in the low trajectory (e.g., parental attitudes toward alcohol use, family religion, parental alcohol use) [11, 29, 63] possibly also explain why adolescents follow an early onset trajectory.

Significant shared environmental influences were found in regard to the low and early onset trajectories, but not in regard to the normative increasing trajectory. This suggests that shared environmental factors play a major role in affecting the onset of alcohol use, either delaying (as in the low trajectory) or promoting (as in the early onset trajectory) its initiation. Early onset of adolescent alcohol use has been associated with psychiatric problems and other maladaptive outcomes (e.g., depression, antisocial behavior, substance use dependency and disorder, risky sexual behavior) later in development [1–5, 12–15]. Our findings thus highlight the potential benefit of targeting sources of shared environmental influences (e.g., parenting practices, family religion, school behavior norms) in interventions to prevent alcohol use and its associated consequences.

Strengths and Limitations

The current study has a few notable strengths. The longitudinal prospective data from a community-based sample allowed us to investigate genetic and environmental influences on differential developmental trajectories of adolescent alcohol use. Methodologically, we accounted for classification uncertainty through the use of ordinalized posterior probabilities, rather than directly assigning individuals to one trajectory dichotomously based on the largest individual posterior probability. However, a few limitations should be considered while interpreting the results. First, we used a single item to measure adolescents' self-reported past year alcohol use. Although single items have been used in many other studies [65], it would be more informative

to measure multiple aspects of alcohol use (e.g., lifetime use, binge drinking). Second, we did not examine specific measured environmental variables that could predict individuals' probability of following different trajectories. For instance, parental abuse of and addiction to alcohol or other drugs are crucial risk factors of adolescents' alcohol use [67]. Parents addicted to substances may influence their children's alcohol use through genetic pathways by passing on genes that predispose to alcohol use or through environmental pathways by providing a family environment that facilitates adolescents' alcohol use (e.g., by having easily accessible alcohol at home). Relatedly, peer alcohol use has been established as another risk factor for adolescent alcohol use [11, 63]. Moreover, twin research has demonstrated gene-environmental correlation between peer and adolescent alcohol use [35, 66], and that peer alcohol use moderates the expression of genetic predisposition to alcohol use [65]. Thus, it would be important in future studies to incorporate direct measures of parental and peers' substance use to investigate potentially different gene-environment correlation and interaction mechanisms associated with different trajectories of adolescents' alcohol use. Third, caution is warranted when interpreting the current results and generalizing to other populations, including non-twins. This is particularly important when considering the finding that MZ twins showed the highest intra-pair correlation in regard to membership in the low trajectory, which also had the lowest non-shared environmental influences, but significant shared environmental influences. Consequently, the seemingly more protective environment against alcohol use for twins following the low trajectory could possibly be due to specific environments and contexts experienced by the participants of our study. For instance, as previously mentioned, study participants were more likely to live with both biological parents and to have parents with a higher annual total income than families lost to attrition.

Conclusions

To our knowledge, the present study represents the first effort to adopt a person-centered approach within a twin study to examine the development of adolescent alcohol use. The findings further highlight that genetic and environmental factors both matter on the initiation and escalation of alcohol use during adolescence, but to different degrees in distinct developmental trajectories. Future studies should further investigate these nuanced differences in other phenotypes (e.g., tobacco, marijuana). In terms of practical implications, the substantial shared environmental influences on membership in the low and early onset trajectories support the notion that family-focused and community-based intervention against substance use could be particularly effective in delaying the onset of alcohol use.

Ethical standards

This work was approved by the appropriate ethics committee. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. All participants and their parents gave their informed consent prior to their inclusion in the study.

Conflict of Interest

The authors declare that they have no conflict of interest.

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Table 1

Descriptive Statistics (M and SD) and Correlations of Adolescent Alcohol Use over Time by Sex

	Grade 7	Grade 8	Grade 9	Grade 11
Grade 8	.45	-	-	-
Grade 9	.33	.53	-	-
Grade 11	.20	.36	.54	-
All (n = 877)	0.30 (0.65)	0.59 (0.91)	1.16 (1.20)	2.14 (1.34)
Male (n = 413)	0.35 (0.68)	0.62 (0.92)	1.19 (1.23)	2.20 (1.40)
Female (n = 464)	0.26 (0.63)	0.56 (0.90)	1.14 (1.18)	2.08 (1.29)
Skewness	2.42	1.53	0.62	-0.21
Kurtosis	6.16	1.84	-0.73	-0.98

Note. All correlations significant at .001 level.

Table 2

Growth Mixture Model Fit Indices

class	LL	AIC	BIC	aBIC	VLM-LRT *p*	LMR-LRT *p*	entropy
2	-3182.54	6397.08	6473.50	6422.69	0.005	0.006	0.78
3	**-3172.50**	**6384.99**	**6480.52**	**6417.01**	**0.186**	**0.196**	**0.75**
4	-3167.25	6382.50	6497.14	6420.92	0.168	0.176	0.65

Note. LL = log-likelihood. AIC = Akaike Information Criteria. BIC = Bayesian Information Criterion. aBIC = sample size-adjusted BIC. VLM-LRT = the Vuong-Lo-Mendell-Rubin likelihood ratio test. LMR-LRT = the Lo-Mendell-Rubin adjusted likelihood ratio test. Best model bolded.

Table 3

Polychoric Cross-twin Correlations (95% CI) by Zygosity and Sex

	MZM	DZM	MZF	DZF	DOS	MZ	DZ
Normative	0.49 (0.25–0.65)	-0.03 (-0.28–0.23)	0.38 (0.15–0.55)	0.22 (-0.05–0.45)	0.15 (-0.05–0.33)	0.42 (0.27–0.56)	0.12 (-0.02–0.25)
Low	0.69 (0.52–0.80)	0.43 (0.14–0.64)	0.71 (0.56–0.81)	0.62 (0.43–0.76)	0.58 (0.43–0.69)	0.70 (0.59–0.78)	0.56 (0.46–0.65)
Early	0.50 (0.20–0.70)	0.30 (-0.14–0.61)	0.61 (0.39–0.76)	0.62 (0.31–0.80)	0.33 (0.11–0.52)	0.56 (0.39–0.70)	0.39 (0.22–0.53)

Note. MZM = male monozygotic twins. DZM = male dizygotic twins. MZF = female monozygotic twins. DZF = female dizygotic twins. DOS = opposite-sex dizygotic twins. For the normative increasing, low, and early starter trajectory respectively, there was no significant difference in the intra-pair correlations across sex within each zygosity group, χ^2 (3) = 2.39, 1.73, and 3.56, *p*s = 0.50, 0.63, and 0.31, respectively.

Table 4

Genetic and Environmental Influences (95% CI) on Trajectory Membership

	Model	-2LL (df)	AIC	Comparison Model	$\Delta\chi^2$ (Δdf)	*p*	A	C/D	E
Normative									
	ACE	2675.03 (839)	997.03	--	--	--	0.376 (0.122–0.504)	0.000 (0.000–0.173)	0.624 (0.496–0.766)
	ADE	2673.49 (839)	995.49	--	--	--	0.045 (0.000–0.478)	0.380 (0.000–0.555)	0.575 (0.445–1.000)
	AE	2675.03 (840)	995.03	ACE	0 (1)	1	0.377 (0.235–0.505)	--	0.623 (0.495–0.765)
	AE	**2675.03 (840)**	**995.03**	**ADE**	**1.54 (1)**	**.21**	**0.377 (0.235-0.505)**	**--**	**0.623 (0.495–0.765)**
Low									
	ACE	**2497.75 (835)**	**827.75**	**--**	**--**	**--**	**0.276 (0.011–0.534)**	**0.424 (0.201–0.622)**	**0.300 (0.221–0.407)**
	AE	2510.26 (836)	838.26	ACE	12.51 (1)	0	0.737 (0.657–0.801)	--	0.263 (0.199–0.343)
	CE	2501.92 (836)	829.92	ACE	4.17 (1)	.04	--	0.615 (0.537–0.683)	0.385 (0.317–0.463)
Early									
	ACE	**1886.91 (839)**	**208.91**	**--**	**--**	**--**	**0.347 (0.000–0.690)**	**0.215 (0.000–0.530)**	**0.438 (0.304–1.000)**
	AE	1888.33 (840)	208.33	ACE	1.42 (1)	.23	0.594 (0.448–0.711)	--	0.406 (0.289–0.552)
	CE	1889.29 (840)	209.29	ACE	2.38 (1)	.12	--	0.461 (0.338–0.570)	0.539 (0.430–0.662)

Note. The final selected models were bolded. -2LL = minus twice the log-likelihood. AIC = Akaike Information Criteria. A = additive genetic influences. D = dominant genetic influences. C = shared environmental influences. E = non-shared environmental influences.

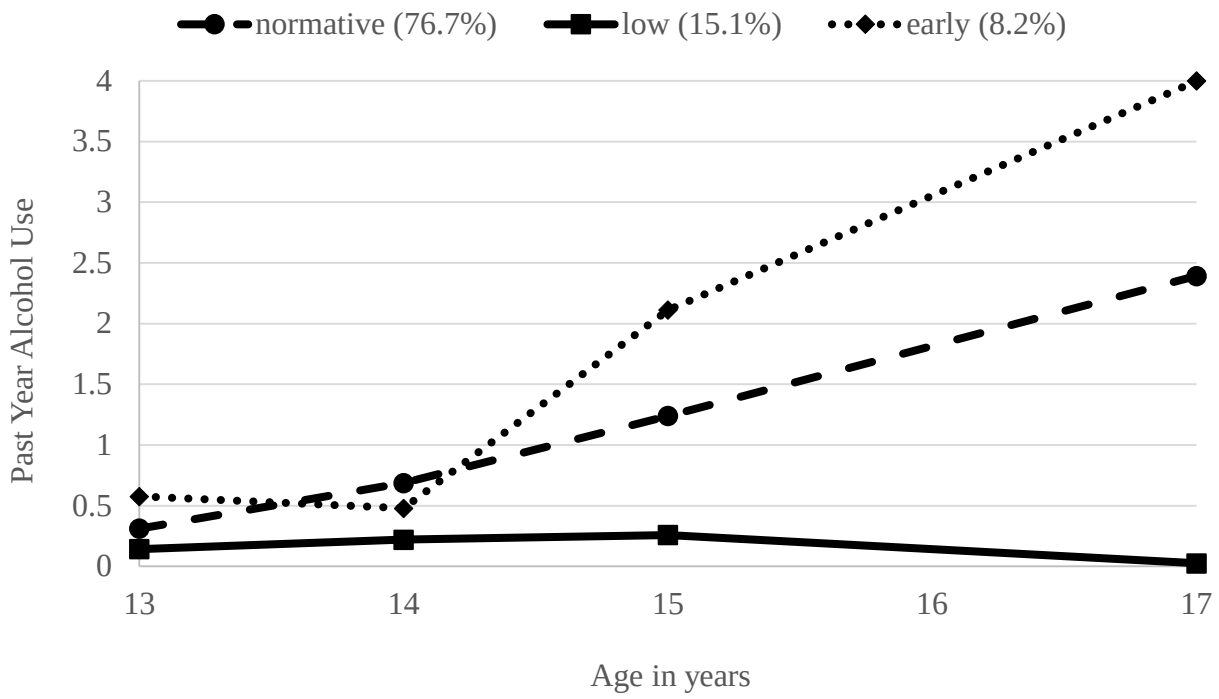

Figure 1. Developmental trajectories of adolescent alcohol use.