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RUNNING HEAD: PEERS AND ALCOHOL FROM 11 TO 34

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Co-development of alcohol use problems and antisocial peer affiliation from ages 11 to 34: Selection, socialization, and genetic and environmental influences

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Abstract

Background and aims: Social context is an important factor in determining the developmental trajectory of alcohol use. We examined the co-development between alcohol use problems and antisocial peer affiliation. We also estimated the genetic and environmental influences on alcohol use problems, antisocial peer affiliation, and their co-development over time.

Design: Longitudinal study using bivariate latent basis models with structured residuals (LBM-SR). A biometric model was then fit to estimate the genetic and environmental influences on the growth factors and their covariances.

Setting: The United States mid-west region.

Participants: Members of the Minnesota Twin Family Study (MTFS), an ongoing, longitudinal study of 3762 (52% female) twins (1,881 pairs).

Measurements: Alcohol use problems were assessed using a composite measure of average number of drinks per occasion in the past 12 months, maximum number of drinks in 24 hours, and *DSM-III-R* symptoms of alcohol abuse and dependence. Antisocial peer affiliation was measured by self-report of the proportion of one's friends that exhibited types of antisocial behaviors.

Findings: The LBM-SR model revealed that there was a large correlation between the growth factors for alcohol use problems and antisocial peer affiliation (r = .78, 95% Confidence Interval [CI]: .76, ,80) and cross-lagged effects consistent with both selection and socialization effects. Additionally, antisocial peer affiliation in adolescence was associated with greater increases in

alcohol use problems over time (r = .57, 95% CI: .54, .60). Genetic influences largely accounted for the association between antisocial peer affiliation in pre-adolescence and growth in alcohol use problems, while shared environmental influences accounted for the correlation between antisocial peer affiliation and alcohol use problems growth factors.

Conclusions: Antisocial peer affiliation in adolescence appears to be a salient, genetically-influenced risk factor for early alcohol use and increase in alcohol use from adolescence through young adulthood.

Alcohol use is intimately tied to social context; consequently, alcohol use problems are strongly tied to socializing with peers who consistently break rules and violate norms (1–5). Given their entwined nature, understanding the association between problem drinking and antisocial peer affiliation requires taking a developmental perspective. In the United States, alcohol use and alcohol use problems exhibit normative age-related patterns, wherein alcohol use typically initiates in middle adolescence (ages 15-17), increases through late adolescence and peaks during emerging adulthood, with onset of alcohol use disorders (ages 18-25), followed by large decreases in alcohol use problems in young adulthood (ages 25-35) (6,7). Mean-levels of antisocial peer affiliation follow the same age-related pattern, and antisocial peers are one of the strongest predictors of persistent, heavy alcohol use (8,9). Further, changes in alcohol use problems are strongly correlated with changes in antisocial peers (10), suggesting common vulnerability processes that link the two over the course of development, some of which may be present even before a person's first drink, such as a disinhibited temperament and adverse home environment.

While antisocial peer affiliation and its link to alcohol use problems are often conceptualized as social-contextual processes, peer characteristics are also heritable (11), as are alcohol use problems (12). Further, much of the association between alcohol use problems and antisocial peer affiliation can be attributed to shared genetic influences (13), and genetic influences on substance use problems increase in contexts of more antisocial peers (14).

Consequently, the mechanisms underlying the association between antisocial peer affiliation and alcohol use problems include both genetic and environmental influences.

In addition to sharing common risk factors, alcohol use and antisocial peer affiliation may directly influence each other through within-person processes, and there is evidence for both *selection* (i.e., people that enjoy drinking seek out like-minded peers) and *socialization* (i.e., spending time with drinking peers establishes norms for drinking) with studies finding evidence for both processes (10,15–22). This is consistent with the idea that genetic influences may play a role on selection effects, either through youth selecting into alcohol permissive environments or friendships (i.e., active rGE) or through heritable expression of antisocial behavior evoking increased engagement from antisocial peers (i.e., evocative rGE (23)). However, most of these studies on selection and socialization began after participants had initiated alcohol use (20,21), making it difficult to establish the temporal precedence of alcohol use and antisocial peer affiliation. Furthermore, there is evidence that the reciprocal influences between alcohol use problems and antisocial peer affiliation change over time. For example, the socialization effects of antisocial peers may be stronger in adolescence and emerging adulthood relative to young adulthood (22).

Because alcohol use problems and antisocial peer affiliation are so enmeshed, no study has provided a comprehensive analysis that parses the various aspects of their co-development. Such an analysis requires (1) accounting for the overlap in their normative age-related change, (2) estimating genetic and environmental influences on these changes, and (3) delineating

within-person processes of selection and socialization. Further, it is necessary to cover a wide age range, ideally, prior to the initiation of alcohol use and extending into young adulthood, past the period of greatest risk when patterns of persistent versus desistent alcohol use problems have been established. Also, most prior research has used a variant of the cross-lagged panel design that confounds between- (i.e., normative change) and within- (i.e., selection and socialization) person effects (21,24).

We addressed this by examining the co-development between alcohol use problems and antisocial peer affiliation in a longitudinal twin sample, beginning prior to the initiation of alcohol use (age 11) and extending 23 years into young adulthood, past the period of greatest risk (age 34), with assessments timed to key developmental phases in alcohol use. We used bivariate latent basis models with structured residuals (25) to parse between- and within-person effects. That is, the latent basis portion of the model was used to account for the normative age-related trends in alcohol use problems and antisocial peer affiliation while the residual structure was used to model within-person processes. The cross-lagged associations within the residual structure then allowed us to test for both socialization (significant antisocial peers to alcohol use paths) and selection (significant alcohol use to antisocial peers paths) effects. We also estimated the genetic and environmental influences on alcohol use problems and antisocial peer affiliation and their co-development over time.

Methods

Participants

Participants were members of the Minnesota Twin Family Study (MTFS), an ongoing, longitudinal study of 3762 (52% female) twins (1,881 pairs) investigating the development of mental health, substance use, and psychosocial adjustment (26,27). All twin pairs were the same sex and living with at least one biological parent at the time of recruitment, living within driving distance to the University of Minnesota laboratories. Exclusion criteria included any cognitive or physical disability that would interfere with study participation. Twins were first recruited when they either turned 11-years old (n = 2510; the younger cohort) or 17-years old (n = 1252; the older cohort). Twins in the younger cohort were born between the years 1977 to 1984 and 1988 to 1994, while twins in the older cohort were born between 1972 and 1979. Families were representative of the area they were drawn from in terms of socioeconomic status, history of mental health treatment, and urban vs rural residence (26). Consistent with the demographics of Minnesota for the target birth years, 96% of participants reported white non-Hispanic race/ethnicity.

Participation rates varied due to attrition (participants who missed an assessment were still recruited for later assessments) and availability of funding, and current age of participants, but ranged from 80% to 93% among those recruited for a given assessment. The younger cohort included 395 male and 394 female monozygotic (MZ) twin pairs, and 220 male and 246 female dizygotic (DZ) twin pairs. The older cohort included 190 male and 226 female MZ twin pairs, and 99 male and 111 female dizygotic twin pairs. Zygosity was confirmed by genome wide genotyping (28).

Alcohol use problems. Alcohol use problems were assessed using a composite measure of average number of drinks per occasion in the past 12 months, maximum number of drinks in 24 hours, and *DSM-III-R* symptoms of alcohol abuse and dependence (the diagnostic system when the study began). Each measure was assessed during structured clinical interviews with trained staff, while the average number of drinks and maximum number of drinks measures were also assessed using a computerized self-report questionnaire at ages 11, 14, and 17 that was completed in private. Free responses to the average quantity and maximum drinks, as well as the number of alcohol abuse and dependence symptoms were converted to scales that ranged from 0 to 8, and the mean of these values was used for participants' alcohol use problems score at each age (median Cronbach's α = .76; Supplemental Table 1)[1]. Twins in the older cohorts (born between 1972 and 1984) scored significantly higher on the Alcohol Use Problems scale at age 14 than the younger cohort (born between 1988 and 1994; Cohen's d =. 22, $p < .001$). There were no other statistically significant cohort differences on the Alcohol Use Problems scale.

Antisocial Peer Affiliation

At each assessment, twins rated the proportion of their friends (1 = *none of my friends are like that*, to 4 = *all of my friends are like that*) that exhibited various types of antisocial behaviors (28). All analyses were also run after excluding the alcohol use item ($r > .95$ with the 6-item scale), and the results did not change (median Cronbach's α = .77; Supplemental Table 1).

[1] The "binning" of responses was performed to create a smoother distribution from the free response distribution and is common practice in measures of alcohol use (29). Participants retained their rank-ordering and there was a high correlation between the free response and converted scales (mean r = .79).

Twins in the older cohorts scored significantly higher on the Antisocial Peer Affiliation scale at ages 14 and 17 (Cohen's *d*'s = .22 and .22, *p*'s < .001). However, twins in the younger cohort scored significantly higher on the Antisocial Peer Affiliation scale at age 24 (Cohen's *d* = .17, *p* < .001).

Data Analytic Strategy

An overview of the data analytic strategy is presented here, for more complete details see the supplemental analytic details. All major analyses were conducted using Mplus version 8.2 (30) with full information maximum likelihood estimation (31). Confidence intervals were derived using percentile bootstrapping (with 1000 draws; clustering was accounted for in the phenotypic model bootstrap procedure), which is particularly effective when estimating confidence intervals with skewed variables (32). Latent basis models with structured residuals (LBM-SR; Figure 1A) were used to simultaneously model developmental trends in alcohol use and antisocial peer affiliation, and the time-specific dynamics between them (25,33). These models include intercept factors that reflect status at the first time point, and slope factors that reflect the rate of change over the course of the study (specified here as using a latent basis approach; 26). Intercept and slope factors were allowed to vary to capture individual differences in growth. The residual structure included occasion-specific latent factors that account for deviations from the intercept and slope implied trajectories. The autoregressive (e.g., within trait association between age 11 and 14) and cross-lagged (e.g., cross-trait association between age 11

and 14) paths linking adjacent residual factors thus capture associations (i.e. selection and socialization effects) between variables over time after accounting for general growth trends.

Univariate LBM-SR were first fit for alcohol use problems and antisocial peer affiliation separately, followed by a bivariate LBM-SR model that included both variables (Figure 1A). Autoregressive and cross-lagged paths in the residual structure initially varied across time. To identify a more parsimonious model, a series of parameter constraints were tested. First, we fit a model that included separate constraints for autoregressive paths and cross-lagged paths within major periods of development: adolescence (paths from age 11 through age 17), emerging adulthood (paths from age 17 through age 24), and young adulthood (paths from age 24 to age 34). These constraints imply invariance in the residual structure within, but not between, developmental periods. Second, we fit a model in which all corresponding coefficients over time were fixed to equality, implying invariance in the residual structure across time. Changes in model fit for the more constrained models were tested using differences in χ^2, CFI, and RMSEA.

A biometric model was then fit to estimate the genetic and environmental influences on the growth model factors and their covariances. Factor scores for the intercept and slope factors for antisocial peer affiliation and the slope factor for alcohol use problems was first estimated from the multivariate LBM-SR. Factor scores from the multivariate LBM-SR were used to reduce computational burden. The variance of the intercept and slope factor scores were decomposed into additive genetic variance (i.e., the proportion of variance attributed to genetic differences among individuals; a^2), shared environmental variance (i.e., the proportion of

variance attributed to environmental factors that contribute to familial similarity; c^2), and non-shared environmental variance (i.e., the proportion of variance attributed to environmental factors that contribute to differences among family members, including measurement error; e^2; 31). A multivariate model was fit to antisocial peer affiliation and alcohol use problems to estimate the genetic and environmental overlap between the intercept and slope factors (Figure 2). A behavioral genetic version of the LBM-SR was not used here as the model encountered serious convergence issues. In addition, ACE components are inherently between-person constructs, making them better suited conceptually for examining the between-person associations between alcohol use problems and antisocial peer affiliation. Analyses were not pre-registered and should be considered exploratory.

Results

Twins in the younger cohort were assessed at ages 11 (M_{age} = 11.78 years; SD = 0.43 years) and 14 (M_{age} = 14.90 years; SD = 0.31 years), and all twins were assessed at ages 17 (M_{age} = 17.85 years; SD = 0.64 years), 21 (M_{age} = 21.08 years; SD = 0.79 years), 24 (M_{age} = 24.87 years; SD = 0.94 years), and 29 (M_{age} = 29.43 years; SD = 0.67 years). A subset of twins from the younger cohort were also assessed at age 34 (n = 866; M_{age} = 34.62 years; SD = 1.30 years). Supplemental Table 1 provides the number of participants for each assessment and descriptive statistics for the study measures. The rank-order stability for adjacent time points ranged from r = 0.41 to 0.68 for alcohol use problems and from r = 0.54 to 0.67 for antisocial peer affiliation for ages 14 to 34. The associations were smaller between alcohol use problems at ages 11 and 14

(r = 0.16) and antisocial peer affiliation at ages 11 and 14 (r = 0.25), due to low variability in both measures at age 11. Alcohol use problems and antisocial peer affiliation were moderately correlated at each age (rs = 0.27 to 0.62, mean r = 0.50; Supplemental Table 1). Both univariate LBM-SR models fit the data well by conventional standards (35; Table 1, Figure 1B, 1C). Figure 1B and 1C display the model-estimated means for alcohol use problems and antisocial peer affiliation, which increased from age 11 to age 20 and then decreased from age 20 to age 34. There was almost no variance in the intercept factor for alcohol use problems at age 11, so this factor variance in the growth part of the model was fixed to 0 (i.e., on average there was little to no alcohol use at age 11, and the variance around this mean was negligible).

All variants of the bivariate LBM-SR model fit well based on conventional standards for absolute fit (Table 1). The partially or developmentally constrained model did not fit worse than the unconstrained model (Sartora-Bentler Chi-Square Difference = 26.98, p =.007). In contrast, the fit of the fully constrained model—indicative of no change in the reciprocal processes between alcohol use problems and antisocial peer affiliation over time—was substantially worse than the unconstrained and developmentally constrained models (Sartora-Bentler Chi-Square Difference = 181.36, p <.001) . Follow-up model comparisons reinforced that the cross-lagged and autoregressive paths in each developmental period were significantly different from each other (Supplemental Table 4 so the original developmentally constrained model was retained as a the final model (Figure 1A). Parameter estimates from the developmentally constrained model are presented in Table 2. This model included a large correlation between the two growth factors

(r = .78, 95% CI: .76, .80) as well as between the intercept of antisocial peer affiliation and the growth factor for alcohol use problems (r = .57, 95% CI: .54, .60).

Selection, Socialization, and Autoregressive Effects

Cross-lagged (selection and socialization) and autoregressive path coefficients are presented in Table 2. The cross-lagged paths among residual variables were statistically significant for all six age-variable combinations. In adolescence (ages 11 to 17), the cross-lagged paths were small in magnitude (Alcohol Use Problems on Antisocial Peer Affiliation b = .52; Antisocial Peer Affiliation on Alcohol Use Problems b = .09) . This indicates that youth with more antisocial peer affiliation reported slightly more alcohol use problems in subsequent waves of assessment and vice versa, even after accounting for their normative mean-level increases during this time and the stability of their residual scores. In emerging adulthood (age 17 to 24), the cross lagged paths were smaller than in adolescence (Alcohol Use Problems on Antisocial Peer Affiliation b = .15). Also, the path from antisocial peer affiliation to alcohol use problems was negative (b = -.04), indicating more antisocial peer affiliation predicted *less* alcohol problems at the next assessment. This negative association is inconsistent with the other cross-lagged effects as well as with the residual covariance (mean r = .29) and the zero-order correlations (mean r = .50) at these ages, suggesting an anomaly that is unlikely of substantive importance. The cross-lagged effects were also smaller in young adulthood relative to adolescence (Alcohol Use Problems on Antisocial Peer Affiliation b = .10; Antisocial Peer Affiliation on Alcohol Use Problems b = .04).

Biometric Associations Between Alcohol Use Problems and Antisocial Peer Affiliation Latent Basis Factors

Multivariate biometric analyses were performed on the latent slope and intercept factors from the bivariate LBM-SR. Table 3 contains the results from the multivariate biometric model as well as the phenotypic correlations between the factor scores. There was a large degree of genetic overlap between all three growth factors, with the first A factor (i.e., A1 in Figure 2) accounting for nearly all the genetic variance in the antisocial peer affiliation intercept (100%), antisocial peer affiliation slope (94.4%), and alcohol use problems slope (91.3%) factors. Although there was little shared environmental variance across all three growth factors, the C2 (Figure 2) factor accounted for 87.5% and 57.1% of the shared environmental variance in the antisocial peer affiliation and alcohol use problems slope factors, respectively. Together, these results indicate a set of common genetic influences underlying variation in early antisocial peer affiliations, as well as changes in antisocial peer affiliation and in alcohol use problems over time. Furthermore, similar shared environmental influences contributed to variability in change over time in antisocial peer affiliation and alcohol use problems.

Discussion

Because alcohol use problems and antisocial peer affiliation are so strongly linked, it has been difficult to disentangle the developmental processes that tie them together. Using a longitudinal twin design that spanned ages 11 to 34, we began to clarify their entwined growth processes. First, there was a large correlation between the slope or growth factors for alcohol use

problems and antisocial peer affiliation. That is, the two are not only strongly associated cross-sectionally, but change in relative lock-step over time, indicating that social contexts (especially peers) are essential to understanding a person's trajectory of alcohol use. Second, antisocial peer affiliation in adolescence (the intercept) was associated with greater increases in alcohol use problems over time, indicating early contextual influences have long-term effects on alcohol use trajectories. Third, genetic influences largely accounted for the association between antisocial peer affiliation in pre-adolescence and growth in both antisocial peer affiliation and alcohol use problems over time. In contrast, common shared environmental influences were only observed between the two slope factors. This indicates that even before a person's first drink, genetic vulnerabilities that contribute to alcohol use problems are being expressed in the form of associating with antisocial peer affiliation in pre-adolescence. Shared environmental influences then help to keep these two processes on a similar trajectory over time. While prior work has identified shared genetic influences on both antisocial peer affiliation and alcohol problems cross-sectionally, this is the first study to examine the genetic and environmental influences from adolescence into young adulthood (36).

Fourth, we also detected socialization and selection effects on alcohol use problems and antisocial peer affiliation over time. Both selection and socialization effects were detected during adolescence, emerging adulthood, and young adulthood, indicating small incremental effects for time-specific influences over and above the normative developmental trends. The one exception was the small, negative socialization effect in emerging adulthood, indicating that a greater level

of antisocial affiliation at this age relative to a person's typical level was associated with slight decreases in alcohol use problems in subsequent assessments. Due to the inconsistency with all other effects and the complexity of the model, we withhold making substantive interpretations about this small and unpredicted effect until it has been replicated.

The study had several limitations. First, antisocial peer affiliation was assessed via self-report rather than by collecting data directly from peers, possibly leading to an increased similarity between self- and peer behavior. Second, the multiple years between assessments limits the inferences that can be drawn about the reciprocal processes between antisocial peer affiliation and alcohol use, given that each can influence the other on a much shorter time scale. Third, the sample had limited racial and ethnic diversity, which limits the generalizability of these findings in non-white populations.

Despite these limitations, the current study provides the most comprehensive analysis of the co-development, heritability, and reciprocal influences between alcohol use problems and antisocial peer affiliation to date. These analyses estimated a large degree of overlap in age-related change between antisocial peer affiliation and alcohol use problems as well as the role that genetic and environmental influences play in creating a diathesis and context for these processes. We were also able to identify the roles of selection and socialization across development. These findings have important implications for the development of interventions, as understanding that early affiliation with antisocial peers overlaps with heritable risk for alcohol use problems over time may serve to identify "high-risk" children even before the onset

of alcohol use. Also, efforts to identify and target the mechanisms of these shared environmental influences can impact both alcohol use and affiliation with antisocial peers. For example, school-based interventions providing education regarding the risks of alcohol use and alternative coping and stress management strategies to peer groups that have previously been identified as engaging in early antisocial behavior may be particularly effective in mitigating later risk for alcohol use.

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Table 1. Indices of Fit of LBM-SR Models

	X^2	*df*	RMSEA	CFI	TLI	SRMR	BIC	SBIC	AIC	MLR Correction Factor
Univariate Models										
Alcohol Use Problems	54.69	14	.028 (.020, 036)	.987	.981	.039	45272.974	45206.25	45142.09	1.49
Antisocial Peer Affiliation	38.62	12	.024 (.016, .033)	.993	.987	.029	66456.805	66383.72	66313.76	1.18
Bivariate Models										
Unconstrained Model	249.11	54	.031 (.027, .035)	.978	.963	.052	108491.533	108284.99	108086.41	1.22
Developmentally								108278.53	108116.60	1.36
Constrained Model	262.95	66	.028 (.025, .032)	.978	.969	.056	108446.935			
Fully Constrained Model	425.90	74	.036 (.032, .039)	.960	.951	.077	108592.403	108449.41	108311.93	1.34

Note. Bivariate model fit statistics derived from non-nested model. In the unconstrained model, autoregressive and cross-lagged paths in the residual structure varied across time. The partially constrained model included constraints within major periods of development: adolescence (paths from age 11 through age 17), emerging adulthood (paths from age 17 through age 24), and young adulthood (paths from age 24 to age 34). In the fully constrained model all corresponding coefficients over time were fixed to equality, implying invariance in the residual structure across time.

Table 2. Path Coefficients from the Partially (Developmentally) Constrained LBM-SR Model

Variables	*b*	SE	95% CI
Cross-lagged effects			
Alcohol Use Problems →			
Antisocial Peer Affiliation			
Age 11 → Age 14 [a]	.52	.10	.35, .72
Age 14 → Age 17 [a]	.52	.10	.35, .72
Age 17 → Age 20 [b]	.15	.04	.07 .23
Age 20 → Age 24 [b]	.15	.04	.07 .23
Age 24 → Age 29 [c]	.10	.05	.01, .13
Age 29 → Age 34 [c]	.10	.05	.01, .13
Antisocial Peer Affiliation →			
Alcohol Use Problems			
Age 11 → Age 14 [d]	.09	.02	.05, .13
Age 14 → Age 17 [d]	.09	.02	.05, .13
Age 17 → Age 20 [e]	-.04	.02	-.07, -.01
Age 20 → Age 24 [e]	-.04	.02	-.07, -.01
Age 24 → Age 29 [f]	.04	.02	.01, .08
Age 29 → Age 34 [f]	.04	.02	.01, .08
Auto-regressive effects			
Alcohol Use Problems			
Age 11 → Age 14 [g]	.57	.07	.45, .71
Age 14 → Age 17 [g]	.57	.07	.45, .71
Age 17 → Age 20 [h]	.23	.03	.18, .28
Age 20 → Age 24 [h]	.23	.03	.18, .28
Age 24 → Age 29 [i]	.27	.04	.20, .35
Age 29 → Age 34 [i]	.27	.04	.20, .35
Antisocial Peer Affiliation			
Age 11 → Age 14 [j]	.31	.03	.26, .36
Age 14 → Age 17 [j]	.31	.03	.26, .36
Age 17 →Age 20 [k]	.05	.04	-.01, .14
Age 20 → Age 24 [k]	.05	.04	-.01, .14

Age 24 → Age 29 [l]	.36	.03	.30, .41
Age 29 → Age 34 [l]	.36	.03	.30, .41

Note. Paths [a-l] fixed to be equal within the best-fitting LBM-SR model. 95% confidence intervals derived via non-parametric percentile bootstrap with 1000 draws. *b* = unstandardized beta coefficient.

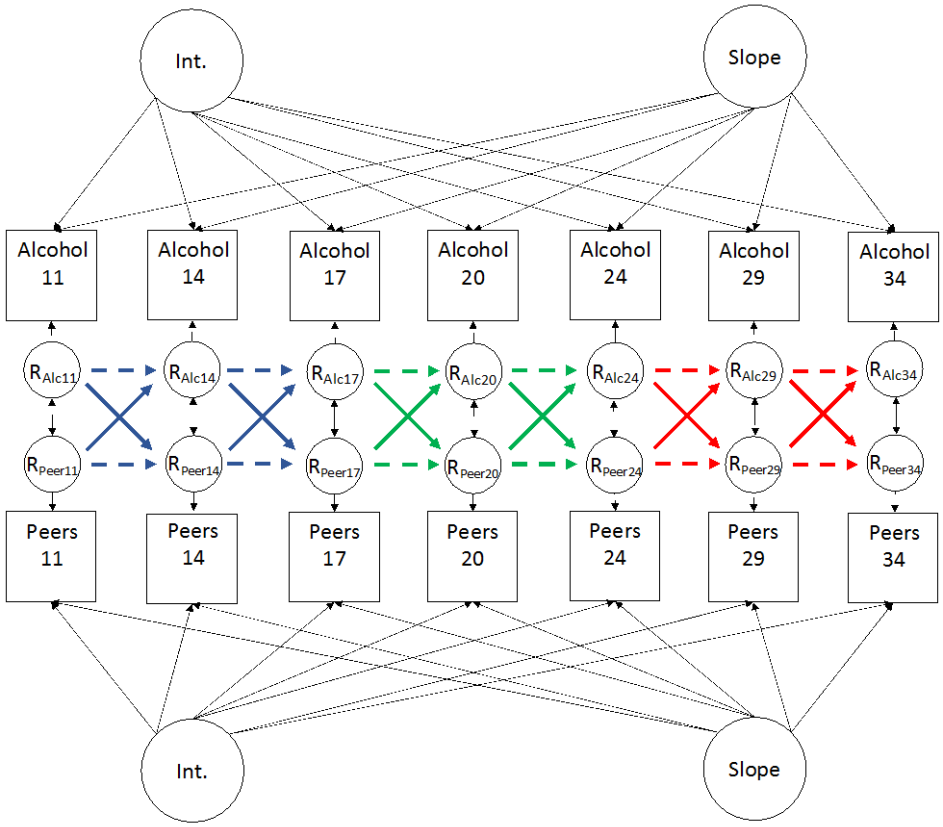
Table 3. Multivariate ACE Model Results for Growth Model Factor Scores

	Intercept Antisocial Peer Affiliation	**Slope Antisocial Peer Affiliation**	**Slope Alcohol Use Problems**
Phenotypic Correlations	*r* (95% CI)	*r* (95% CI)	
Slope Antisocial Peer Affiliation	.28 (.23, .33)		
Slope Alcohol Use Problems	.57 (.54, .60)	.78 (.76, .80)	
Cholesky Decomposition	(95% CI)	(95% CI)	(95% CI)
A1	.15 (.08, .39)	.34 (.08, .46)	.42 (.15, .55)
C1	.26 (.09, .34)	.04 (<.01, .20)	.10 (.02, .25)
E1	.60 (.49, .66)	<.01 (.00, .01)	.04 (.03, .06)
A2		.02 (.01, .30)	.04 (.00, .20)
C2		.28 (.09, .37)	.16 (.02, .23)
E2		.32 (.28, .35)	.09 (.08, .11)
A3			.00 (.00, .11)
C3			.02 (.00, .05)
E3			.12 (.11, .14)
Univariate Estimates			
A	.15 (.08, .39)	.37 (.25, .50)	.46 (.34, .59)
C	.26 (.09, .34)	.32 (.19, .41)	.28 (.16, .39)
E	.60 (.49, .66)	.32 (.29, .36)	.26 (.23, .29)

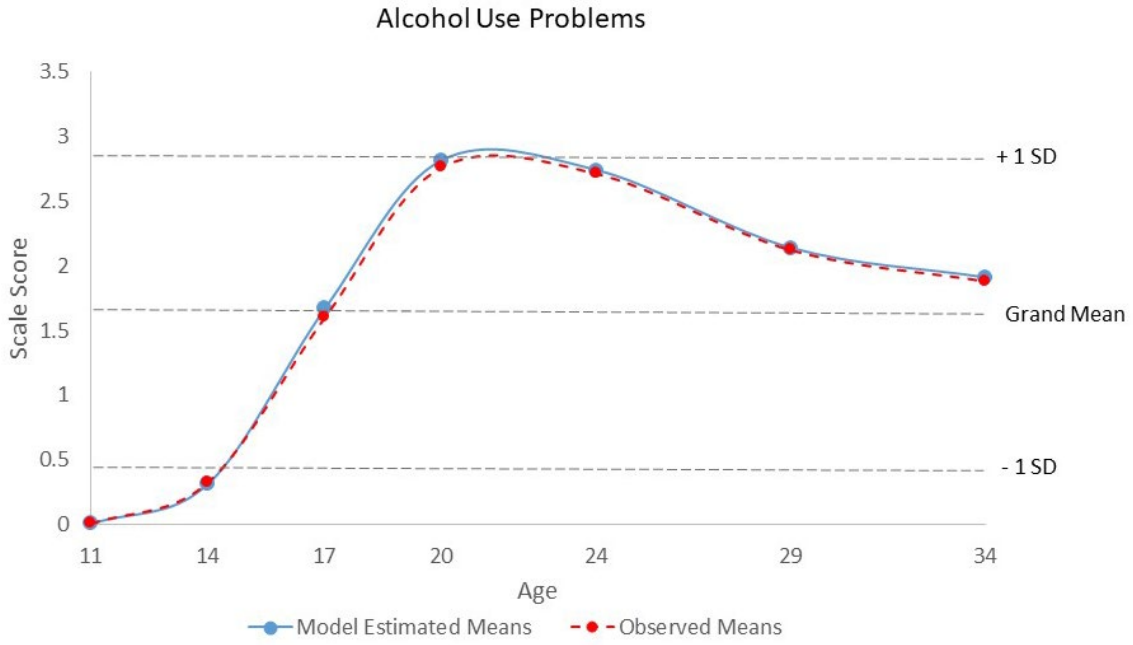
Note. A= additive genetic variance; C= shared environmental variance; E= non-shared environmental variance; all ACE model estimates are standardized; CI = Confidence Interval. 95% confidence intervals derived via non-parametric percentile bootstrap with 1000 draws. The sum of the ACE components in the columns for the intercept and slope is equal to the univariate estimates.

Figure 1. Bivariate Latent Growth Curve Model with Structured Residuals and Univariate Growth Curves. A) Schematic of the bivariate basis model and residual structure of the partially (developmentally) constrained bivariate latent basis model. Cross-lagged paths constrained to be equal are represented with same color solid lines and autoregressive paths constrained to be equal are represented with same color dashed lines. Alcohol = alcohol use problems variable for ages 11-34; Peers= antisocial peer affiliation variable for ages 11-34; R= residual factor; Int. = intercept factor, Slope= slope factor from latent basis model; Mean structure and variances/residual variances omitted from figure. Alcohol factor loadings from 11 to 34: 0, 0.150, 0.589, 1.052, 1.108, 0.974, 1.00; Peers factor loadings from 11 to 34: 0, 0.611, 1.082, 1.778, 1.480, 1.206, 1.00. B) Estimated model means (blue) from the univariate growth curve model for alcohol use problems and observed means (red). Horizontal dotted lines indicate the grand mean (M = 1.67) and one standard deviation above and below the grand mean (SD = 1.18). C) Estimated model means (blue) from the univariate growth curve model of antisocial peer affiliation and observed means (red). Horizontal dotted lines indicate the grand mean (M = 9.80) and one standard deviation above and below the grand mean (SD = 2.23).

A.



B.



C.

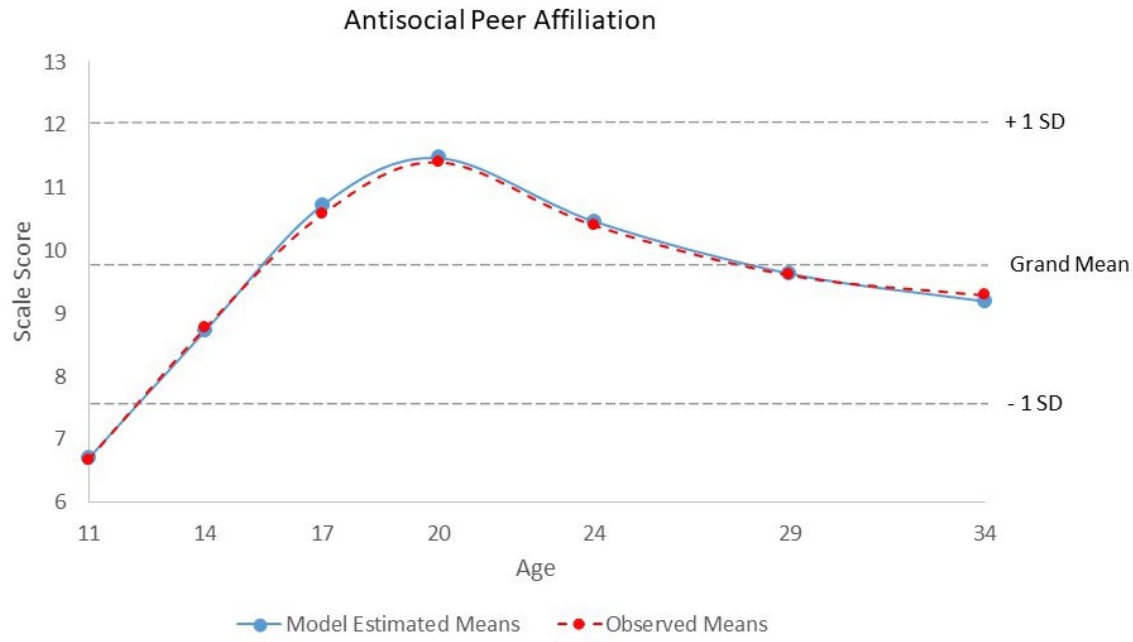


Figure 2. Schematic of Multivariate Biometric Model. A1-A3 = additive genetic variance components; C1-C3 = shared environmental variance components; E1-E3 = non-shared environmental variance components. Shared and non-shared environmental variance components, and mean structure, omitted from figure for clarity of presentation. Twin 2 model also not pictured. Two equivalent models were specified, one for twin 1 and one for twin 2, and covariances were added between the twin 1 and 2 A and C variance components. The covariance between the A components was fixed to either 1.0 (MZ twins) or 0.5 (DZ twins), and the covariance between the C components was fixed to 1.0 (MZ and DZ twins).