Genetic and Environmental Influences on Gambling and Substance Use in Early Adolescence

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Abstract

This study examined the genetic and environmental architecture of early gambling involvement and substance use to determine whether genetic or environmental factors that contribute to substance use also put young adolescents at risk for early involvement in gambling. Self-reports of substance use and gambling involvement were collected at age 13 years from 279 Monozygotic and Dizygotic twin pairs. Univariate ACE modeling revealed that genetic and nonshared environmental factors almost equally accounted for gambling involvement, with no contribution from shared environmental factors. In contrast, both shared and nonshared environmental factors played important roles in substance use; the contribution of genetic factors was also substantial. Bivariate analyses identified a significant, albeit modest, overlap between the genetic influence on gambling involvement and the genetic influence on substance use. The results shed light on the etiology of early gambling involvement and substance use, suggesting that preventive interventions targeting common risk factors may also need to be complemented by modules that are specific to each behavior.

Keywords: ACE modeling, gambling, substance use, early adolescence

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In many Western countries, one 13 year-old out of five has used drugs or alcohol during the past year and at least as many report participation in gambling activities (Johnston et al. 2013; Volberg et al. 2011). Substance use and gambling tend to co-occur throughout adolescence. Common risk factors inherent to the child (e.g., impulsivity) and common risk factors related to the child's social environment (e.g., affiliation with delinquent peers) have been found to contribute, at least partially, to the co-occurrence of gambling and substance use in adolescence (Barnes et al. 1999; Vitaro et al. 2001). The exact source of these common etiological factors, however, remains unknown. Impulsivity is moderately heritable, but environmental factors are also an important source of influence (Bezdjian et al. 2011). Environmental features, such as affiliation with delinquent peers, may also be influenced by genetic factors via a gene-environment correlation (Button et al. 2007). As a consequence, it is unclear the extent to which the co-occurrence of adolescent gambling and substance use is due to common underlying personal, heritable characteristics of the child or to common underlying influences from the child's environment. We address this issue with data from a genetically informed study of monozygotic and dizygotic adolescent twins raised together.

A number of studies indicate that shared environmental factors play an important role with respect to substance use in adolescence, with the rest of the variance explained by genetic and nonshared environmental factors (Hopfer et al. 2003; Kendler et al. 2008). Less is known about early gambling involvement (i.e., frequency of gambling behavior) and the evidence that is available during adolescence is limited and contradictory. The only study examining the gene-environment architecture of the frequency of adolescent gambling involvement found evidence

of considerable shared environmental influences, with minimal genetic contribution (Loehlin and Nichols 1976). Worth noting, however, is the fact that these data were collected more than 50 years ago, when gambling was less popular and less accepted than it is now. The other studies to examine the gene-environment architecture of gambling focused on adults. Two reports found that genetic factors played a significant role in gambling involvement (Slutske et al. 2009; Winters and Rich 1998), but a third found no evidence of genetic influence (Slutske and Richmond-Rakerd 2014). Instead, the latter found that gambling involvement was influenced by family (i.e., shared) and non-shared environmental factors.

To better understand the etiology of gambling involvement and substance use, a first important question that needs to be addressed is whether the relative contributions of genetic, shared and nonshared environmental factors to early adolescent gambling involvement are similar to those found for early substance use. A second, equally important question is whether the same genetic, shared and nonshared environmental factors that influence early gambling involvement also influence early substance use. There is some empirical support for the assertion that the same genetic factors and, to a lesser degree, the same environmental factors are involved in gambling and substance use. This support, however, is from adult studies and refers to gambling problems and substance\alcohol abuse and dependence (Slutske et al. 2000; Slutske et al. 2013). The degree to which these findings generalize to the initiation of gambling and substance use during early adolescence is not clear.

The answers to these questions have important theoretical and clinical consequences. First, knowledge about the role of genetic and environmental influences on gambling involvement (and substance use) during early adolescence, when most youth first engage in these behaviors will help to clarify initiation pathways. Second, delineation of the origin of the overlap

between early adolescent gambling and substance use will provide a clearer insight into the common etiology of these two behaviors than previous studies of adults, because the phenotypic association between gambling and substance use at initiation is relatively uncontaminated by snowball effects resulting from mutual influences between the two behaviors over time (Wanner et al. 2009). Finally, a better understanding of the onset of these problem behaviors will inform prevention programs, which should help avoid adjustment problems downstream (DuRant et al. 1999; Rahman et al. 2012).

For example, if genetic factors are important, then heritable phenotypes known to predict gambling and substance use, such as impulsivity, could be targeted. If shared environmental factors turn out to be important, then family factors can be targeted. Unique social experiences (i.e., peers) should be considered if nonshared environmental influences are important. Finally, if common genetic or environmental factors are involved with both types of problem behaviors, then prevention programs could adopt a generic perspective by targeting common risk factors shared by both types of problem behaviors, as has been proposed elsewhere (Derevensky et al. 2004; Potenza 2003).

To address these issues, the first objective of the present study was to model the genetic and (shared and nonshared) environmental influences on gambling involvement and on substance use during early adolescence. The second objective of the present study was to examine the extent to which gambling involvement and substance use during early adolescence can be explained by the same underlying genetic, shared, and nonshared environmental factors.

Method

Participants

The sample is part of an ongoing longitudinal study of a population-based sample of

twins drawn from the Quebec Newborn Twin Registry of all twin births occurring in the Province of Quebec, Canada, between 1996 and 1998. A total of 650 families agreed to participate in the study (113 MZ female pairs, 110 MZ male pairs, 115 DZ female pairs, 107 DZ male pairs, 205 mixed-sex DZ twin pairs). Zygosity was assessed by genetic marker analysis of 8-10 highly polymorphous genetic markers and twins were diagnosed as MZ when concordant for every genetic marker. When genetic material was insufficient or unavailable due to parental refusal (43% of cases), zygosity was determined based on physical resemblance questionnaires at 18 months and again at age 9 (Goldsmith 1991). The comparison of zygosity based on genotyping with zygosity based on physical resemblance in a subsample of 237 same-sex pairs revealed a 94% correspondence rate, which is similar to rates obtained in other studies (Magnusson et al. 2013; Spitz et al. 1996). Eighty-four percent (n = 546) of the families were of European descent, 3% (n = 19) were of African descent, 2% (n = 13) were of Asian descent, 2% (n = 13) were Native North Americans, and 9% (n = 59) did not specify ethnicity. At 5 months of age, the demographic characteristics of the twin families resembled that of a representative sample of singleton families from large urban centers in the province of Quebec (Santé Québec et al. 1998). At the outset, 95% of parents lived together; 44% of the twins were the first born children; 66% of mothers and 60% of fathers were between 25 and 34 years old; 17% of mothers and 14% of fathers had not finished high school; 28% of mothers and 27% of fathers held a university degree; 83% of the parents were employed; 10% of the families received social welfare or unemployment insurance.

Data were collected every year between the ages of 1 and 13. The present study involves the first assessment of gambling and substance use, which took place at age 13 (between 2009

and 2011). Data collection at this wave was approved by the Sainte-Justine Hospital Research Centre ethics committee. Parent consent and child assent for participation was obtained.

Of the 650 pairs in the initial sample, 401 (61.7%) participated in data collection at age 13. Same- and mixed-sex DZ twins displayed differences in the multivariate covariance matrix ($\chi^2(6) = 87.94$, $p < .001$), so the 122 mixed-sex DZ twin pairs were excluded from the analyses. The final sample included 279 twin pairs (n = 160 MZ and n = 119 same-sex DZ). The final sample did not differ significantly from the 207 MZ and same-sex DZ twin pairs that did not participate in the data collection at age 13 in initial family income ($t(299) = 0.17$, $p = .86$), family structure ($\chi^2(2) = 0.34$, $p = .84$), or birth weight ($t(840) = 0.20$, $p = .84$). Those that did and did not participate in the age 13 data collection also did not differ significantly on a variety of problem behaviors, including disruptiveness (K: $t(551) = 0.71$, $p = .48$; G1: $t(604) = -0.18$, $p = .86$), proactive aggression (K: $t(551) = 1.15$, $p = .25$; G1: $t(603) = 0.29$, $p = .77$), physical aggression (K: $t(551) = -0.13$, $p = .89$; G1: $t(604) = -0.66$, $p = 51.$), indirect aggression (K: $t(550) = 1.13$, $p = .26$; G1: $t(604) = -0.34$, $p = .73$), and reactive aggression (K: $t(551) = 0.38$, $p = .70$; 1: $t(604) = -0.60$, $p = .55$) at age 6 or age 7 years, nor delinquency at age 10 years ($t(610) = -1.22$, $p = .22$). Of the 279 twin pairs participating in the age 13 data collection, missing data accounted for an average of 1.6% of reports (range: 1.3%-1.8%). Missing data were handled with full information maximum-likelihood estimation (FIML), which allowed participants with incomplete data to be included in the models. Little's test indicated that data were missing completely at random, $\chi^2(2) = 0.37$, $p = .83$.

Measures

Instruments were administered either in English (21%) or in French (79%), depending on the language spoken by the children and their parents. Back-translation procedures were

employed and bilingual translators verified the semantic similarity between the back-translated items and the original items in the questionnaire.

Gambling Involvement. Frequency of involvement in gambling was assessed with the South Oaks Gambling Screen for adolescents (SOGS-RA) (Winters et al. 1993). Involvement was computed by averaging the frequency scores of 12 gambling activities over the past 12-months (e.g., purchased lottery tickets (6/49 or Banco), played Mise-O-Jeu (online sports betting game), bought scratch offs, played Bingo for money, bet on games on the internet, played video lottery terminal games, played cards or games with others for money, bet on sporting events/games for money, gambled at a casino, bet on games of skill (pool, basketball), played dice games for money, and bet on other games). Participants rated the frequency of each activity on a scale ranging from 1 (*never*) to 7 (*daily*) (Cronbach's $\alpha = .65$). MZ and DZ twins had identical average scores ($M = 1.03$; MZ $SD = 0.09$; DZ $SD = 0.10$). Approximately 20% of participants engaged in at least one gambling activity at least once. This proportion is in line with other prevalence studies (Pica et al. 2012; Volberg et al. 2011). Approximately 1.1% of participants engaged in at least one gambling activity regularly (i.e., more than once a month). Given the positively skewed distribution of gambling involvement, the negative reciprocal of the measure was calculated and then z-standardized within sex, normalizing the measure (skew before and after transformation = 7.20 and 3.16).

Substance Use. Frequency of substance use was assessed with the Personal Experience Screening Questionnaire (PESQ) (Henly and Winters 1989; Winters et al. 1990-91). Substance use was computed by averaging the standardized scores for four items (frequency of alcohol, marijuana, and other drug use and binge drinking). Participants rated the frequency of substance use over the past 12 months on a scale ranging from 1 (*never*) to 7 (*daily*), with the exception of

binge drinking, which was rated on a scale from 1 (*never*) to 6 (*5 times or more*) (Cronbach's α = .74). MZ and DZ twins had near identical average scores (MZ *M* = 1.14, *SD* = 0.25; DZ *M* = 1.15, *SD* = 0.36). Approximately 21% of participants reported experience with at least one substance at least once. This proportion is also in line with past prevalence studies (Johnston et al. 2013; Pica et al. 2012). Approximately 2.1% of participants used at least one substance regularly (i.e., more than once a month). Given the positively skewed distribution of substance use, the negative reciprocal of the measure was calculated and then z-standardized within sex, normalizing the measure (skew before and after transformation = 10.20 and 2.10). To take into account the non-normality of the data in reference to both substance use and gambling involvement, we used maximum likelihood estimation with robust standard errors (MLR).

Plan of Analysis

Univariate Genetic Models. The data were analyzed using Mplus v7.0 (Muthén and Muthén 1998-2013). Univariate models were fit to the data to estimate the relative contribution of genetic and environmental factors to substance use and gambling involvement. By comparing within-pair correlations for MZ twins (who are genetically identical) and DZ twins (who on average share only half of their genes), sources of variability in a measured variable (phenotype) can be estimated as latent additive genetic (A), latent shared environmental (C), and latent nonshared environmental (E) factors (Neale and Cardon 1992).

Within-twin pair correlations of the latent genetic factors (A) were fixed to 1.0 for MZ twins and to 0.5 for DZ twins. Within-twin pair correlations of the latent shared environmental factors (C) were fixed to 1.0 for both MZ and DZ twins. Within-twin pair correlations of the latent nonshared environmental factors (E) were fixed to 0 for both MZ and DZ twins. The estimated coefficients a, c, and e were fixed to be equal across the two members of a twin pair

and across MZ and DZ twins. These coefficients are factor loadings that provide information about the relative contribution of the latent factors A, C, and E to the total variance V_T of each phenotype ($V_T = a^2 + c^2 + e^2$). Measurement error is included in e^2.

A model estimating a possible dominant genetic effect (Neale and Cardon 1992) would also be modeled if the within-pair correlation for MZ twins is more than twice as strong as the within-pair correlation for DZ-twins. Latent factors are removed from the models if model fit improves or if parsimony improves without sacrificing model fit. Univariate models were chosen on the basis of parsimony, low and nonsignificant χ^2 values, and values of *RMSEA* below .08.

Bivariate Genetic Models. The bivariate model combines the two final best-fitting univariate models. Bivariate genetic models are used to examine whether the phenotypic association between substance use and gambling involvement can be explained by common underlying factors. A bivariate Cholesky model was specified such that the covariance structure of substance use and gambling involvement was partitioned into (1) *common* latent genetic, shared and nonshared environmental factors that influence both substance use and gambling, and (2) *unique* latent genetic, shared and nonshared environmental factors that are specific to gambling. A significant contribution of a common latent factor (genetic, shared or nonshared environmental) to both phenotypes indicates an overlap or correlation between substance use and gambling involvement. Overlap was only estimated for latent factors that were statistically significant in both univariate models. Latent factor effects that were estimated to be zero were removed to maximize model parsimony and statistical power. The final bivariate results represent the best-fitting, most parsimonious model.

A multiple group framework was used to examine sex differences in the final bivariate model. Effects for boys and girls were constrained to be equal. There were no statistically

significant changes in model fit, indicating that the pattern of results did not differ as a function of sex.

Results

Preliminary Analyses

Two sets of nested χ^2-difference tests examined mean level differences in substance use and gambling involvement. Sex differences emerged for gambling involvement, $\chi^2(1) = 12.74$, $p < .001$. Boys reported more gambling involvement than girls. There were no sex differences in substance use, $\chi^2(1) = 0.48$, $p = .49$.To control for the mean level differences, scores were z-standardized by sex, separately for MZ and DZ pairs (see Arseneault et al. 2003; Van den Oord et al. 2000). There were no differences between MZ and DZ twins on substance use, $\chi^2(1) = 0.002$, $p = .96$, or gambling involvement, $\chi^2(1) = 0.01$, $p = .92$.

Statistically significant within-pair correlations emerged for MZ twin pairs and DZ twin pairs on substance use (MZ $r = .54$, DZ $r = .39$) and gambling involvement (MZ $r = .55$, DZ $r = .18$). Correlation contrasts revealed that the within-pair correlation on gambling involvement was stronger for MZ twin pairs than for DZ twin pairs ($p < .001$). There were no statistically significant differences between MZ twin pairs and DZ twin pairs in the within-pair correlation on substance use. There were no sex differences on the within-pair correlations for substance use (male $r = .41$, female $r = .52$) or gambling involvement (male $r = .33$, female $r = .46$). The phenotypic correlation between substance use and gambling involvement was $r = .21$, $p < .001$ (CI = 0.12; 0.29).

Univariate Genetic Models

Table 1 summarizes the results of the univariate models for substance use and for gambling involvement. For substance use, genetic factors explained about 33% of the variance,

shared environmental sources explained about 22% of the variance, and nonshared environmental factors accounted for 45% of the variance. For gambling involvement, genetic factors and nonshared environmental factors each accounted for about half of the variance; the effect of shared environmental factors was estimated to be 0. When the shared environmental effects were fixed to zero in an AE model of gambling involvement, there was no statistically significant change in model fit, $\Delta\chi^2(1) = .00$, $p = 1.00$. The within-pair correlation for MZ twins was more than twice as strong as the within-pair correlation for DZ-twins, so we also estimated an ADE model with a dominant genetic effect for gambling involvement (Neale and Cardon 1992). The AE model provided a better fit to the data than the ADE or the ACE model.

Bivariate Genetic Models

The bivariate Cholesky model based on the final univariate models included two components: (1) common latent factors A_C, C_C, and E_C that influence both phenotypes, and (2) unique latent factors A_{UG}, C_{UG}, and E_{UG} specific to gambling involvement (see Figure 1). The shared environmental effect on gambling involvement was not statistically significant in univariate analyses, so the C component (c_{UG}) and C overlap (c_{CG}) parameters were fixed to zero in the bivariate model. Because the nonshared environmental overlap was estimated to be zero in the full bivariate model, the parameter estimate of the factor loading of gambling on E_C was fixed to zero in the final bivariate model (see Table 2). There was no statistically significant difference in model fit between the full model estimating the E overlap (i.e., e_{CG}) and the model constraining this coefficient to zero, $\Delta\chi^2(1) = 0.26$, $p = .61$. The final model (Figure 1) refers to the most parsimonious model with the nonsignificant coefficients constrained to zero. The model fit the data well, $\chi^2(8) = 3.86$, $p = .87$; *RMSEA* = .0.05, *CFI* = .96.

The results revealed that the relative contribution of the ‘common’ latent genetic factor

A_C to the genetic variance of gambling involvement ($a_{CG}^2/(a_{CG}^2 + a_{UG}^2)$) is 21.9% ($.33^2/(.33^2 + .63^2)$). Thus, 22% of the genetic variance of gambling involvement was explained by genetic factors that also influenced substance use. The phenotypic association between substance use and gambling involvement was entirely explained by common underlying genetic influences; all other covariance paths were zero.

Supplemental Analyses

Analyses were rerun with count variables for gambling involvement and substance use. Count variables were calculated as the number of gambling activities the participants engaged in or the number of substances the participants used over the past 12 months. The patterns of significance were the same in the final model with one exception: the shared environment (C) component of substance use was weaker and the A component was stronger.

Discussion

This study represents the first twin study of gambling involvement during early adolescence. Its aims were a) to examine the relative contribution of genetic and environmental factors to gambling involvement and substance use during an age period (i.e., early adolescence) when most youth first engage in these behaviors, and b) to investigate whether the concomitant early emergence of gambling involvement and substance use can be explained by common underlying genetic factors and common underlying environmental factors.

As expected, age 13 gambling involvement and substance use were correlated, albeit modestly, at the phenotypic level. This correlation did not differ for males and females. Similar correlations between gambling involvement and substance use have been found in studies with adolescent samples (Barnes et al. 2005; Richmond-Rakerd et al. 2013; Vitaro et al. 2001).

The results from our univariate ACE models for substance use are consistent with past

findings indicating that genetic, shared, and nonshared environmental factors play a significant role in early adolescent substance use (Hopfer et al. 2003; Kendler et al. 2008). The results for early adolescent gambling involvement, however, resemble those for gambling involvement and problem gambling in adults (Beaver et al. 2010; Eisen et al. 1998; Slutske et al. 2009; Slutske et al. 2010; Winters and Rich 1998), in that half or more of the variance was explained by genetic factors and the rest by nonshared environmental factors (see, however, Slutske and Richmond-Rakerd 2014, for an exception). The present results raise two questions. First, why did we find genetic influences for gambling involvement in early adolescents, whereas the only other study that examined gambling involvement in adolescents (i.e., Loehlin and Nichols 1976) did not? One line of explanation lies in social norms. Gambling data for the Loehlin and Nichols (1976) study were collected in an era (the early 1960s) when gambling was generally prohibited. In contrast, the current data were collected in more permissive times. As a consequence, adolescents in earlier studies, although older than the adolescents in the present study, may have been less likely to express their genetic dispositions towards gambling than adolescents in the current study. In other words, social norms may have suppressed the expression of genetic influences through a gene x environment interaction. The existence of moderating effects of social norms on the expression of genetic risk for other phenotypes, such as aggression, gives credence to this position (Brendgen et al. 2013).

A similar line of explanation can help address a second question: Why do shared environmental factors play a role with respect to substance use during early adolescence and not with respect to gambling involvement? Results from a recent survey of parents with adolescents between the ages of 13 and 18 implicate parent attitudes. In this survey, parents indicated that they view alcohol and drug use as a much greater problem than gambling (Campbell et al. 2011).

A general absence of parental norms or family rules that discourage gambling in most families may thus explain why environmental factors shared by twins raised together did not significantly account for interindividual differences in early gambling involvement in the present study. In contrast, the likely greater variability of family rules regarding substance use – with very lenient rules in some families and very strict rules in other families – may explain why shared environmental factors contributed to substance use during early adolescence in the present study.

Our bivariate Cholesky models identified significant overlap in the genetic factors associated with both substance use and gambling involvement, which entirely explained the phenotypic correlation between these two variables. Such findings are consistent with molecular genetic studies indicating that gambling and substance use share biological mechanisms that encompass impulse control networks. In particular, serotonin, involved with behavioral inhibition, and dopamine, involved with learning, motivation, and reward sensitivity, may contribute to both types of disorders (Potenza 2008). The degree to which overlapping genetic factors influence substance use and gambling involvement is typically greater among adults (Slutske et al. 2000). Studies with adults also reveal overlapping nonshared environmental factors, which we did not find in our sample of early adolescents. The correlation between gambling and substance use is typically greater in adults than in adolescents (Pietrzak et al. 2007) and this increased correlation may be due to an escalating interplay (i.e. mutual influence) between substance use and gambling in adulthood (Vanyukov et al. 2012). It is thus possible that the greater overlap of genetic and environmental influences observed in adults than in our early adolescent sample arises from increased reciprocal influences between gambling and substance use that amplify the covariance between the two behaviors over the course of development. Further longitudinal studies are necessary to clarify this issue.

The present study is the first to examine the genetic-environmental architecture of gambling involvement and substance use during the critical early adolescent years, which typically encompass the onset of involvement with substances use and gambling. Nevertheless, several limitations merit mention. First, our interest on early adolescence gave rise to a focus on the frequency of gambling and substance use and not on substance- or gambling-related problems, which are rare at age 13. Although early involvement in substance use and gambling is non-normative (only one out of five adolescents is involved) and although both behaviors are established precursors of later problems, caution should be exercised in generalizing our findings to the etiology of addictions. Second, we note our exclusive reliance on self-reports to assess gambling involvement and substance use. Finally, the relatively small sample size and resulting limited statistical power may explain the absence of sex differences.

We close with three tentative conclusions. First, gambling and substance use share a common genetic basis in early adolescence, although most of the genetic and all of the environmental variance is unshared during this age period. In that respect, the common etiology of gambling involvement and substance use is, at best, partial. It is nevertheless important to examine what specific endophenotype(s), which may themselves be heritable, mediate this common, although modest genetic overlap. The identification of such phenotypes could be useful both for early screening of at-risk individuals and as a potential target for preventive interventions. As indicated in the introduction, response inhibition/impulsivity could play a role (Bezdjian et al. 2011), but other endophenotypes such as reward dependence/delayed discounting (i.e., how much a reward loses values based on its distance in time) (MacKillop 2013) or a more general externalizing problem syndrome (Krueger et al. 2007) could be involved. Second, prevention programs that - in accordance with a generic perspective of prevention (Derevensky

et al. 2004; Potenza 2003) - target common risk factors shared by risk taking behaviors such as gambling and substance use may be effective in early adolescence, but only partially if they are not complemented by modules that are specific to each type of behavior. Finally, future research should include contextual measures such as peer group or family norms, which could play an important role for conditioning the expression of genetic liability. The contribution of these variables may vary across development, because the genetic-environmental architecture of both gambling involvement and substance use could change with age as adolescents become more autonomous and as problem behaviors become more normative or more specialized (Vrieze et al. 2012).

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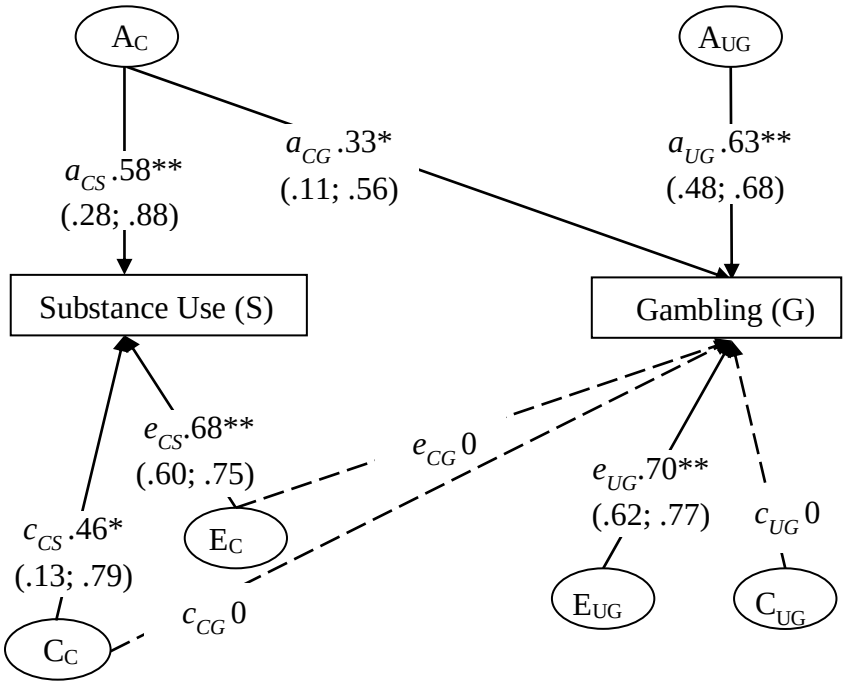
Table 1

Univariate Model Results

	%a^2	%c^2	%e^2	%d^2	AIC	BIC	CFI	RMSEA	χ^2 (df)	*p*
				Substance Use						
ACE	**32.8 (-2.2;67.8)**	**22.0 (-8.7;52.7)**	**45.2 (34.5;55.9)**		**1491.9**	**1506.4**	**0.95**	**0.07**	**0.01 (2)**	**0.99**
AE	56.4 (46.7;66.1)	-	43.6 (33.9;53.3)		1491.6	1502.5	0.94	0.07	1.77 (3)	0.62
CE	-	47.5 (38.1;56.8)	52.5 (43.2;61.9)		1493.4	1504.3	0.91	0.08	3.56 (3)	0.31
E	-	-	100.0 (--;--)		1558.2	1565.5	0.00	0.25	70.40 (4)	0.00
				Gambling Involvement						
ACE	51.9 (41.3;62.4)	0 (.0;.0)	48.1 (37.6;58.7)		1513.2	1527.8	1.00	0.00	1.06 (2)	0.59
AE	**51.9 (41.3;62.4)**	-	**48.1 (37.6;58.7)**		**1511.2**	**1522.1**	**1.00**	**0.00**	**1.06 (3)**	**0.79**
CE	-	39.6 (29.6;49.6)	60.4 (50.4;70.4)		1522.5	1533.4	0.88	0.08	12.35 (3)	0.01
E	-	-	100 (--;--)		1566.7	1574.0	0.07	0.22	58.56 (4)	0.00
ADE	20.9 (-53.0;94.7)		47.0 (36.6;57.4)	32.1 (-43.4;107.7)	1512.5	1527.0	1.00	0.00	0.33 (2)	0.85

Note. Best fitting models are in bold.

Figure 1. Bivariate Cholesky model of substance use and gambling involvement for one member of each twin pair.



Note. The model partitions variance into (1) common latent factors A_C, C_C, and E_C that influence both phenotypes, and (2) unique latent factors A_{UG}, C_{UG}, and E_{UG} specific to gambling involvement. Coefficients a_{CS}, c_{CS}, and e_{CS} represent the factor loadings of substance use (S) on the common latent factors A_C, C_C, and E_C, respectively. Coefficients a_{CG}, c_{CG}, and e_{CG} represent the factor loadings of gambling involvement (G) on the common latent factors A_C, C_C, and E_C, respectively. Coefficients a_{UG}, c_{UG}, and e_{UG} represent the factor loadings of gambling involvement on the unique latent factors A_{UG}, C_{UG}, and E_{UG}. Univariate analyses found no significant shared environmental effect on gambling involvement, so the C component of gambling (c_{UG}) and C overlap (c_{CG}) parameters were fixed to zero. The parameter estimate e_{CG} of the factor loading of gambling on E_C was estimated to be zero in a preliminary model and therefore fixed to be zero in the final model. Confidence intervals (95%) are shown in parentheses. $N = 279$ twin pairs. *$p < .05$, **$p < .001$, two-tailed.