Behavior Genetics (2021) 51:528–542
https://doi.org/10.1007/s10519-021-10063-x

ORIGINAL RESEARCH

Maternal Parenting Behaviors Amplify Environmental Influences on Developmental Trajectories of Alcohol Use During Adolescence

Yao Zheng[1] · Mara Brendgen[2,3] · Zachary Meyer[1] · Frank Vitaro[3,4] · Ginette Dionne[3,5] · Michel Boivin[3,5]

Received: 30 January 2021 / Accepted: 15 May 2021 / Published online: 19 May 2021

Abstract

Compared to peer alcohol use, less is known on how parenting practices may modify genetic and environmental contributions to alcohol use longitudinally across adolescence. This study examined whether two maternal parenting behaviors, supervision and harsh parenting, may suppress or amplify genetic and environmental influences on three distinct developmental trajectories of adolescent alcohol use: normative increasing, early-onset, and low trajectories. Participants were drawn from a longitudinal study of a population-based twin sample (N = 842, 84% European descent, 52.7% female). Adolescents self-reported their past year alcohol use at ages 13, 14, 15, and 17 years, and their mothers reported their supervision and harsh parenting when twins were 13, 15, and 17 years old. Maternal supervision amplified non-shared environmental influence on the normative increasing and early-onset trajectories, whereas maternal harsh parenting amplified shared environmental influence on the early-onset trajectory and non-shared environmental influence on the low trajectory, respectively. The findings suggest maternal parenting practices as a potent developmental context that modulates the environmental influences of other proximal processes on adolescent alcohol use, and suggest that family-based parenting-focused intervention could be especially beneficial for adolescents following the early-onset trajectory.

Keywords Adolescent · Alcohol use · Developmental trajectory · Maternal supervision · Harsh parenting · Gene-environment interaction

Introduction

Alcohol use is a global public health concern that brings tremendous cost and burden to society (Rehm et al. 2009). Alcohol use during adolescence—in particular, early onset and higher rates of growth—is linked with a wide variety of short- and long-term consequences (Ellickson et al. 2003; Mason et al. 2010; Odgers et al. 2008). Therefore, a developmental approach is warranted to understanding the onset and development of alcohol use during adolescence, as well as its risk and protective factors (Chassin et al. 2013; Zucker 2006). Adolescents follow distinct developmental trajectories of alcohol use characterized by varying times of onset, levels of use, and rates of growth. Three common trajectories are consistently identified across studies: (1) a normative increasing trajectory that typically represents the majority of the sample and indicates a relatively normative use pattern, where adolescents start at a low level that steadily increases over time; (2) a more problematic use pattern, where adolescents start alcohol use early, escalate quickly over time, and remain at a high level of use; and (3) a low trajectory,

✉ Yao Zheng
yao.zheng@ualberta.ca

✉ Mara Brendgen
Brendgen.Mara@uqam.ca

1 Department of Psychology, University of Alberta, P-217 Biological Sciences Building, Edmonton, AB T6G 2E9, Canada

2 Department of Psychology, Université du Québec à Montréal, C.P. 8888 succursale Centre-ville, Montreal, QC H3C 3P8, Canada

3 Research Unit for Children's Psychosocial Maladjustment, Montreal, QC, Canada

4 School of Psycho-Education, Université de Montréal, Québec City, QC, Canada

5 Department of Psychology, Laval University, Québec City, QC, Canada

where adolescents maintain a low level of use consistently over time (e.g., Flory et al. 2004; Nelson et al. 2015; Zheng et al. 2019a; see Fig. 1). An important next step would be to identify differential genetic and/or environmental aetiologies and developmental mechanisms underlying these distinct developmental trajectories.

Genetic and environmental factors both contribute to the onset and development of alcohol use during adolescence (Dick 2011; Dick et al. 2009). Longitudinal twin studies have found that genetic influence on alcohol use increases, whereas shared environmental influence decreases through adolescence (Kendler et al. 2008; van Beek et al. 2012). There is also evidence of high genetic stability but low non-shared environmental stability over development (Baker et al. 2011; van Beek et al. 2012). Biometric growth curve models also suggest genetic influence on the gradual growth of risk for adolescent alcohol use, but an accumulative pattern of risk over time for non-shared environmental influence (Long et al. 2017). Notably, recent twin studies have provided evidence of differential genetic and environmental contributions to distinct developmental trajectories of externalizing behaviors. Fontaine et al. (2010) revealed substantial shared environmental influence in girls who showed a stable high level of callous-unemotional traits—a strong predictor of adolescent substance use (Anderson et al. 2018) and particularly chronic and severe patterns of antisocial behaviors (Frick et al. 2014). In contrast, shared environmental influence was only modest to negligible in girls who showed a stable low or decreasing level of callous-unemotional traits from ages 7 to 12 years. Zheng and Cleveland (2015) found substantial genetic influence in male delinquents who consistently showed moderate levels of antisocial behaviors (akin to life-course-persistent delinquents), but no genetic influence in male delinquents demonstrating a decreasing pattern (akin to the adolescent-limited delinquents), from adolescence to young adulthood.

As for developmental trajectories of adolescent alcohol use, Zheng et al. (2019a) found that the normative increasing trajectory tended to show larger genetic influence than the low trajectory; moreover, the low and normative increasing trajectories showed the smallest and largest non-shared environmental influence among all trajectories, respectively; both the low and early-onset trajectories showed salient shared environmental influence but not the normative increasing trajectory. Vachon et al. (2017) identified six trajectories of alcohol use from age 14 to 29 years in a large sample of twins, and found that heritability on trajectory memberships monotonically increases for trajectories with more severe alcohol use, and non-shared environmental influence decreases, while shared environmental influence is generally low. Dynamic genetic influences (e.g., genetic innovation and attenuation) differentially triggered or suppressed by environmental experiences in distinct developmental trajectories could partly explain these findings. Collectively, these findings underscore the importance of identifying measured environmental experiences or contexts that could predict different developmental trajectories of externalizing behaviors and examining how these

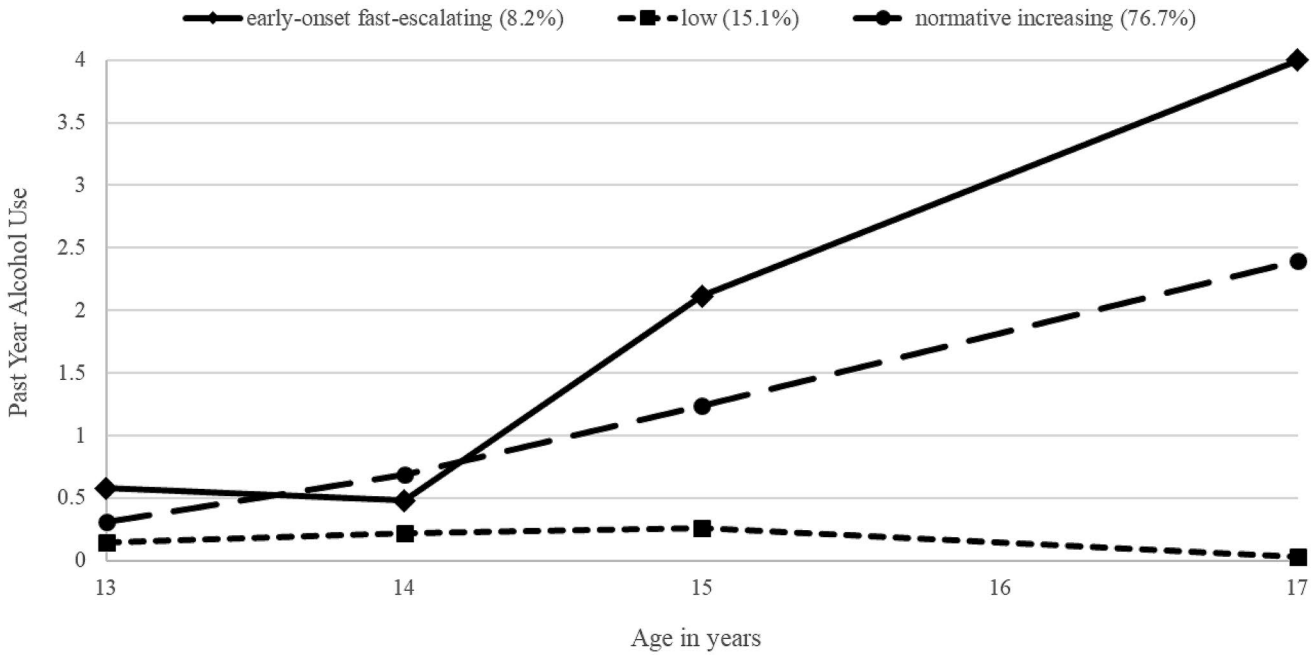

Fig. 1 Model-estimated developmental trajectories (prevalence) of adolescent alcohol use. Adapted from Zheng et al. (2019a) with permission from Springer Nature

environmental experiences may suppress or potentiate genetic liability for different trajectories.

In terms of potential environmental influences, both peer deviance (e.g., peer alcohol use) and parenting practices (e.g., low control and closeness) have been linked with adolescent alcohol use (Chassin and Handley 2006; Hawkins et al. 1992; Ryan et al. 2010). Indeed, even after controlling for common genetic factors, there remains a link between adolescent alcohol use and peer alcohol use (Cruz et al. 2012; Edwards et al. 2015) and parenting practices (Latendresse et al. 2010) through environmental factors. There is also ample evidence in twin research that peer alcohol use can amplify the expression of genetic predisposition for alcohol use in adolescents (Cooke et al. 2015; Dick et al. 2007; Harden et al. 2008; Zheng et al. 2019b).

The literature is less clear, however, on the role of parenting practices in their potential moderation of genetic (G × E) and environmental (E × E) influences on adolescent alcohol use. In a group of 8–17 (*M*age = 14.55) year-old American female twins, Miles et al. (2005) found that genetic and environmental influences on adolescents' lifetime alcohol use without permission from a responsible adult did not differ across families with different levels of strict discipline. However, Hicks et al. (2009) found that mother–child and father–child relationship problems (e.g., conflict and involvement) moderated genetic and environmental influences on externalizing problems (composite scores of antisocial behavior, alcohol, nicotine, and illicit drug abuse/dependence, and teacher ratings of externalizing behavior) in 17-year-old American twins. Specifically, the absolute genetic and non-shared environmental variances were larger in families with more mother–child and father–child relationship problems, whereas the absolute shared environmental variance was slightly larger in families with lower levels of mother–child relationship problems. Dick et al. (2007) found that adolescent-reported parental knowledge (of adolescents' daily plans, interests and activities, whereabouts, and friends' identity) did not moderate genetic or environmental influence on Finnish adolescents' past month alcohol use at age 14 and 17. Using the same data and more advanced modeling techniques, Cooke et al. (2015) nonetheless showed that parental knowledge did moderate both genetic and environmental influences on alcohol use at age 14. Specifically, the absolute genetic, shared and non-shared environmental variances were smaller at high levels of parental knowledge. However, only the moderation of non-shared environmental influence was retained when modeling with the square root or logarithmic transformed data. In the two aforementioned studies that demonstrated evidence of G × E, absolute genetic influences on alcohol use were larger in more problematic environmental contexts (i.e., more parent–child relationship problems and low parental knowledge) characterized by lack of social control or more opportunities to use alcohol. It is important to note, however, that Hicks et al. (2009) found larger *relative* genetic influence at higher levels of parent–child relationship problems (a more adverse environment), whereas Cooke et al. (2015) found larger *relative* genetic influence at *higher* levels of parental knowledge (a more benign environment).

All the aforementioned G × E twin studies involving parenting practices are cross-sectional by design. However, there is evidence that the pattern of G × E in alcohol use (Dick 2011) and conduct problems (Burt 2015) may change over development. Longitudinal twin studies are thus needed to elucidate different G × E and E × E patterns over development, particularly in adolescents who follow distinct developmental trajectories of alcohol use. Along this line, Zheng et al. (2019b) examined whether genetic and environmental influences on different developmental trajectories of adolescent alcohol use varied as a function of friends' alcohol use. The results showed that different G × E and E × E mechanisms were involved in different trajectories. Specifically, whereas both absolute and relative genetic influence was larger for the low and early-onset trajectories at higher levels of friends' alcohol use, absolute and relative non-shared environmental influence was larger for the normative increasing and early-onset trajectories. Given the current limited and inconsistent G × E findings regarding the potential role of parenting practices in adolescent alcohol use, the present study aimed to extend previous research by using the same sample as in Zheng et al. (2019a, b) and incorporating two maternal parenting practices that directly extended from previously examined parenting practices, maternal supervision and harsh parenting. Parental supervision is a combination of parental knowledge and monitoring, whereas harsh parenting indicates the behavioral correlates underlying a negative parent–child relationship. Identifying parenting practices involved in different G × E and E × E mechanisms in distinct trajectories of alcohol use can provide additional malleable environmental targets to offset genetic liability, besides peer alcohol use, to inform tailored interventions (e.g., family-based parenting-focused) for adolescents following different trajectories. Specifically, by directly building on the previously identified three developmental trajectories (low, normative increasing, and early-onset) in a population-based sample of Canadian adolescent twins, this study examined whether maternal supervision and harsh parenting could modify genetic and environmental influences on these trajectories. Given the scarce literature, an exploratory approach was employed. While both parenting practices were expected to potentially modify genetic and/or environmental influences on alcohol use trajectories, moderation pattern may differ across distinct trajectories. Moreover, the moderation patterns may also differ between the two maternal parenting behaviors.

Methods

Participants and procedures

The current sample was drawn from the population-based longitudinal Québec Newborn Twin Study from the greater Montréal area, which includes a sample of 662 monozygotic (MZ) and dizygotic (DZ) twin pairs who were recruited at birth between November 1995 and July 1998 (Boivin et al. 2019). Demographic characteristics of the participating families were comparable to those of a sample of single births representative of the population in the large urban centers of Québec province when the children were 5 months old (Santé Québec et al. 1998). In the total sample, 84% of the families were of European descent, 3% were of African descent, 2% were Native Americans, and 2% were of Asian descent. The remaining families (9%) did not provide ethnicity information. 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.

Zygosity was determined through genetic marker analyses of eight to ten highly polymorphous genetic markers, supplemented by diagnoses based on physical similarity using the Zygosity Questionnaire for Young Twins (Goldsmith 1991) and chorionicity data (Spitz et al. 1996) at 18 months and again at age 9 years. A subsample of same-sex twins with both genetic marker and physical similarity diagnoses showed a 96% concordance rate (Forget-Dubois et al. 2003). Genetic marker diagnoses were used whenever available, while physical similarity diagnoses were used when genetic marker diagnoses were not available. The current study used data assessed at grades 7, 8, 9, and 11 when the twins were 13, 14, 15, and 17 years old, respectively, with an overall average attrition rate of 2% per year. The current sample included twins with at least two waves of data (14.9% with two waves of data, 27.9% with three waves of data, 57.1% with all four waves of data) for both members of a pair, resulting in a total of 421 twin pairs (73 MZ males, 61 DZ males, 90 MZ females, 67 DZ females, and 130 DZ opposite sex). Families included in analyses were more likely to be of European descent, intact families, and had higher annual total income, than excluded families or those lost due to attrition. Data collection took place via personal interviews in the twin's 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

Adolescent alcohol use and developmental trajectories

At the age of 13, 14, 15, and 17 years, adolescents self-reported their past year alcohol use frequency using one item: "During the past 12 months, how frequently have you consumed/drank alcohol?", defining 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. Responses were given on a 5-point scale (0 = "I have not consumed alcohol in the past 12 months", 1 = "just once, to try", 2 = "less than one time per month", 3 = "about once a month", 4 = "one or two times a week or more"). Using growth mixture modeling (GMM), Zheng et al. (2019a) identified three developmental trajectories of adolescents' alcohol use from grade 7 through 11 in this sample: low (15.1%), early-onset (8.2%), and normative increasing (76.7%), as depicted in Fig. 1. The entropy (0.75) for the 3-class model was acceptable, nonetheless suggesting some classification uncertainty, which could partly be due to some participants having only two or three waves of data. In the current study, the posterior probabilities of belonging to each of these identified specific trajectories were used as manifest variables to investigate potential G × E with maternal parenting behaviors.

Maternal supervision

When twins were 13, 15, and 17 years old, mothers of twins reported their supervision for each twin separately using three items, which were selected to reflect the two main aspects of parents' targeted supervision during adolescence (i.e., active parental monitoring and parental knowledge) proposed in Flanagan et al. (2019). Two items ("when the child goes out with friends, do you find out where they are going and what they are planning?", "when the child does an activity outside, do you get to know others involved in the activity?") were rated on a 4-point scale (1 = "never", 2 = "rarely", 3 = "sometimes", 4 = "always"). The third item asked the mother how many of the child's close friends she knew by sight and by their first and last names, with a 5-point response scale (1 = "all", 2 = "Most", 3 = "About half", 4 = "Only a few", 5 = "None". At each grade, each item was first z-standardized and then an average score was calculated across the three items. An average score across the three grades was then created to represent the general level of maternal supervision during adolescence ($rs = 0.37$

and 0.46 at two adjacent time points, $ps < 0.001$). Ordinal Cronbach's α ranged between 0.62 and 0.65 at each wave, and ω ranged between 0.65 and 0.70. Higher scores indicated more maternal supervision.

Maternal harsh parenting

When twins were 13, 15, and 17 years old, mothers of twins reported—separately for each of their twins—the frequency of the following parenting behaviors in the past year based on four items: "firmly grasp or shake your child when he/she was being difficult", "hit your child when he/she being difficult", "raise voice, scold or yell at your child when he/she disobeyed you", "inflict or give corporal punishment on your child when he/she disobeyed you". Responses for the first two items, which were adapted for adolescents from the Parental Cognitions and Conduct Scale (Boivin et al. 2005), were given on a 5-point scale (1 = "never", 2 = "once a month or less", 3 = "once every 2 weeks", 4 = "once a week", 5 = "several times a week") scale. Responses for the last two items, which were based on the Hostile/Ineffective scale used in the National Longitudinal Survey of Children and Youth (Statistics Canada 1995) were also given on a 5-point (1 = "never", 2 = "rarely", 3 = "sometimes", 4 = "often", 5 = "always") scale. Due to the rarity of harsh parenting as well as the unequal anchoring between items, the four items were recoded into three levels, with values of 3 or above all coded as 3. An average score was first created at each grade; then an average score across the three grades was created to represent the general level of maternal harsh parenting during adolescence ($rs = 0.63$ and 0.59 at two adjacent time points, $ps < 0.001$). Ordinal Cronbach's α ranged between 0.82 and 0.87 at each wave, and ω ranged between 0.83 and 0.88. Higher scores indicated more maternal harsh parenting.

Analytic strategy

For each identified trajectory, a univariate biometric liability threshold model (Neale and Maes 2004) was fit to its ordinalized posterior probabilities to examine genetic and environmental influences on the latent liability of belonging to each identified trajectory. Consistent with previous studies (e.g., Fontaine et al. 2010; Zheng and Cleveland 2015; Zheng et al. 2019a, b), a 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). Table 1 shows the descriptive statistics of model estimated posterior probabilities and frequency of ordinalized values. The use of ordinalized posterior probabilities rather than directly assigning adolescents into one most likely trajectory accounts for classification uncertainty and measurement errors. Preliminary analyses suggested different thresholds across sex for the low trajectory only, hence they were allowed to differ between males and females. Conventional univariate biometric models (Neale and Maes 2004) were fit to the two maternal parenting behaviors measures.

Bivariate biometric models (Neale and Maes 2004) were fit to examine genetic and environmental correlations between each of the maternal parenting behaviors and each of the three trajectory memberships, respectively. Preliminary analyses revealed that none of the two maternal parenting behaviors showed any significant phenotypic correlation with any of the three trajectory memberships, except for a modest correlation between maternal supervision and the ordinalized probability of the low trajectory membership (see results section). Hence, a bivariate biometric model with a correlation approach was only fit to maternal supervision and the low trajectory membership to examine the correlations between their respective additive genetic (r_a), shared (r_c) and non-shared (r_e) environmental factors.

Because there were no phenotypic correlations between maternal parenting behaviors and trajectory memberships except for maternal supervision and low trajectory membership, no common genetic or environmental paths were possible between parenting behaviors and trajectory membership. Consequently, there was no potential confounding in the moderation paths of genetic and environmental influences that could be due to the correlation between the moderator and the outcome (van der Sluis et al. 2012). Hence, instead of the bivariate moderation model or extended univariate moderation model (van der Sluis et al. 2012), we directly adopted the univariate moderation model (Purcell 2002) to examine potential G × E involving parenting behaviors. As shown in Fig. 2, paths a,

Table 1 Descriptive statistics of the model-estimated posterior probabilities and frequency of ordinalized values

Trajectory	Posterior probabilities						Ordinalized frequency (%)				
	M	*SD*	Minimum	Maximum	Skewness	Kurtosis	0	1	2	3	4
Low	0.151	0.292	0.000	0.895	1.775	1.399	31.4%	17.7%	16.9%	19.4%	14.7%
Normative increasing	0.767	0.307	0.105	1.000	− 1.061	− 0.432	21.3%	21.0%	24.5%	17.7%	15.5%
Early-onset	0.082	0.177	0.000	0.829	2.479	5.267	64.1%	9.0%	9.1%	8.9%	8.9%

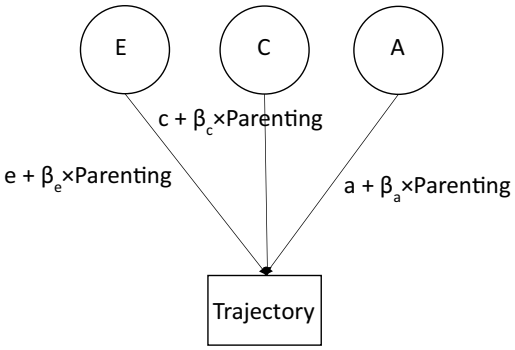

Fig. 2 Univariate biometric moderation of additive genetic (A), shared environmental (C), and non-shared environmental (E) influences on trajectory memberships by parenting practices, shown for one twin

c, and e can vary along the level of parenting behaviors, as indicated by β_a, β_c, and β_e. Because OpenMx (Neale et al. 2016) does not permit missing values on moderators, 12 and 28 families, respectively, were further excluded from the moderation analysis due to missing values on either maternal harsh parenting or supervision. To facilitate interpretation of the moderation results, both parenting behaviors were z-standardized within the sample prior to analyses.

All phenotypic and biometric models were fit using the OpenMx 2.0 package (Neale et al. 2016) in R 3.5.1 (R Development Core Team 2017) with raw data maximum likelihood estimation. OpenMx 2.0 provides parameter estimates, 95% confidence intervals (CIs), as well as several model fit indices. A smaller AIC value indicates a better model fit. Minus twice the log likelihood (-2LL) was used to evaluate the goodness of model fit, and χ^2 difference tests were used to evaluate difference in -2LL between a reduced model and a full model, with the degree of freedom equal to the difference in the number of parameters estimated in the reduced and full model, respectively. A significant χ^2 test suggests that the reduced model fits the data worse and favors the full model.

Results

Univariate biometric model results

As originally reported in Zheng et al. (2019a) and summarized in Table 2, additive genetic influence on the ordinalized probabilities of the low, normative increasing, and early-onset trajectory membership was 27.6%, 37.7%, an 34.7%, respectively. Shared environmental factors only contributed to the low and early-onset trajectory membership (42.4% and 21.5%, respectively). Non-shared environmental contribution to membership in the three trajectories was 30.0%, 62.3%, and 43.8%, respectively. Maternal harsh parenting and supervision demonstrated similar patterns in their genetic and environmental influences. Specifically, both maternal parenting behaviors were primarily explained by shared environmental influence (51.7% and 58.3% for maternal harsh parenting and supervision, respectively), and to a lesser degree by additive genetic influence (32.1% and 26.8%, respectively), with the remaining variance explained by non-shared environmental influence (16.2% and 14.9%, respectively).

Bivariate phenotypic correlations and bivariate biometric model results

There was minimal evidence of phenotypic correlation between both maternal parenting behaviors and the ordinalized probabilities of following any of the trajectories. Specifically, maternal harsh parenting did not show significant links with either trajectory (rs = − 0.07, 0.03, and 0.08 for membership in the normative increasing, low, and early-onset trajectories, respectively). Similarly, maternal supervision was uncorrelated with the normative increasing and early-onset trajectories (rs = 0.03 and 0.07, respectively). However, maternal supervision was negatively correlated with the probability of following the low trajectory (r = − 0.13, 95% CI − 0.22, − 0.04), such that adolescents with a lower level of maternal supervision were more likely to follow the low trajectory of alcohol use. The biometric correlation results from the bivariate model revealed that the

Table 2 Univariate biometric model estimates (95% confidence intervals) for trajectory membership and maternal parenting

	A	C	E
Trajectory membership			
Low	0.276 (0.011, 0.534)	0.424 (0.201, 0.622)	0.300 (0.211, 0.407)
Normative increasing	0.377 (0.235, 0.505)	–	0.623 (0.495, 0.765)
Early-onset	0.347 (0.000, 0.690)	0.215 (0.000, 0.530)	0.438 (0.304, 1.00)
Maternal parenting			
Harsh parenting	0.321 (0.192, 0.463)	0.517 (0.383, 0.629)	0.162 (0.127, 0.209)
Supervision	0.268 (0.151, 0.397)	0.583 (0.462, 0.684)	0.149 (0.116, 0.192)

phenotypic correlation between maternal supervision and the low trajectory of adolescents' alcohol use was due to both correlated additive genetic factors ($r_a = -0.62$, 95% CI −1.00, −0.14) and correlated non-shared environmental factors ($r_e = 0.23$, 95% CI 0.05, 1.00), whereas the correlation between shared environmental factors was not significant ($r_c = -0.05$, 95% CI −0.30, 0.22).

Biometric moderation model results

Results of the biometric moderation models (Table 3) provided some preliminary evidence that maternal harsh parenting moderated non-shared environmental influence on the probability of being in the low trajectory ($\beta_e = 0.081$, SE = 0.048, $p = 0.088$). Dropping the genetic and shared environmental interaction coefficients β_a and β_c together (model 5) did not significantly deteriorate model fit, nested model difference $\chi^2(2) = 1.25$, $p = 0.535$. However, further dropping β_e (model 6) marginally worsened the model fit, $\chi^2(1) = 3.04$, $p = 0.081$. In contrast, maternal harsh parenting tended to moderate shared environmental influence on the probability of being in the early-onset trajectory ($\beta_c = 0.138$, SE = 0.073, $p = 0.059$). Dropping β_a and β_e together (model 5) did not significantly deteriorate model fit, nested model difference $\chi^2(2) = 0.56$, $p = 0.756$. However, further dropping β_c (model 6) marginally worsened the model fit, $\chi^2(1) = 3.63$, $p = 0.057$. Figure 3a and c show the estimated absolute and relative variance components for the liability of following the low (top) and early-onset (bottom) trajectory of adolescent alcohol use, respectively, as a function of maternal harsh parenting. As can be seen, whereas relative non-shared environmental influence explained around 25% of the probability of being in the low trajectory at very low (− 1.5 SD) levels of maternal harsh parenting, it explained about 40% of the variance at very high (+ 1.5 SD) levels of maternal harsh parenting, to the detriment of genetic and shared environmental influences. A similar pattern of an increasing contribution at higher levels of maternal harsh parenting was found for shared environmental influence on the early-onset trajectory.

Maternal supervision significantly moderated the non-shared environmental influence on the probabilities of following the normative increasing and early-onset trajectories ($\beta_e = 0.087$ and 0.138, SEs = 0.037 and 0.042, ps = 0.021 and 0.001, respectively). Dropping β_a for the normative increasing trajectory (model 2), and dropping β_a and β_c together for the early-onset trajectory (model 5), did not significantly deteriorate model fit for the normative increasing trajectory (nested model difference $\chi^2(1) = 0.08$, $p = 0.777$) or the early-onset trajectory (nested model difference $\chi^2(2) = 0.70$, $p = 0.705$). However, further dropping β_e (model 4 for the normative increasing trajectory and model 6 for the early-onset trajectory) led to a significantly worse fit, $\chi^2(1) = 4.59$, $p = 0.032$, and $\chi^2(1) = 8.06$, $p = 0.005$, respectively. Figure 4b and c show the estimated absolute and relative variance components for the liability of following the normative increasing (middle) and early-onset (bottom) trajectory of adolescent alcohol use, respectively, as a function of maternal supervision. As can be seen, whereas genetic and non-shared environmental factors explained roughly equivalent portions of the variance of following the normative increasing alcohol use trajectory at very low (− 1.5 SD) levels of maternal supervision, non-shared environmental influence became relatively more important with increasing levels of maternal supervision. Similarly, as maternal supervision increased, the relative non-shared environmental influence increased while the genetic and shared environmental influences decreased for the probability of following the early-onset trajectory of adolescent alcohol use.

Discussion

The current study aimed to extend a recent study (Zheng et al. 2019b) to investigate whether and how two maternal parenting practices (maternal supervision and harsh parenting) may moderate genetic and environmental influences on three distinct developmental trajectories of adolescent alcohol use. Consistent with previous research (Klahr and Burt 2014), both maternal parenting practices were significantly influenced by genetic factors of the child, although shared environmental influence explained the largest portion of variance. The moderate genetic influence on maternal parenting practices could be explained by evocative gene-environment correlation, where adolescent behaviors elicit specific maternal parenting behaviors (Plomin et al. 1977). Alternatively, as argued by Neiderhiser et al. (2004), passive gene-environment correlation is most likely reflected by large shared environmental influence on parenting in child-based designs, as was also the case in the present study. However, the current findings provide little evidence that maternal supervision or harsh parenting are elicited specifically by adolescents' trajectories of alcohol use. Indeed, only maternal supervision and the low trajectory of alcohol use showed a modest, negative phenotypic correlation. The direction of this correlation is nonetheless consistent with an evocative mechanism, that is, mothers may lower their supervision when their children behave well, such as not engaging in alcohol use.

The general lack of phenotypic correlation between both maternal parenting practices and alcohol use trajectories is surprising, given that research has linked parenting behaviors with adolescent alcohol use (Hawkins et al. 1992; Ryan et al. 2010). The findings also contrast with the significant phenotypic correlation found between friends' alcohol use and these trajectories reported in Zheng et al. (2019b).

Table 3 Univariate biometric moderation model estimates for maternal harsh parenting and supervision

	Model	− 2LnL (df)	AIC	Comparison model	$\Delta\chi^2$ (Δdf)	*p*
Maternal harsh parenting						
Low						
β_a, β_c, & β_e	1	2417.63 (805)	807.63	–	–	–
Drop β_a	2	2418.13 (806)	806.13	1	0.51 (1)	0.48
Drop β_c	3	2417.77 (806)	805.77	1	0.15 (1)	0.70
Drop β_e	4	2419.92 (806)	807.92	1	2.29 (1)	0.13
Drop β_a & β_c	**5**	**2418.88 (807)**	**804.88**	**1**	**1.25 (2)**	**0.54**
Drop all	6	2421.91 (808)	805.91	5	3.04 (1)	0.08
Normative increasing						
β_a & β_e	1	2586.70 (811)	964.70	–	–	–
Drop β_a	2	2586.94 (812)	962.94	1	0.24 (1)	0.63
Drop β_e	3	2589.17 (812)	965.17	1	2.47 (1)	0.12
Drop both	**4**	**2589.20 (813)**	**963.20**	**1**	**2.50 (2)**	**0.29**
Early-onset						
β_a, β_c, & β_e	1	1791.02 (809)	173.02	–	–	–
Drop β_a	2	1791.02 (810)	171.02	1	0.00 (1)	0.98
Drop β_c	3	1792.88 (810)	172.88	1	1.86 (1)	0.17
Drop β_e	4	1791.57 (810)	171.57	1	0.55 (1)	0.46
Drop β_a & β_e	**5**	**1791.57 (811)**	**169.57**	**1**	**0.56 (2)**	**0.76**
Drop all	6	1795.21 (812)	171.21	5	3.63 (1)	0.06
Maternal supervision						
Low						
β_a, β_c, & β_e	1	2318.39 (773)	772.39	–	–	–
Drop β_a	2	2319.44 (774)	771.44	1	1.05 (1)	0.31
Drop β_c	3	2320.54 (774)	772.54	1	2.14 (1)	0.14
Drop β_e	4	2320.33 (774)	772.33	1	1.94 (1)	0.16
Drop all	**5**	**2322.02 (776)**	**770.02**	**1**	**3.62 (3)**	**0.31**
Normative increasing						
β_a & β_e	1	2483.44 (779)	925.44	–	–	–
Drop β_a	**2**	**2483.52 (780)**	**923.52**	**1**	**0.08 (1)**	**0.78**
Drop β_e	3	2486.70 (780)	926.70	1	3.26 (1)	0.07
Drop both	4	2488.11 (781)	926.11	2	4.59 (1)	0.03
Early-onset						
β_a, β_c, & β_e	1	1729.95 (777)	175.95	–	–	–
Drop β_a	2	1730.62 (778)	174.62	1	0.67 (1)	0.41
Drop β_c	3	1730.36 (778)	174.36	1	0.41 (1)	0.52
Drop β_e	4	1736.10 (778)	180.10	1	6.15 (1)	0.01
Drop β_a & β_c	**5**	**1730.65 (779)**	**172.65**	**1**	**0.70 (2)**	**0.71**
Drop all	6	1738.71 (780)	178.71	5	8.06 (1)	0.00

The *p*-values indicate tests in model fit between the full and the reduced models. Final selected models bolded

-2LnL -2 log-likelihood, *df* degrees of freedom, *AIC* Akaike information criterion

Nonetheless, the findings are consistent with the observation that parenting practices may exert a less direct influence on the onset and development of adolescent alcohol use than peer deviance (Duncan et al. 2006; Prins et al. 2020), especially in twin studies (Deutsch et al. 2017; Walden et al. 2004). Instead, the current findings are congruent with the argument that parenting practices act more as distal contexts on adolescent alcohol use (Chassin and Handley 2006), potentially through modifying the influences of other factors (Marceau et al. 2015; Marceau and Jackson 2017). Despite non-significant phenotypic correlation, maternal parenting practices nonetheless can still play an important role as an

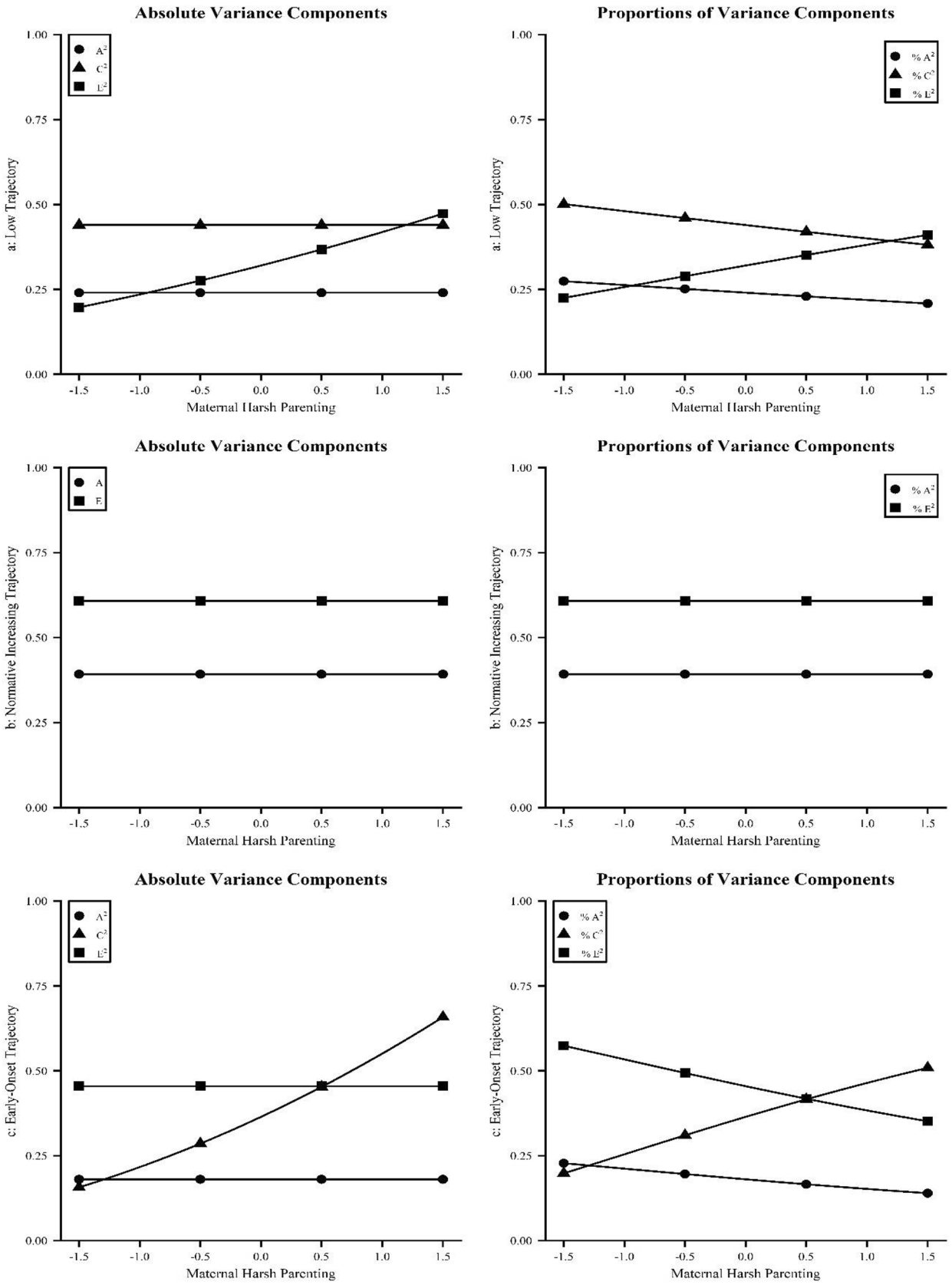

Fig. 3 Estimated absolute variance components (left) and their proportions (right) for the liability of following the **a** low (top), **b** normative increasing (middle), or **c** early-onset (bottom) trajectory of adolescent alcohol use, respectively, as a function of maternal harsh parenting

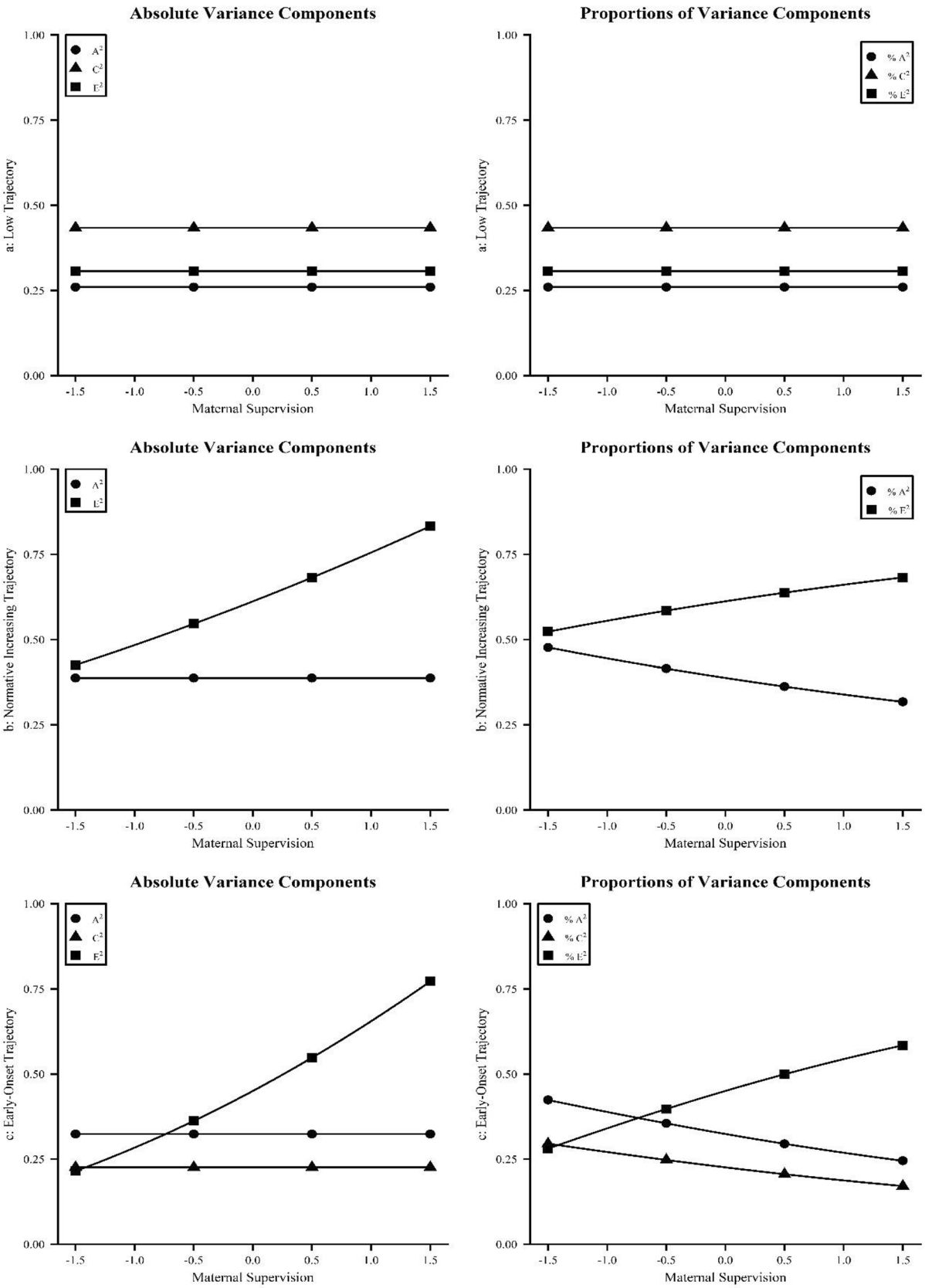

Fig. 4 Estimated absolute variance components (left) and their proportions (right) for the liability of following the **a** low (top), **b** normative increasing (middle), or **c** early-onset (bottom) trajectory of adolescent alcohol use, respectively, as a function of maternal supervision

moderator in shaping the developmental trajectories of alcohol use during adolescence.

Parenting practices as contextual modifiers for adolescent alcohol use development are exemplified in their moderation of environmental influences on trajectory memberships. The contribution of non-shared environmental factors to the normative increasing and early-onset trajectories was diminished—and the relative role of genetic influence was stronger—at lower levels of maternal supervision. In the contexts of low social control, youth likely have more opportunities to access alcohol either during or even prior to adolescence and thus to express their genetic susceptibility to following one of the two trajectories of alcohol use. In contrast, the contribution of shared environmental factors to the early-onset trajectory and that of non-shared environmental factors to the low trajectory were amplified at higher levels of maternal harsh parenting. Accordingly, the relative contribution of genetic influence was diminished at higher levels of maternal harsh parenting. From this perspective, findings pertaining to both parenting behaviors are in line with a genetic suppression process, where certain controlling or demanding environmental conditions 'override' individuals' genetic dispositions for specific behaviors such as alcohol use (Brendgen 2012). Under the conditions of harsh parenting, the degree of exposure to other environmental influences such as parents' or peers' alcohol use as well as societal or religious norms may thus largely determine whether adolescents' follow an early-onset or a consistently low trajectory of alcohol use during adolescence. Previous studies reported similar findings of smaller non-shared environmental influences on child behavioral problems in low SES families (Hendriks et al. 2020) and child conduct problems in families with high parent–child conflict (Burt and Klump 2014).

It is noteworthy that E × E evidence was stronger for maternal supervision than harsh parenting in the current study, the latter at best marginal. This finding resonates with the observation that the relative influence of different parenting practices on child and adolescent problem behaviors might differ (Hoeve et al. 2009; Pinquart 2017) and change over development (e.g., Lansford et al. 2018). Harsh parenting usually decreases substantially from childhood to adolescence (Lansford et al. 2009), whereas parental supervision remains at about the same level, especially in at-risk families (Racz et al. 2019). These developmental differences may—at least in part—explain why parental supervision played a more prominent role than harsh parenting for youth's trajectories of alcohol use during adolescence.

The present findings also revealed that different trajectories involved distinct E × E patterns with maternal parenting practices. Specifically, whereas only the non-shared environmental influence on the low and normative increasing trajectories was moderated by maternal harsh parenting and supervision, respectively, both the shared and non-shared environmental influences on the early-onset trajectory were modulated by maternal parenting. In other words, the two investigated parenting behaviors seemed to be especially important for adolescents' risk of following the early-onset trajectory of alcohol use. Despite being the smallest trajectory (8.2%), the early-onset trajectory represents the most problematic drinking pattern and usually is linked with worse alcohol-related outcomes in adulthood (e.g., Nelson et al. 2015). The current findings thus suggest that focusing on parents' use of supervision—and possibly also harsh punishment—in family-based interventions might eventually deter adolescents from using alcohol already at a young age.

Strengths, limitations, and conclusion

The current study has several notable strengths. First, as opposed to parental knowledge of adolescents' whereabouts and plans assessed in previous studies (Cooke et al. 2015; Dick et al. 2007), which could not differentiate adolescents' active self-disclosure from parents' active seeking of their whereabouts, the current study measured maternal active supervision of adolescents' behaviors. The fact that both maternal parenting practices were reported by mothers whereas adolescents reported about their own drinking behavior helped avoid inflated associations due to shared method variance. This multi-informant assessment approach may also, at least in part, explain the lack of phenotypic correlation between maternal parenting practices and developmental trajectories of alcohol use. Second, different from most previous longitudinal twin studies that examined age-to-age genetic and environmental influences and their stability, the current study adopted a person-centered approach. This made it possible to examine how genetic and environmental factors could differentially contribute to between-person differences in distinct within-person developmental patterns of alcohol use and to reveal nuanced patterns of gene-environment transactions in distinct trajectories. Compared to previous twin studies that examined G × E of parenting in alcohol use, which were all cross-sectional, the developmental approach of the present study is thus more in line with the perspective of changing G × E over development (Burt 2015; Dick 2011).

Nevertheless, the current findings should be considered within the contexts of several limitations. First, adolescent alcohol use was measured with a single item. Although single item measures of alcohol use have been employed in many previous studies (e.g., Cooke et al. 2015; Dick et al. 2007; Latendresse et al. 2010), assessing multiple aspects of alcohol use (e.g., binge drinking) would provide more information regarding adolescent alcohol use. Second, despite being based on very similar items of parental supervision as those used in previous research (Flanagan et al. 2019),

internal consistency of the parental supervision measure was relatively modest in the current study. This did not seem to overly compromise statistical power, however, as moderation effects for parental supervision were nevertheless more pronounced than those found for harsh parenting. Third, close attention should be paid to several concerns regarding GMM in the literature (Sher et al. 2011). Notably, caution should be taken not to apply GMM indiscriminately to all developmental processes and to subsequently consider any identified trajectories as true subgroups. As Vachon et al. (2017) suggest, GMM possibly only extracts categorized subgroups of alcohol use statistically from a true continuous developmental process. These trajectories may only differ in their severity or levels and retain their rank-order over time. In such a case, a latent growth curve approach will likely be a better analytic option. In contrast, when the research questions focus on whether measured environmental experiences (e.g., parenting practices) could differentially moderate genetic and environmental influences on trajectories that vary both in their levels (i.e., severity) and rates of growths (e.g., increasing, stable), GMM may be a more viable option to shed light on these distinct processes and reveal some nuanced patterns. Fourth, future research should examine potential sex differences in the genetic and environmental pathways of alcohol use development.

Finally, it is worth mentioning that the current sample was primarily of European descent (84%). Twin studies on adolescent alcohol use among racial/ethnic minority groups or populations of different cultures and societies are scarce (but see Unger et al. 2011). Different genetic backgrounds and ancestries as well as social-cultural differences between European populations and other racial/ethnic minority groups or non-European societies (e.g., Eastern countries with collectivistic cultures) could lead to different genetic and environmental influences on alcohol use. Different socialization experiences and processes across cultures (e.g., parent–child relationships, peer relationships; Chen and Chen 2010; Chen and French 2008) could also result in different moderating patterns by parenting practices in genetic and environmental influences on alcohol use. Thus, our results are primarily applicable to other European populations with similar demographic compositions and can not be directly generalized to other racial/ethic populations. Notably, recent twin studies have shown that genetic and environmental influences on externalizing (e.g., ADHD, Zheng et al. 2020) and internalizing (e.g., depression and anxiety, Zheng et al. 2016) problems may differ across cultures and social contexts. Given the recent call for more racial/ethnic diverse genetic research on alcohol use (Chartier 2019; Chartier et al. 2017), longitudinal twin studies on alcohol use in ethnically and racially diverse populations are much needed to directly investigate population unique genetic and environmental aetiologies.

Despite its limitations, the current study represents the first effort to elucidate the role of parenting practices in modulating genetic and environmental influences on distinct developmental trajectories of alcohol use during adolescence. The findings further highlight the unique contribution of twin studies, and genetically informed research more broadly, to developmental research on adolescent alcohol use. Different genetic and environmental aetiologies are involved in distinct developmental trajectories characterized with varying times of onset, rates of growth, and levels of use. While parenting practices such as supervision or harsh parenting may not directly influence adolescents' alcohol use, they nonetheless provide a developmental context that further potentiates the environmental influences of other proximal factors. Family-based parenting-focused intervention against substance use could target these amenable parenting practices and tailor different intervention components to adolescents following different trajectories to achieve optimal preventive effectiveness, especially for those following the problematic pattern of early-onset alcohol use.

Author contributions MB, FV, GD, and MB designed the longitudinal study and collected the data. YZ and MB conceptualized the current study. YZ conducted the analyses. YZ and MB drafted the manuscript. All authors provided critical feedback to the manuscript.

Funding This study was funded by the Canadian Institutes of Health Research (MOP 97882, MOP 123342, and MOP199546), the Fonds de Recherche du Québec (Société et Culture; 2014-JU-172894), and the Québec Ministry of Health and Social Services. Mara Brendgen was supported by a Senior Researcher Salary award from the Fonds de Recherche du Québéc sur la Société et la Culture. Michel Boivin was supported by the Canada Research Chair program from the Canadian Institutes of Health Research. The funding sources had no involvement in the study design, the collection, analysis and interpretation of data, the writing of the report, or the decision to submit the article for publication. We thank the twins and their families for participating in this study.

Data availability Available upon request from the corresponding author.

Code availability Available upon request from the corresponding author.

Declarations

Conflict of interest Yao Zheng, Mara Brendgen, Zachary Meyer, Frank Vitaro, Ginette Dionne, MichelBoivin have not any potential conflicts of interest, real or perceived, or financial disclosures to report.

Ethical approval 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. The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Consent to participate Active written consent from the twins and their parents was obtained.

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