Published in final edited form as:

Alcohol Clin Exp Res. 2017 December ; 41(12): 2151–2162. doi:10.1111/acer.13506.

Developmental etiologies of alcohol use and their relations to parent and peer influences over adolescence and young adulthood: A genetically informed approach

Arielle R. Deutsch, PhD¹, Phillip K. Wood, PhD², and Wendy S. Slutske, PhD²

¹Health Outcomes & Population Research, Sanford Research

²Department of Psychological Sciences University of Missouri

Abstract

Background—Distinct changes in alcohol use etiologies occur during adolescence and young adulthood. Additionally, measured environments known to influence alcohol use such as peers and parenting practice can interact or be associated with this genetic influence. However, change in genetic and environmental influence over age, as well as how associations with measured environments change over age, is understudied.

Methods—The National Longitudinal Study of Adolescent Health (Add Health) sibling subsample was used to examine data-driven biometric models of alcohol use over ages 13 to 27. Associations between friends' drinking, parental autonomy granting, and maternal closeness were also examined.

Results—The best fitting model included a five-factor model consisting of early (age 13–20) and overall (age 13–27) additive genetic and unique environmental factors, as well as one overall common environment factor. The overall additive genetic factor and the early unique environment factor explained the preponderance of mean differences in the alcohol use over this portion of the lifespan. The most important factors explaining variance attributed to alcohol use changed over age. Additionally, friend use had the strongest associations with genetic and environmental factors at all ages, while parenting practices had almost no associations at any age.

Conclusion—These results supplement previous studies indicating changes in genetic and environmental influence in alcohol use over adolescence and adulthood. However, prior research suggesting that constraining exogenous predictors of genetic and environmental factors to have effects of the same magnitude across age overlooks the differential role of factors associated with alcohol use during adolescence. Consonant with previous research, friend use appears to have a more pervasive influence on alcohol use than parental influence during this age. Interventions and prevention programs geared toward reducing alcohol use in younger populations may benefit from focus on peer influence.

Alcohol use etiologies dynamically change over adolescence and young adulthood. Alcohol use twin studies indicate that shared/common environment (C) influences are strongest in early adolescence, and then decline in strength until young adulthood (Kendler et al., 2008; Selgem et al., 2016). Unique environmental influences (E) are moderate, but stable, over adolescence and young adulthood. Finally, additive genetic influences (A) are weakest in early adolescence, and steadily increase in strength until young adulthood (Kendler et al., 2008).

Such trends may overlook developmental nuances. Genetic influences that become important in later adolescence and early adulthood may pertain specifically to genes that directly relate to alcohol use (e.g., *ALDH* cluster; Irons et al., 2012), or are more influential for severe alcohol use (e.g., AUD; van Beek et al., 2012). However, there may be earlier genetic influences (e.g., early-mid adolescence) on alcohol use that relate to broader externalizing behavior and on general alcohol consumption (Aliev et al., 2015; Kendler et al., 2011; Meyers, et al., 2014). The measured environmental influences that contribute to shared and unique environmental latent factors may be age-specific as well. For example, measured environments specific to adolescence may include constraints (e.g., barriers to alcohol availability, higher behavioral monitoring) that impede alcohol use. Although behavior genetics research focuses less on specifying measured environmental influence compared to measured genetic influence, identifying age-specific environments may be important for further examination of gene-environment interplay. For example, Kendler et al., (2011) documented that genetic influences for alcohol use are stronger for adolescents living in more permissive environments with easier access to alcohol.

Traditional biometric approaches often fail to represent qualitatively unique genetic and environmental alcohol-use etiologies (i.e., broad-externalizing versus alcohol-specific) as a function of age. Few twin models estimate multiple genetic and/or environment factors (e.g., Adkins et al., 2015; Edwards et al., 2016), or include age-specific etiologies over development. Those that do so present mixed results on age-specific latent etiologies. Edwards and Kendler's (2013) examination of multiple A, C or E factors for alcohol use from adolescence to young adulthood documented both an early adolescence and late adolescence additive genetic factor. Similarly, van Beek (2012) documented that a single all-ages A factor accounted for heritability in alcohol abuse and dependence over adolescence and adulthood, although there were age-specific E factors.

Parent and Peer Influences

Developmental alcohol use models (e.g., deviance proneness, Sher 1991) propose that familial contexts marked by negative parenting styles and positive reinforcement for alcohol use (modeling, availability) are risky environments for early childhood or adolescent drinking (Chartier, Hesselbrock & Hesselbrock, 2010). Parenting practices associated with adolescent alcohol use include lower behavioral control or monitoring and lack of parental closeness (Barnes et al., 2000; Dick et al., 2007; Ennett et al., 2008). Parenting practice – adolescent alcohol use relationships tend to be reciprocal; poor parenting practices influence subsequent alcohol use, and adolescent alcohol use leads to a decline in the quality of parenting practices (e.g., Clark et al., 2008). As adolescents age, parenting influences

diminish in importance. Parenting effects on alcohol use become mediated or moderated by peer influence (Abar and Turrisi, 2008; Nash et al., 2005; Windle 2000), although parental influence on alcohol use does not completely disappear by young adulthood (Wood et al., 2004). Peer influences, typified by selection of and socialization by peers, become more prominent over adolescence and young adulthood (Brechwald & Prinstien, 2011). Alcohol use becomes an important criterion for selecting friends in adolescence; both abstinent and drinking adolescents prefer to select friends who drink (Osgood et al., 2013). In addition to selection influences, adolescents' and young adults' level of alcohol use is maintained/escalated via friend and peer alcohol use norms and friends' own drinking behaviors (Ennett et al., 2008; Bosari et al., 2007).

From a behavioral genetic perspective, parent and peer influences are social environments that contribute to both C and E factors. For example, peer deviance affiliation and parental knowledge relate to both C and E variance explaining alcohol use (Cooke et al., 2015). However, parent and peer influences can relate to genetic influence. For example, both parents and peers moderate heritability of alcohol use (Cooke et al., 2015; Harden et al., 2008; Miles et al., 2005). Previous research has also documented passive (heavier drinking parents have poorer parenting, Latendresse et al., 2008; Van Zundert et al., 2006) and evocative (child's predisposition to alcohol use influences parenting practices, Bountress et al., 2016) gene-environment correlations. Active gene-environment correlations are evident in peer influence, particularly selection. Adolescents are more likely to select peers with similar genotypes (i.e., assortative friendship), and friendship groups tend to be more homophilous for specific gene variants (e.g., Fowler et al., 2011). Children and adolescents genetically predisposed to externalizing behavior are more likely to select alcohol-using peers (Fowler et al., 2007; Harden et al., 2008).

Current Study

This study had two main purposes. First, we wanted to examine a developmental, biometric model of alcohol use across adolescence and young adulthood assessing both early-use trajectories (e.g., broad-externalizing), as well as normative, alcohol specific trajectories of alcohol use. Second, we wanted to examine relationships between underlying additive genetic, unique environment, and common environment etiologies of alcohol use and three commonly related measured environments: peer use, parental autonomy granting, and maternal closeness. We were interested to see if these measured environments would be more influential at specific ages and to specific etiologies.

Methods

Participants

Participants were 2,447 twin and sibling pairs (166 male MZ; 162 female MZ; 130 male DZ; 145 female DZ; 223 opposite-sex DZ; 329 male FS, 356 female FS, 498 opposite sex FS, 116 male HS, 113 female HS, 209 opposite sex HS) from the National Longitudinal Study of Adolescent and Adult Health (Add Health). Ages ranged from 12 – 21 at WI to 17–27 at WIII. Only the first three waves were used, due to lack of assessment of covariates (i.e., autonomy granting and peer influence) at WIV.

Procedure

The study consisted of the first three in-home waves (WI collected 1994–1995; WII collected 1995 – 1996; WIII 2001–2002). Procedures regarding twin and sibling data collection are further discussed in Harris et al., (2006).

Measures

Alcohol use—Alcohol use was a composite four-item measure at all waves. Past-year frequency of typical (any) drinking, binge (five or more drinks) drinking, and drunkenness, were measured by a 7-point likert scale from 0 (*never*) to 7 (*every day or almost every day*). Quantity of drinking on a typical day was initially measured by an open-ended frequency count, and was recoded into a 6-point scale where ‘0’ indicated “no drinks/never drank” and “5” indicated “five or more drinks. Both past-year typical and risky drinking items were included in this measure to capture broad alcohol use; this measure has previously been established as highly reliable across time in genetically informative designs (Agrawal et al., 2011). Abstainers at each wave were coded as 0. These four questions were then summed and standardized across age for all participants. This allowed us to contextualize the mean with respect to an individual’s natural trajectory of alcohol use over age. An over-age approach was used for all standardized variables.

Autonomy Granting—Autonomy granting was measured only at WI and WII. Adolescents were asked whether they were allowed to make seven every-day decisions on their own (e.g., “*Do your parents let you make your own decisions about: the time you must be home on weekend nights; the people you hang around with*”). Items were assessed by a binary yes/no score. These items were averaged and standardized across age⁷⁰. Higher scores indicated greater parental control.

Maternal Closeness—Maternal closeness was measured from WI – WIII. There were differences in measurement between the WI and WII measure and the WIII measure. Adolescents in WI and WII responded to five items regarding maternal relationship. Responses ranged from 1 (not at all) to 5 (very much) for two questions (e.g., “*How close do you feel to your mother?*”) and from 1 (strongly agree) to 5 (strongly disagree) for three statements (e.g. “*Most of the time, your mom is warm and loving towards you*”). At WIII, closeness was measured by three questions. For two questions, responses ranged from 1 (strongly agree) to 5 (strongly disagree) (e.g., “*you enjoy doing things with your mother*”). Response ranged from 1 (extremely close) to 5 (not close at all) for the third question (“*how close do you feel to your mother?*”). Items were recoded such that higher responses reflected higher closeness. Measures were tested for configural invariance by placing all items in a latent factor. The model fit was adequate ($\chi^2(114) = 6,930.949$; CFI = 0.91, RMSEA = 0.06; SRMR = 0.05), indicating good equivalency in the measurement of the construct. All answers at each wave were then averaged together, and standardized across age.

Friends’ drinking—Friends’ drinking was measured at WI-WIII, with the question “*Out of your three best friends, how many drink alcohol at least once a month?*”, and was scored on a scale from 0 (no friends) to 3 (all friends).

Analytic Plans

Age Restructuring—Data was restructured from a wave-based to an age-based format, similar to an accelerated cohort design (e.g., Bell, 1953). Although this approach increased missingness within the variables (e.g., alcohol use at age 13, compared to alcohol use at WI), such an approach was critical for estimating developmental change. As noted by Grimm, Ram and Estabrook (2017, p. 237), we categorized the continuous ages into bins or time-windows to characterize differential effects in the model across age. Age categories were selected to maximize data at each age each age (data) point¹ and secure an adequate amount of data in each age bin. Ages were accordingly binned as 13–14, 15–16, 17–18, 19–20, 21–22, 23–24, and 25–27, for all variables except autonomy granting. Autonomy granting extended from age bins 13–14 to 19–20, as it was only assessed in the first two waves of measurement. WI and WII were only one year apart, therefore some individuals were counted twice in one bin. For these participants, we took the average of their age-based data points.

Biometric Modeling—All biometric models were based on a psychometric, factor modeling approach as outlined in Wang et al., (2015). The psychometric approach allows for fine-grained age-based assessments through estimating dimensionality of the observed data. Psychometric analyses of phenotypic data can be used as a basis for uncovering multiple dimensions of latent A, C and E factors that involve multiple data points (Franic et al., 2013). We first fit psychometric confirmatory factor analysis models based on the observed covariance structures to assess data dimensionality. We then tested a traditional biometric model (one factor per latent construct) to a multidimensional biometric model based on the best-fitting psychometric model. Better fit of the multidimensional model would indicate unique, developmentally sensitive A, C, and E effects on alcohol use, compared to a unidimensional genetic or environmental structure that persists in influence over time.

Figure 1 displays an example biometric model estimation for age bins 13/14 – 17/18. Each twin or sibling had individual A and E latent factors. Additive genetic factors were correlated based on heritability, unique environment factors were orthogonal between twin/siblings, and common environment factors were shared between twins/siblings, to increase parsimony. Item loadings remained freely estimated, factors were identified by constraining variances to one, and item means were set to zero. Item loadings, residual variances, and factor means were all constrained across twins/siblings. Using this process, we calculated model predicted variable means (e.g., mean scores of alcohol use at each age as predicted by the individual factors) and the variances attributed to the A, C, and E factors at each time point (age). Model predicted means for individual age bins within each A, C, and E factor were calculated by multiplying the unstandardized item loading by the unstandardized factor mean. Variance attributed to A, C and E factors at each time point were calculated by dividing the square of the standardized individual factor loading by the sum of all squared factor loadings across A, C and E dimensions. For example, using the factor structure for

¹Binning ages was done after initial attempts to estimate the biometric models discussed in the manuscript using individual ages. These models failed to converge due to sparseness. Syntax and output of these attempts are available by request.

the, best fitting model (see below) the proportion of variance of alcohol use explained by early additive genetic influence at age bin 13–14 can be expressed by Equation 1:

$$\frac{(\text{early A at 13–14})^2}{[(\text{early A at 13–14})^2 + (\text{overall A at 13–14})^2 + (\text{early E at 13–14})^2 + (\text{overall E at 13–14})^2 + (\text{overall C at 13–14})^2 + \text{residual at 13–14}]}$$

All models were estimated using Mplus Version 7.1 (Muthén & Muthén, 1998 – 2012).

Examining Covariate Relations with Biometric Etiologies—We examined correlations between alcohol use and the covariates (friend use, maternal closeness, and autonomy granting) two different ways. First, we correlated the covariates at each age with the latent A, C, and E factors. Although all three covariates could be measured simultaneously, we examined one age bin at a time, as adding more than one age tended to result in model non-convergence. Second, we correlated the covariates with the item loadings themselves (i.e., alcohol use at each age). The first method examined how much parenting practices and friend use related to variance of alcohol use explained by A, C, and E at each age bin. The second method examined covariance contributions to the residual at the individual alcohol use item level at each age bin (e.g., how the covariates may relate to the residual variance at each age; in other words, the alcohol use variance *not* explained by A, C and E). Thus, the second method served as a follow-up examination that probed for unique differential correlation beyond covariate association with the latent variable. Chi-square difference tests comparing the two methods allowed us to examine relative parsimony of the two methods.

Results

Phenotypic Descriptives and Twin Correlations

Table 1 displays the number, percentage, and gender of sibling pairs and gender of sibling pairs. The average age of discordance for full siblings was 2.22 (SD = 1.15), the age range was 0 – 8 years, and 95% of full siblings had a four year or less age difference. The average age of discordance for half siblings was 2.72 (SD = 1.38), the age range was 0 – 7 years, and 88% of half siblings had a four year or less age difference. The mean age for alcohol use onset was 16.74 (SD = 3.10). Figure 2 documents standardized means (standardized across age) for all variables. As evidenced from Figure 2, alcohol use, friends' alcohol use, and autonomy all increased over age at a similar rate over time, while maternal closeness was slightly higher than average for 13 and 14 year olds, and then remained stable from 15 to 27. Table 2 displays the within-pair correlations for alcohol use. Twin/sibling alcohol use scores were similarly correlated for all twin/sibling groups at the earliest age bin (13–14). Correlations between MZ twins were higher compared to all other twin/sibling pair correlations starting at age bin 15–16, indicating genetic influence.

Phenotypic and Biometric Model Fitting

We estimated psychometric models to examine the underlying latent structure of the age-based alcohol use data. Supplemental Table 1 displays model fit indices for all analyses described below. A two-factor age-based model, that included an early factor (ages 13–14 –

19–20) and an overall factor was the best fitting phenotypic model². We then examined a multiple-group gender model to examine if the two-factor model would fit well for both men and women. The multiple group model did not perform significantly worse than the full-sample model (see Supplemental Table 1), demonstrating longitudinal configural invariance (Newsom 2015). Items in the multiple group model were then constrained to be equal between men and women to test for metric invariance. Although the model fit became significantly worse indicating metric non-invariance, model fit indices indicated that including both genders in one model was acceptable.

We then fit a biometric model that included early overall additive genetic (A), common environment (C) and unique environment (E) factors. Table 3 displays model comparisons for a two-factor multidimensional model compared to a) a traditional unidimensional A, C, and E model, and b) a model that dropped A, C, and E early factors individually. The multidimensional two-factor model was a significantly better fit compared to the unidimensional model. Dropping the early C latent factor did not significantly worsen the model, however dropping the early A and E factors made the model fit significantly worse (see Table 3). Therefore, the best-fitting model had five factors: early and overall additive genetic factors (EA and OA), early and overall unique environment factors (EE and OE), and an overall common environment factor (OC). Early factors related to age bins 13–14 to 19–20, and overall factors related to age bins 13–14 to 25–27.

As there was metric non-invariance between genders for the phenotypic models, we wanted to control for potential gender differences. Only the relationships between gender and OA ($\beta = -0.23$, $p < .05$) and OE ($\beta = -0.33$, $p < .05$) were significant, and kept for the final model. This model fit significantly worse (see Table 3), however we felt that it was important to control for potential gender differences, given previous research that indicates genetic sources of variance in men and women do not overlap completely (e.g., Prescott, Aggen & Kendler, 1999).

Final Biometric Model

Table 4 displays the biometric item loadings and means for all early and overall A, C, and E factors, which were used to first estimate model predicted variable means. Figure 3 portrays the model predicted variable means for all factors separately by time. The OA and EE factors had the largest influence on alcohol use mean scores. Model predicted variable means are interpreted by taking into account the unstandardized item loadings and means for the factor. Positive factor means with positive model predicted means indicates a positive relationship between the factor and the mean alcohol use score at a specific age bin. This is the relationship between the OA factor and the mean alcohol use score starting in late

²Initially, a three-factor model that included an overall, early, and late factor (which contained age bins 19–20 to 25–27) was the best fitting model (see supplemental Table 1), however when this was estimated as a biometric model there was no convergence. Additional models were estimated, removing the late factor from the A, C, or E models individually, to try and attain convergence. Only the late additive genetic factor could be added for model convergence. This slightly improved the model fit when comparing this model to the best-fitting five factor model ($\chi^2(402) = 666.19$, CFI = .90, RMSEA = .03, SRMR = .14; $\chi^2(5) = 11.36$, $p > .05$; chi square difference test based on comparison with best fitting model from Table 3). Due to increasing computational demands needed for the covariate models, the late additive genetic factor was not included. This decision was also made due to the fact that an early-factor only model fit significantly better than a late-factor only model (see supplemental Figure 1). Model fit for all tested models is available upon request.

adolescence (e.g., age 19–20 through 25–27). As seen in Figure 3, OA variance is responsible for a majority of the mean differences in alcohol use in late adolescence, distinguishing higher drinkers from other individuals (positive relationship with drinking). In other words, having higher OA factor scores (e.g., specific SNP variants) relates to higher levels of drinking. However in earliest adolescence (age 13–14) the same factor relates to mean differences within a population distinguishing *lower* drinkers from other individuals (a negative relationship with drinking). In other words, having that same OA score in early adolescence related to lower drinking.

The other major contributor to model predicted mean scores was the EE factor. For this factor, a negative mean with negative model predicted means also indicated a positive relationship, but as a protective, rather than a risk effect (i.e., lower drinking). Higher EE factor scores related to mean differences within a population distinguishing lower drinkers from other individuals, as indicated by the factor means (Table 4) and model predicted means (Figure 3).

Figure 4 displays proportions of variance of the alcohol use phenotype explained by all factors by age bin. The standardized scores are presented in Supplemental Table 2. Variance explaining earliest alcohol use was primarily attributed to EE (43% at age bin 13–14). At ages 15–16 to 17–18, however, the EA factor explained the majority of variance attributed to alcohol use (15–16: 32%; 17–18: 40%). In young adulthood, there were three sequential peaks for the overall factors. Alcohol use at ages 19–20 was explained mostly by the OA factor (32%), at ages 21–22 was explained mostly by the OC factor (30%), and at ages 23–24 to 25–27 was explained mostly by the OE factor (23–24: 30%, 25–27: 24%). Additionally, a large portion of variance in age bins 21–22 to 25–27 was not captured by these factors, and related to the error variance (21–22: 45%, 23–24: 41% 25–27: 42% respectively), as seen in Supplemental Table 2.

Covariate Relationships

Table 5 displays all covariate correlations with the ACE factors (factor model) and the individual alcohol use items (item model). Friend use had a stronger unique association with alcohol use compared to parenting practices, for both factor and item associations. Friend use was associated with both environmental and genetic variance, although most consistently and positively associated to OC (e.g., friend use was a positive contributor to the environmental influences explaining alcohol use). Friend use had associations with genetic influences in early and later adolescence, but not early adulthood. At the item level, friend use positively correlated with alcohol use at every age. Therefore, friend use was associated with both the variance attributed to alcohol use by A, C, and E, as well as the residual variance not captured by the biometric factors.

Parenting practices had little association with the factors. The only significant association was a negative association between OE and maternal closeness. Parenting practices had more significant associations with the individual manifest alcohol use items; there were negative associations between maternal closeness and alcohol use at ages 15–16, 17–18 and 25–27, and a positive association between autonomy granting and alcohol use at age 17–18.

Table 6 documents model fit indices comparing all factor models and item models. In every instance, correlating the covariates with the factor had better fit than correlating the covariates with the individual items. This indicated that overall, any influence that friends or parents may have on alcohol use is typically accounted for by the variance explained by the factors.

Discussion

The purpose of this study was to a) investigate a longitudinal, biometric model of alcohol use based on the phenotypic data, and b) examine how such a model related to three predictors of alcohol use (friend use, parental behavior control, and maternal closeness). Results supported previous work reporting age-specific pathways for additive genetic influences over adolescence and young adulthood. Covariates related to etiologies differently over time, and these covariates related to genetic and environmental influences in different ways.

Developmental etiologies of alcohol use

The best-fitting model, using a psychometrically-informed approach (Franic et al., 2013; Wang et al., 2015), had five factors. There were adolescent-only and overall factors for additive genetic and unique environment variance. These results indicate that some genes and environments explain alcohol use over adolescence and adulthood, while others are uniquely important for adolescent alcohol use. Consistent with previous literature (e.g., Kendler et al., 2008; Selgem et al., 2016), variance of alcohol use explained by A, C, and E factors varied across age. However, specific ages in which A, C, or E had the strongest influence differed in the current study compared to previous studies. Previous literature documents that alcohol use is influenced by C in early adolescence, and A effects in late adolescence/young adulthood, while E influences play a consistent but smaller role. The current study indicates that EA influence seems to account for the most variance in early adolescence (ages 15–16 to 17–18), followed quickly by shifting peaks in OA (age 19–20), OC (age 21–22), and OE (23–24 –25–27). Due to these novel results, the current study requires replication. However, results in previous studies may have been due in part to the constraints placed on forcing unique factors of early and later etiological variance to be represented by one factor over time, or by presupposing a temporal factor structure (e.g. Edwards & Kendler, 2013), rather than first estimating the underlying patterns of change over time.

Early adolescent influences: Genes—EA influence was responsible for most variance attributed to adolescent alcohol use, although both EA and OA factors had influence on model means for adolescent alcohol use. This is consistent with previous research documenting that earlier alcohol use in adolescence is driven by genes related to broader externalizing behaviors (e.g., Kendler et al., 2011; Meyers et al., 2014). For example, Aliev et al., 2015 examined potential genes related to both externalizing behaviors and alcohol use separately in adolescent and young adult samples, finding considerable overlap in the genes related to alcohol use and externalizing behaviors. However, in young adulthood, genes related to specific characteristics of alcohol use, such as metabolism (e.g., the *ALDH* cluster) start to play significant roles in alcohol use (e.g, Irons et al., 2012). Therefore, it is

likely that the OA factor as documented in this study relates to genes that have such alcohol influences in adulthood, while the EA influences represent more externalizing influences, potentially explaining previous findings documenting low additive genetic influences in adolescence.

Early adolescent influences: Unique environments—The EE factor also had an important influence on alcohol use. This factor was protective, such that it related to lower alcohol use. Although behavior genetics research has increasingly paid attention to examining nuanced ways in which measured environments may interact with genes in relation to specific phenotypes (e.g., Young-Wolff, Enoch & Prescott, 2011) few studies have examined what influences may be limited to specific developmental periods. The impact of EE was strongest at ages 13–14 for the model predicted means. This was also the time period that EE accounted for the most amount of variance explaining alcohol use. The EE factor modestly related to lower model predicted means of alcohol use for ages 15–16 to ages 19–20, although alcohol use variance explained by EE dropped sharply at this time. This drop in variance can be explained by the substantial increase in the amount of variance explained by EA from ages 13–14 to 15–16, as increases in proportions of variance explained by one factor would require decreases for other factors. Given that specific environments can influence genetic dispositions for alcohol use (e.g., Dick et al., 2001; Rose et al., 2001), these unique environmental factor may set the stage for genetic predispositions relating to alcohol (e.g., deviance-proneness model, Sher 1991; developmental cascade model, Masten & Cicchetti, 2010). This could potentially explain the long-lasting effect of EE on means of alcohol use, despite the reduced amount of variance actually attributed to EE within this developmental period.

Adult-specific factors—When examining the amount of variance accounted for, the five-factor model was able to account for approximately 100% of variance for age bins 13–14 to 18–20. However, for the last three age bins, there was considerable residual variance. Presumably, this would mean that either the residual variance should actually be part of the overall unique environmental factor, or that there should be an additional adult-specific unique environmental factor. The latter has been previously explored by van Beek et al (2012), who documented variability within unique environmental factors over adolescence and adulthood. Indeed, there are adult-specific environments (or environments that are typically adult-specific) such marriage and parenthood, that are related to alcohol consumption (e.g., Dawson et al., 2006; Staff et al., 2014). An additional adult-specific unique environmental factor may better explain variance in alcohol use for young adults. When biometric models with adult-specific factors were examined, only the late genetic factor biometric model was able to converge, indicating that there was too much data missingness to estimate these models. However, given that adding an adult-specific genetic factor improved model fit compared to the final five-factor model (see footnote 2), further explorations of adult-specific etiologies of alcohol consumption are warranted.

How do covariates relate to alcohol use?

Importance of friends—Overall, friends had a more pervasive effect on both genetic and environmental alcohol use factors across developmental stage. The strong relations with

friend use at various age points and various etiologies is consistent with the large body of literature citing friends as a primary risk influence for drinking behavior in adolescence and young adulthood (Brechwald & Prinstein, 2011; Ennett et al., 2008). Friends' drinking was positively related to environmental variance (more so for the common than unique factors), and individual item drinking at each time point. The better model fit indices for factor, rather than item correlations indicated that friend drinking as a measured outcome may contribute to a substantial part of the general variance attributed to either common or unique environment for drinking. The association between genetic variance and friend use is also supported by previous literature documenting gene-environment correlations or interactions (Edwards et al., 2015; Harden et al., 2008). However, these associations stopped after late adolescence. This finding is consistent with research indicating gene-environment correlations for deviant / externalizing friends. If this is the primary genetic influence also responsible for alcohol use in adolescence, there is most likely overlap between genetic variance explaining adolescent drinking behavior and genetic variance explaining selection of drinking friends.

(Un)importance of parents—Parenting practices had little overall influence, consistent with research indicating that although parents are important early on, parental influences on alcohol use start to become more distal as adolescents become more independent (Abar and Turrise, 2008; Windle, 2000). Parenting practices also had no relation with genetic alcohol use variance. Potential nuances of gene-environment correlations or interactions, particularly with weaker influences or time-bound effects which are more finely resolved than those captured by the age bins considered here, may not be captured by the models in this study. The few relations captured are consistent with previous literature, with negative relationships between environmental alcohol use variance and maternal closeness and positive relations between environmental alcohol use variance and autonomy granting (e.g., Ennett et al., 2008). Parenting practices were more strongly associated with the residual than with the genetic and environmental factor variance explaining alcohol use. There may be parenting practices (e.g., alcohol-specific monitoring) more relevant to the common/unique environments explaining alcohol use at different ages. Associations with residual variance may also represent distal, rather than proximal effects; parenting practices tend to become mediated by friend use (Abar and Turrise, 2008; Windle 2000). Therefore, parenting practices may influence the measured environments that make up the latent C and E factors, but not be directly part of them.

Limitations and future directions

The main limitation of this study is the amount of missing data within our sample. We were able to examine a large age range (age 13 to age 27), however the nature of the data (three waves, each wave with an age range of 10 years) forced us to make compromises to our model, particularly with the covariate analyses. Adding covariates for more than one age bin at a time resulted in non-convergence, therefore we were not able to comprehensively examine all covariates simultaneously in one model (which would have allowed for examining if a covariate at a specific age was influential while controlling for all other ages). Additionally, due to the large amounts of missingness, we decided to model men and women simultaneously while controlling for gender, rather than testing for gender-specific model

and accompanying invariance analyses. Original phenotypic models had configural invariance, indicating that the overall factor structures were similar for men and women. However, the model lacked metric invariance, indicating that items (e.g., alcohol scores at each age) related to the factors differently in men and women. Future research should examine how gender may uniquely play a role in the relation between early or overall etiologic factors and covariates, as well as the possibility of differential measurement reliability as a function of sex across ages examined. A model with less missing data would better permit estimation of the models and a more fine-grained resolution of the age-related differences. We were also not able to further explore a late (adulthood-only) factor. Initial phenotypic exploration indicated that such a model may be a better fit compared to the five-factor model. This idea was further supported by the large amount of residual variance that was not captured by the overall A C and E factors in young adulthood. Results may have been different if we included only twins, compared to twins and siblings, as age differences between siblings may have produced uniquely different influences (i.e., older sibling influences) compared to twins (Poelen et al., 2007; Scholte et al., 2008). Additionally, results may have differed if we constrained the sample to individuals who reported initiating at WI (i.e., “pure” non-abstainers). To test this, we attempted to test models in which only those who reported drinking at wave 1 were included in the models (similar to the tests by Edwards and Kendler, 2013, who use a similar approach of allowing for natural age differences in onset behavior), however none of these models were able to be estimated, most likely due to the fact that the substantially reduced N was paired with increased sparseness (and possible lack of coverage at all age bins).

Future research should examine how etiologies may change as a combined function of age and stage of use, as onset of alcohol use is more related to environmental factors, i and progression to heavier use is influenced more by genetics (e.g., Fowler et al., 2007; Pagan et al., 2006). Few studies have attempted to disentangle age and stage, and it may be prudent, particularly given the current results, to examine either stage of use across time or age by stage of use. For example, Poelen et al., (2008) examined different stages in early adolescents exclusively; contrary to other studies, the authors found that there was a substantial genetic contribution to initiation. Future research may benefit from examining adolescent-specific environments relating to drinking behavior. Research indicates that early adolescent alcohol use is a possibly causal risk factor for higher adulthood alcohol (e.g., Deutsch et al., 2013; Deutsch et al., 2016). Extreme risk factors linked to high levels of alcohol use that exclusively occur in childhood and adolescence (e.g., child abuse, Dube et al., 2006; parental divorce, Sartor, Lynskey, Heath, Jacob & True, 2007), may account for environmental variance related to alcohol use.

Conclusions

Twin studies continue to be a powerful way to examine phenotypic infrastructure. They allow us to assess the weight of impact of measured environments, in addition to how phenotypes manifest from an interrelation of genetic and environmental influences. It is critical to account for dynamic changes in phenotypes and underlying mechanisms. This is particularly important for complex, developmental phenotypes such as alcohol use, a

behavior that transitions from taboo, to tolerated, to treasured, over adolescence and young adulthood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This work was supported by National Institute on Alcohol Abuse and Alcoholism Grant AA023133

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

References

- Abar C, Turrisi R. How important are parents during the college years? A longitudinal perspective of indirect influences parents yield on their college teens' alcohol use. *Addict Behav.* 2008; 33:1360–1368. [PubMed: 18635318]
- Aliev F, Wertherill L, Bierut L, Bucholz KK, Edenberg H, Foroud T, ... Dick DM. Genes associated with alcohol outcomes show enrichment of effects with broad externalizing and impulsivity phenotypes in an independent sample. *J Stud Alcohol Drugs.* 2015; 76:38–46. [PubMed: 25486392]
- Agrawal A, Lynskey MT, Heath AC, Chassin L. Developing a genetically informative measure of alcohol consumption using past 12 month indices. *J Stud Alcohol Drugs.* 2011; 72:444–452. [PubMed: 21513681]
- Adkins DE, Clark SL, Copeland WE, Kenedy M, Conway K, Angold A, Maes H, Liu Y, Kumar G, Erkanil A, Patkar AA, Silberg J, Brown TH, Fergusson DM, Horwood LJ, Eaves L, van den Oord EJCG, Sullivan PF, Costello EJ. Genome-wide meta-analysis of longitudinal alcohol consumption across youth and early adulthood. *Twin Res Hum Genet.* 2015; 18:335–347. [PubMed: 26081443]
- Barnes GM, Reifman AS, Farrell MP, Dintcheff BA. The effects of parenting on the development of adolescent alcohol misuse: A six-wave latent growth model. *J Marriage Fam.* 2000; 62:175–186.
- Bell RQ. Convergence: an accelerated longitudinal approach. *Child Dev.* 1953; 24:145–52. [PubMed: 13141335]
- Bosasri B, Mourphy JG, Barnett NP. Predictors of alcohol use during the first year of college: Implications for prevention. *Addict Behav.* 2007; 32:2062–2086. [PubMed: 17321059]
- Bouteress K, Chassin L, Lemery-Chalfant K. Parent and peer influences on emerging adult substance use disorder: A genetically informed study. *Dev Psychopath.* 2016
- Brechwald WA, Prinstein MJ. Beyond homophily: A decade of advances in understanding peer influence processes. *J Res Adolesc.* 2011; 21:166–179. [PubMed: 23730122]
- Chartier KG, Hesselbrock MN, Hesselbrock VM. Development and vulnerability factors in adolescent alcohol use. *Child Adolesc Psychiatr Clin N Am.* 2010; 19:493–504. [PubMed: 20682217]
- Clark DB, Kirisci L, Mezzich A, Chung T. Parental supervision and alcohol use in adolescence: Developmentally specific interactions. *J Dev Behav Pediatr.* 2008; 29:285–292. [PubMed: 18562983]
- Cooke ME, Meyers JL, Latvala A, Korhonen T, Rose RJ, Kaprio J, Salvatore JE, Dick DM. Gene-environment interaction effects of peer deviance, parental knowledge, and stressful life events on adolescent alcohol use. *Twin Res Hum Genet.* 2015; 18:507–517. [PubMed: 26290350]
- Dawson DA, Grant BF, Stinson FS, Chou PS. Maturing out of alcohol dependence: The impact of transitional life events. *J Stud Alcohol.* 2006; 67:195–203. [PubMed: 16568565]

- Deutsch AR, Slutske WS, Lynskey MT, Bucholz KK, Madden PAF, Heath AC, Martin NG. From alcohol initiation to tolerance to problems: Discordant twin modeling of a developmental process. *Dev Psychopath.* in press.
- Deutsch AR, Slutske WS, Richmond-Rakerd LS, Chernyavskiy P, Heath AC, Martin NG. Causal influence of age at first drink on alcohol involvement in adulthood and its moderation by familial context. *J Stud Alcohol Drugs.* 2013; 74:703–713. [PubMed: 23948529]
- Dick DM. Developmental changes in genetic influences on alcohol use and dependence. *Child Dev Perspect.* 2011; 5:223–230.
- Dick DM, Cho SB, Latendresse SJ, Alev F, Nurnberger JI, Edenberg HJ, ... Kuperman S. Genetic influences on alcohol use across stages of development: GABRA2 and longitudinal trajectories of drunkenness from adolescence to young adulthood. *Addict Biol.* 2014; 19:1055–1064. [PubMed: 23692184]
- Dick DM, Pagan JL, Viken R, Purcell S, Kaprio J, Pulkkinen L, Rose RJ. Changing environmental influences on substance use across development. *Twin Res Hum Genet.* 2007; 10:315–326. [PubMed: 17564520]
- Dick DM, Rose RJ, Viken RJ, Kaprio J, Koskenvuo M. Exploring gene-environment interactions. Socioregional moderation of alcohol use. *J Abnorm Psychol.* 2001; 110:625–632. [PubMed: 11727951]
- Dube SR, Miller JW, Brown DW, Giles WH, Felitti VJ, Dong M, Anda RF. Adverse childhood experiences and the association with ever using alcohol and initiation alcohol use during adolescence. *J Adolesc Health.* 2006; 38:444e1–444.e10. [PubMed: 16549308]
- Edwards AC, Kendler KS. Alcohol consumption in men is influenced by qualitatively genetic factors in adolescence and adulthood. *Psychol Med.* 2013; 43:1857–1868. [PubMed: 23282961]
- Edwards AC, Maes HH, Prescott CA, Kendler KS. Multiple mechanisms influencing the relationship between alcohol consumption and peer alcohol use. *Alcohol Clin Exp Res.* 2015; 39:324–322. [PubMed: 25597346]
- Edwards AC, Heron J, Vladimirov V, Wolen AR, Adkins DE, Aliev F, Hickman M, Kendler KS. The rate of change in alcohol misuse across adolescence is heritable. *Alcohol Clin Exp Res.* 2016; 41:57–64. [PubMed: 27892595]
- Ennett ST, Foshee VA, Buman KE, Hussong A, Cai L, Luz H, ... DuRant R. The social ecology of adolescent alcohol misuse. *Child Dev.* 2008; 79:1777–1791. [PubMed: 19037949]
- Fowler JH, Settle JE, Christakis NA. Correlated genotypes in friendship networks. *PNAS.* 2011; 108:1993–1997. [PubMed: 21245293]
- Fowler T, Shelton K, Lifford K, Rice F, McBride A, Nikolov I, ... van der Bree MBM. Genetic and environmental influences on the relationship between peer alcohol use and own alcohol use in adolescents. *Addict.* 2007; 102:894–903.
- Franic S, Dolan C, Borsboom D, Hudziak JJ, van Beijsterveldt CEM, Boomsma DI. Can genetics help psychometrics? Improving dimensionality assessment through genetic factor modeling. *Psychol Meth.* 2013; 18:406–433.
- Grimm, KJ., Ram, N., Estabrook, R. Growth modeling: Structural equation and multilevel modeling approaches. New York: Guilford; 2017.
- Harden KP, Hill JE, Turkheimer E, Emery RE. Gene-environment correlation and interaction in peer effects on adolescent alcohol and tobacco use. *Behav Genet.* 2008; 38:339–347. [PubMed: 18368474]
- Harris KM, Halpern CT, Smolen A, Haberstick BC. The national longitudinal study of adolescent health (Add Health) twin data. *Twin Res Hum Genet.* 2006; 9:988–997. [PubMed: 17254442]
- Irons DE, Iacono WG, Oetting WS, McGue M. Developmental trajectory and environmental moderation of the effect of ALDH2 polymorphism on alcohol use. *Alcohol Clin Exper Res.* 2012; 36:1882–1891. [PubMed: 22563891]
- Kendler KS, Gardner C, Dick DM. Predicting alcohol consumption in adolescence from alcohol-specific and general externalizing genetic risk factors, key environmental exposures, and their interaction. *Psychol Med.* 2011; 41:1507–1516. [PubMed: 20942993]

- Kendler KS, Jacobson KC, Gardner CO, Gillepsie N, Aggen SA, Prescott CA. Creating a social world: A developmental twin study of peer-group deviance. *Arch Gen Psychiatry*. 2007; 64:958–965. [PubMed: 17679640]
- Kendler KS, Schmitt E, Aggen SH, Prescott CA. Genetic and environmental influences on alcohol caffeine cannabis and nicotine use from early adolescence to middle adulthood. *Arch Gen Psychiatr*. 2008; 65:674–682. [PubMed: 18519825]
- Koopmans JR, Boomsma DI. Familial resemblances in alcohol use: genetic or cultural transmission? *Journal of Studies on Alcohol*. 1996; 57:19–28. [PubMed: 8747497]
- Latendresse SJ, Rose RJ, Viken RJ, Pulkkinen L, Kaprio J, Dick DM. Parenting mechanisms in links between parents' and adolescents' alcohol use behaviors. *Alcohol Clin Exper Res*. 2008; 32:322–330. [PubMed: 18162066]
- McGue M, Sharma A, Benson P. Parent and sibling influences on adolescent alcohol use and misuse: Evidence from a U.S. adoption cohort. *J Stud Alcohol*. 1996; 57:8–18. [PubMed: 8747496]
- Masten AS, Cicchetti D. Developmental cascades. *Dev Psychopath*. 2010; 22:491–495.
- Meyers JL, Salvatore JE, Vuoksima E, Korhonen T, Pulkkinen L, Rose RJ, ... Dick DM. Genetic influences on alcohol use behaviors have diverging developmental trajectories: A prospective study among male and female twins. *Alcohol Clin Exper Res*. 2014; 38:2869–2877. [PubMed: 25421521]
- Miles DR, Silberg JL, Pickens RW, Eaves LJ. Familial influences on alcohol use in adolescent female twins: Testing for genetic and environmental influences. *J Stud Alcohol*. 2005; 66:445–451. [PubMed: 16240551]
- Muthén, LK., Muthén, BO. *Mplus User's Guide*. 7. Los Angeles, CA: Muthén & Muthén; 1998–2012.
- Nash SG, McQueen A, Bray JH. Pathways to adolescent alcohol use: Family environment peer influence and parental expectations. *J Adolesc Health*. 2005; 37:19–28. [PubMed: 15963903]
- Newsom, JT. *Longitudinal structural equation modeling*. New York: Routledge; 2015.
- Osgood DW, Ragan DT, Wallace L, Gest SD, Feinberg ME, Moody J. Peers and the emergence of alcohol use: Influences and selection processes in adolescent friendship networks. *J Res Adolesc*. 2013; 23:500–512.
- Pagan JL, Rose RJ, Viken RJ, Pulkkinen L, Kaprio J, Dick DM. Genetic and environmental influences on stages of alcohol use across adolescence and into young adulthood. *Behav Genet*. 2006; 36:483–497. [PubMed: 16586152]
- Poelen EAP, Derks EM, Engles CMER, Van Leeuwe JFJ, Scholte RHJ, Willemsen G, Boomsma D. The relative contribution of genes and environment to alcohol use in early adolescents: Are similar factors related to initiation of alcohol use and frequency of drinking? *Alcohol Clin Exper Res*. 2008; 32:975–982. [PubMed: 18445102]
- Poelen EAP, Scholte RHJ, Willemsen G, Boomsma DI, Engels RCME. Drinking by parents, siblings and friends as predictors of regular alcohol use in adolescents and young adults: A longitudinal twin-family study. *Alcohol Alcoholism*. 2007; 42:362–369. [PubMed: 17537828]
- Prescott CA, Aggen SH, Kendler KS. Sex differences in the source of genetic liability to alcohol abuse and dependence in a population-based sample of U S twins. *Alcohol Clin Exper Res*. 1999; 23:1136–1144. [PubMed: 10443978]
- Rose RJ, Dick DM, Viken RJ, Kaprio J. Gene-environment interaction in patterns of adolescent drinking: Regional residency moderates longitudinal influences on alcohol use. *Alcohol Clin Exper Res*. 2001; 25:637–643. [PubMed: 11371711]
- Salvatore JE, Aliev F, Bucholz K, Agrawal A, Hesselbrock V, Hesselbrock M, ... Dick DM. Polygenic risk for externalizing disorder: Gene-by-development and gene-by environment effects in adolescents and young adults. *Clin Psychol Sci*. 2014; 3:189–201.
- Sartor CE, Lynskey MT, Heath AC, Jacob T, True W. The role of childhood risk factors in initiation of alcohol use and progression to alcohol dependence. *Addiction*. 2007; 102:216–225. [PubMed: 17222275]
- Seglem KB, Waaktaar T, Ask H, Torgersen S. Sex differences in genetic and environmental contributions to alcohol consumption from early adolescence to young adulthood. *Addiction*. 2016; 111:1188–1195. [PubMed: 26802679]

- Sher, KJ. Children of alcoholics: A critical appraisal of theory and research. Chicago: University of Chicago Press; 1991.
- Scholte RHJ, Poelen EAP, Willemsen G, Boomsma DI, Engels RCME. Relative risks of adolescent and young adult alcohol use: The role of drinking fathers, mothers, siblings and friends. *Addict Behav.* 2008; 33:1–14. [PubMed: 17490824]
- Staff J, Greene KM, Maggs JL, Schoon. Family transitions and changes in drinking from adolescence through midlife. *Addiction.* 2014; 109:227–236. [PubMed: 24571025]
- Van Beek JHDA, Kendler KS, de Moor MHM, Geels LM, Bartels M, Vink JM, ... Boomsma DI. Stable genetic effects on symptoms of alcohol abuse and dependence from adolescence into early adulthood. *Behav Genet.* 2012; 42:40–56. [PubMed: 21818662]
- Van der Vorst H, Engels RCME, Meeus W, Dekovic M, Vermulst A. Parental attachment parental control and early development of alcohol use: A longitudinal study. *Psychol Addict Behav.* 2006; 20:107–116. [PubMed: 16784352]
- Van Zundert RMP, Van Der Vorst H, Vermulst AA, Engels RMC. Pathways to alcohol use among Dutch students in regular education and education for adolescents with behavioral problems: The role of parental alcohol use general parenting practices and alcohol-specific parenting practices. *J Fam Psychol.* 2006; 20:456–567. [PubMed: 16938004]
- Wang, T., Wood, PK., Heath, AC. Can psychometric measurement models inform behavior genetic models? A Bayesian model comparison approach. In: Stemmler, M., et al., editors. *Dependent Data in Social Sciences Research*. 2015. p. 231-259. *Proceedings in Mathematics and Statistics* 145
- Windle M. Parental sibling and peer influences on adolescent substance use and alcohol problems. *Applied Developmental Science.* 2000; 4:98–110.
- White HR, Jackson K. Social and psychological influences on emerging adult drinking behavior. *Alcohol Res Health.* 2004; 28:182–190.
- Wood MD, Read JP, Mitchell RE, Brand NH. Do parents still matter? Parent and peer influences on alcohol involvement among recent high school graduates. *Psychol Addict Behav.* 2004; 18:19–30. [PubMed: 15008682]
- Young-Wolff KC, Enoch M, Prescott CA. The influence of gene-environment interactions on alcohol consumption and alcohol use disorders: A comprehensive review. *Clin Psychol Rev.* 2011; 31:800–816. [PubMed: 21530476]

Figure 1.
Psychometric biometric twin/sibling model examining additive genetic common environment and unique environment of alcohol use over ages bins

Figure 2.
Standardized means for alcohol use friends' use autonomy granting and maternal support from ages 13 – 27.

Figure 3.

Model predicted means for latent phenotypic biometric model for alcohol use across adolescence and young adulthood.

Figure 4. Variance attributed to etiological latent factors (early and overall additive genetic early and overall unique environment and overall common environment) for alcohol use from adolescence to young adulthood.

Table 1

Percentage of twin/sibling pairs in sample as well as percentage of same and opposite sex pairs within each twin/sibling group.

Sibling pair type	N (percent of sample)	Gender composition			Age discordance mean (SD)
		Female same sex (%)	Male same sex (%)	Opposite sex (%)	
Monozygotic Twins	328 (13.4%)	49.69	50.31	---	---
Dizygotic Twins	498 (20.35%)	25.56	30.47	43.96	---
Full Siblings	1183 (48.34%)	30.23	27.43	42.33	2.22 (1.15)
Half Siblings	438 (17.90%)	24.76	26.67	48.57	2.72 (1.38)

Table 2

Twin/Sibling within-pair correlations for alcohol use for each age bin

Age Bins	Monozygotic Twins	Dizygotic Twins	Full Siblings [^]	Half Siblings [^]
13–14	0.44** (n=67)	0.43** (n=120)	0.33* (n=53)	0.06 (n=25)
15–16	0.57** (n=153)	0.46** (n=229)	0.27** (n=208)	0.12 (n=56)
17–18	0.69** (n=135)	0.42** (n=188)	0.48** (n=242)	0.35** (n=56)
19–20	0.68** (n=59)	0.35** (n=100)	0.28* (n=60)	0.51** (n=36)
21–22	0.51** (n=73)	0.44** (n=91)	0.40* (n=40)	0.06 (n=10)
23–24	0.52** (n=53)	0.07 (n=77)	0.07 (n=51)	0.65 (n=4)
25–27	0.59** (n=46)	0.22* (n=98)	0.52** (n=57)	–0.04 (n=26)

[^] Note: Full and half sibling cells have lower sample sizes due to cell discordance (i.e., siblings are in two different age bins).

Table 3

Model fit scores for comparisons among all phenotypically-based biometric models.

Model	χ^2	Df	CFI	RMSEA (95% CI)	SRMR	χ^2 Difference Tests
1. Three factors (One A C and E)	740.54	417	0.89	0.05 (0.05 – 0.06)	0.14	-
2. Six factors (Two A C and E)	670.53	402	0.91	0.03 (0.03 – 0.04)	0.14	1 vs 2: χ^2 (15) = 70.01
3. Five factors (Early A dropped)	700.66	407	0.90	0.03 (0.03 – 0.04)	0.14	3 vs 2: χ^2 (5) = 30.13
4. Five factors (Early E dropped)	682.17	407	0.90	0.03 (0.03 – 0.04)	0.14	4 vs 2: χ^2 (5) = 11.64
5. Five factors * (Early C dropped)	677.55	407	0.91	0.03 (0.03 – 0.04)	0.14	5 vs 2: χ^2 (5) = 7.02
6. Five factors with gender associations for overall A and overall E	871.31	517	.89	0.03 (0.03 – 0.04)	0.12	5 vs 6: χ^2 (110) = 193.76

Note: A = Additive Genetic, C = Common Environment, E = Unique Environment, CFI = Comparative Fit Index, RMSEA = Root Mean Square Error of Approximation, SRMR = Standardized root mean square residual

* best fitting model

Table 4

Unstandardized item loadings and factor means for the best – fitting phenotypic biometric model

Age	Early Additive Genetic	Overall Additive Genetic	Early Unique Environment	Overall Unique Environment	Overall Common Environment
13–14	0.11	–0.29**	0.41**	0.14**	0.31**
15–16	0.46**	–0.19	0.13*	0.31**	0.35**
17–18	0.57**	0.23	0.25**	0.33**	0.37**
19–20	0.24**	0.52**	0.28**	0.25**	0.41**
21–22	-	0.30**	-	0.30**	0.49**
23–24	-	0.26**	-	0.43**	0.36**
25–27	-	0.32**	-	0.39**	0.37**
Factor Mean	–0.12	0.81	–0.77**	–0.04	0.14

Table 5

Item loadings means and correlations between biometric latent etiologies (additive genetic, common environment and unique environment) at each age bin explaining alcohol use with measured environments (friend use, maternal closeness and autonomy granting).

	Measured Environment	Overall Additive Genetic	Early Additive Genetic	Overall Unique Environment	Early Unique Environment	Overall Common Environment	With Item
1314							
	Friend Use	0.09	0.21**	0.01	0.35**	0.29**	0.24**
	Maternal Closeness	-0.06	-0.05	0.10	0.02	-0.02	-0.01
	Autonomy Granting	0.04	0.03	0.01	-0.01	0.01	0.01
1516							
	Friend Use	0.06	0.15	-0.16	-0.92	0.33	0.27**
	Maternal Closeness	-0.37	0.19	-0.47	-0.74	0.05	-0.04**
	Autonomy Granting	0.16	-0.02	0.18	0.38	0.02	0.01
1718							
	Friend Use	-0.03	-0.40**	0.35**	-0.07	0.31**	0.33**
	Maternal Closeness	-0.02	0.02	-0.11**	0.17	-0.01	-0.03**
	Autonomy Granting	0.02	0.04	0.06	0.06	0.02	0.02*
1920							
	Friend Use	0.34**	0.09	0.11	0.23*	0.40**	.32**
	Maternal Closeness	0.01	0.07	-0.09	-0.01	-0.01	-0.01
	Autonomy Granting	-	-	-	-	-	-
2122							
	Friend Use	-0.19	-	0.55**	-	0.34**	0.41**
	Maternal Closeness	-0.03	-	-0.08	-	0.03	-0.01
2324							
	Friend Use	0.03	-	0.45*	-	0.24**	0.36**
	Maternal Closeness	-0.01	-	0.02	-	-0.02	0.01
2527							
	Friend Use	0.13	-	0.62**	-	0.39**	0.29**
	Maternal Closeness	0.01	-	-0.10	-	-0.05	-0.04**

Table 6
Model fit indices for measured environment models correlating parenting practices and friend use at each age by model factors or factor items.

Model (covariates included by age)	χ^2 (DF)		CFI		RMSEA		SRMR	
	Correlated with Factor	Correlated with Item	Correlated with Factor	Correlated with Item	Correlated with Factor	Correlated with Item	Correlated with Factor	Correlated with Item
13–14 covariates	1520.89 (806)	2387.30 (898)	0.81	0.66	0.04	0.05	0.15	0.18
15–16 covariates	1549.16 (830)	2025.34 (842)	0.83	0.72	0.04	0.05	0.11	0.16
17–18 covariates	1582.13 (819)	2077.71 (831)	0.83	0.72	0.04	0.05	0.14	0.17
19–20 covariates	1161.47 (633)	1979.36 (966)	0.86	0.75	0.04	0.04	0.14	0.18
21–22 covariates	1201.68 (653)	1594.25 (799)	0.85	0.80	0.04	0.04	0.15	0.16
23–24 covariates	1082.20 (633)	1617.93 (779)	0.87	0.85	0.03	0.04	0.15	0.17
25–27 covariates	1136.37 (630)	1453.99 (776)	0.85	0.81	0.04	0.04	0.17	0.17