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Genetic and Environmental Origins of Gambling Behaviors From Ages 18 to 25: A Longitudinal Twin Family Study

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Gambling behaviors tend to increase in prevalence from late adolescence to young adulthood, and the underlying genetic and environmental influences during this period remain largely understudied. We examined the genetic and environmental influences on gambling behaviors contributing to stability and change from ages 18 to 25 in a longitudinal, behavioral genetic mixed-sex twin study design. Participants were enrolled in the Minnesota Twin Family Study. A range of gambling behaviors (maximum frequency, average frequency, money lost, and gambling problems) were assessed at ages 18 and 25. The results of our study support the following conclusions: (a) the genetic and environmental factors impacting a range of gambling behaviors are largely similar in men and women, (b) genetic factors increase in influence from 18 to 25 (21% at age 18 to 57% at age 25), (c) shared environmental factors are influential at age 18, but tend to decrease from ages 18 to 25 (55% at age 18 to 10% at age 25), and (d) nonshared environmental influences are similarly significant and are small to moderate in magnitude at both ages. The findings add to a small yet important research area regarding determinants of youth gambling behaviors and have the potential to inform prevention and intervention efforts.

Keywords: gambling, genetics, longitudinal, twins, young adulthood

Adolescence to young adulthood represents a high risk period in the development of regular and problematic patterns of gambling and other addictive behaviors (Bray et al., 2014; Carbonneau, Vitaro, Brendgen, & Tremblay, 2015). Gambling behaviors tend to increase from late adolescence to young adulthood (Delfabbro, King, & Griffiths, 2014; Slutske, Jackson, & Sher, 2003; Winters, Stinchfield, Botzet, & Anderson, 2002; Winters, Stinchfield, Botzet, & Slutske, 2005) and adolescent onset gambling is related to risk for later problem gambling (Kessler et al., 2008). There is significant instability and change in gambling behaviors across this period (Delfabbro, Winefield, & Anderson, 2009). During the transition to young adulthood, a wider range of environmental opportunities may lead to increased risk for engaging in risky behaviors, including gambling. Gambling problems may be conceptualized in the context of a developmental life course model (Blaszczynski & Nower, 2002), and the period of adolescence to young adulthood may serve as a critical role transition in the development of problem gambling (Bray et al., 2014; Edgerton, Melnyk, & Roberts, 2015). Despite developmental change in the frequency of gambling over this period, very few studies have directly examined the genetic and environmental influences during the transition.

Age of gambling onset predicts the escalation and involvement in a variety of gambling activities (Carbonneau et al., 2015; Slutske, Piasecki, Deutsch, Statham, & Martin, 2015). Although the prevalence of a diagnosable gambling disorder is relatively rare from adolescence to young adulthood (Fröberg et al., 2015), timing in the onset of gambling initiation may be predictive of future problematic behaviors. Several twin studies spanning preadolescence to young adulthood have examined the extent to which genetic and environmental factors influence the onset and persistence of gambling behaviors. Findings from a recent twin study suggested that nonshared environmental factors were responsible for age at first drink and age at initiation of gambling (Richmond-Rakerd et al., 2014). Early gambling and substance use may originate from different environmental and genetic factors (shared, nonshared and additive genetic factors; Vitaro et al., 2014). Specifically, a recent twin study of early adolescence suggested that nonshared environment and genetics accounted equally for early involvement in gambling, and in contrast, shared, nonshared, and

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genetic factors accounted for early substance use (Vitaro et al., 2014). The genes and environments impacting age at first gambling and age at first drink seem to be largely distinct (Richmond-Rakerd et al., 2014).

Twin studies are uniquely equipped to parse genetic and environmental influences on behaviors. The classical twin methodology partitions the total phenotypic variance into that which is due to additive genetic (A), shared environmental (C; environmental factors that are common to twins and act to make them similar), and nonshared environmental (E; environmental factors that are not common to twins and that act to make them dissimilar) influences (Plomin, Defries, McClearn, & Rutter, 1997). Because members of MZ twin pairs share 100% of their genetic endowment where DZ twins share, on average, only 50%, A is inferred when MZ twin pairs are more similar than DZ pairs. Further, because this approach assumes that the rearing environments of MZ twins are no more similar than that of DZ twins, C is inferred when the MZ twin correlation is less than twice the DZ twin correlation. Finally, E is inferred when MZ twin similarity is less than perfect.

Twin studies on gambling have primarily utilized adult samples and most studies have examined problem gambling (as compared to frequency measures or typical gambling behaviors). Adult twin studies have demonstrated a range of significant genetic effects on the etiology of pathological gambling (genetic factors accounting for 32% to 57.5% of variance; Blanco et al., 2012; Slutske & colleagues, 2010; Xian et al., 2007) and the results may be influenced by the age range of samples. One review of adult twin studies suggested a moderate genetic influence and little evidence for environmental influences (Lobo & Kennedy, 2009). Twin studies on gambling behaviors have tended to demonstrate a more substantial contribution of nonshared environment, with relatively fewer studies suggesting shared environmental effects (Beaver et al., 2010; see Lobo & Kennedy, 2009 for review; Eisen et al., 1998; Slutske et al., 2010; Slutske, Zhu, Meier, & Martin, 2011). Effects have varied on the basis of measurement approach, sample characteristics, and age ranges studied. Adult twin studies have demonstrated few sex differences with respect to the relative impact of genetics and environment on problem gambling behaviors (Shah, Eisen, Xian, & Potenza, 2005; Slutske, Zhu, Meier, & Martin, 2010). When the overlapping effects of other psychological factors (e.g., personality) are taken into account, sex differences in the contributions of genes and environment may emerge (Slutske, Cho, Piasecki, & Martin, 2013). In sum, most adult twin studies of gambling find few, if any, sex differences in the genetic and environmental influences on gambling.

There have been few youth twin studies on gambling and some studies have relied on retrospective report of youth gambling in adulthood. In one of the few published twin studies on youth gambling, 75% of the variance in gambling problems in adolescents was attributable to genetic factors, and the remaining variance was explained by nonshared environmental factors (Beaver et al., 2010). Findings from a young adult twin study demonstrated that nonproblem gambling had a smaller heritability estimate than disordered gambling (Blanco, Myers, & Kendler, 2012). One investigation found that gambling behaviors were significantly more heritable in young men than young women (Beaver et al., 2010), whereas another found no sex differences in genetic and environmental influences in a young adult twin sample (Blanco, Myers, & Kendler, 2012). A follow-up study of the same sample suggested that there was no evidence for sex differences in the genetic and environmental influences on gambling etiology and family environmental factors explained individual differences in gambling involvement (Slutske & Richmond-Rakerd, 2014). An adolescent twin study by Winters and Rich (1998) failed to find any effects of genetic factors on girls' gambling behaviors compared to a significant influence of genetics on boys' gambling, suggesting a sex difference in etiology. A recent study of adults suggested that different etiological factors may influence age at first gambling and drink in men and women (Richmond-Rakerd et al., 2014).

The current study is significant and adds to the literature in several ways. Previous studies have relied primarily on large scale, retrospective studies of adults, and very few have utilized a prospective longitudinal twin study design. Our study design allows for a novel examination of correlated and uncorrelated genetic and environmental effects on gambling behaviors in a large scale mixed-sex sample collected at two waves (age 18 and age 25).

We estimate the degree of additive genetic, nonshared, and shared environmental influences on gambling behaviors from ages 18 to 25 and determine whether the genetic and environmental architecture of gambling behaviors are similar or different among men and women. We hypothesize that genetic factors will become increasingly important from 18 to 25 and that the genetic and environmental architecture of gambling behaviors will be similar in men and women. Additionally, we predict that genetic and nonshared environmental factors will predict stability and change in gambling behaviors during this period.

Method

Participants

The Minnesota Twin Family Study (MTFS) is a longitudinal, population-based sample of twins and their families examining the development of substance use disorders and related psychopathology. Prior to data collection, the study was approved by the university Institutional Review Board. Twin pairs were ascertained from Minnesota state birth records from 1978 to 1982. Of those eligible to participate (i.e., lived within a day's drive and twins had no physical [e.g., blindness] or intellectual disability that would preclude completing the day-long, in-person assessment), 84% accepted our invitation to participate. Participating families differed significantly from nonparticipating families only in percent of MZ twins (67% vs. 57%) and parental education (on average by 0.6 years; Iacono et al., 1999). Participants included 756 twin pairs (male: MZ = 252, DZ = 124; female: MZ = 233, DZ = 147), along with their parents, who were first assessed at age 11. Approximately 98% of the sample was Caucasian. Subsequent to their initial visit, twins participated in follow-up assessments scheduled approximately every three years through age 29. Both intake and follow-up assessments were conducted at the University of Minnesota. Rates of follow-up participation range from 87% to 92%, with minimal diagnostic differences between participants and nonparticipants.

Zygosity was determined by comparing three estimates: (a) parental reports of physical resemblance on a zygosity questionnaire, (b) staff evaluation of physical similarity, including comparison of eyes and ears, and (c) an algorithmic value combining

ponderal index, cephalic index, and fingerprint ridge count. When these estimates were inconsistent, DNA-based confirmation of zygosity was obtained through serological analysis. We have previously shown that this method of zygosity determination is highly valid (Iacono et al., 1999).

Measures: Gambling Behaviors

To utilize a developmentally relevant measure of gambling, our analysis used three gambling variables. These variables were based on the Gambling Survey, a self-report instrument adapted from the South Oaks Gambling Screen-Revised Adolescent (Winters, Stinchfield, & Fulkerson, 1993). The SOGS-RA, which was adapted from the adult tool, the South Oaks Gambling Screen (Lesieur & Blume, 1987), is a relatively brief and yet psychometrically sound measure to assess gambling involvement and problems in adolescents (Poulin, 2002; Winters et al., 1993). We chose this tool because its gambling problem items closely align with the DSM-based criteria for a gambling disorder at the time of the study, and it included items on gambling frequency across multiple activities. We also added items pertaining to amount spent on gambling. The specifics of the three variables are provided below.

Gambling frequency. For the previous 12 months, respondents were asked to indicate how frequently they engaged in 10 different gambling activities (e.g., "playing cards for money", "betting on sports teams"); response options were *never* (1), *less than monthly* (2), *monthly* (3), and *weekly or more* (4).

Amount spent. respondents were asked to indicate the largest amount of money ever lost in a single day; response options ranged from *less than a dollar* (1) to *1000 dollars or more* (5). Respondents who had never gambled were coded '0.'

Gambling problems. we coded problems as either *present* (1) or *not present* (0) to 12 symptoms indicative of problem gambling. Some items were simple Yes/No (e.g., "Was there ever a time you felt you would have liked to stop betting money but did not think you could?"). For those items that asked how often a behavior occurred, we coded "sometimes" as indicative of "present." We based the problem gambling behavior variable on the count of present problem gambling symptoms in order to create an informative continuous variable.

From the questions, four scales were created to assess gambling behaviors at each age:

1. The maximum frequency with which the respondent engaged in any of the 10 gambling behaviors (max frequency);
2. The mean of the response frequencies for all 10–12 gambling activities (typical frequency; $\alpha = .77$ at age 18 and .78 at age 25);
3. The maximum amount of money lost in a single day (max amount); and
4. The sum of the 12 gambling problem behaviors (problem behavior; $\alpha = .72$ at age 18 and .63 at age 25).

Statistical Analyses

A repeated-measures analysis of variance (R-M ANOVA) was conducted to evaluate the effects of sex and age on the gambling variables, using SAS PROC MIXED to account for the correlated nature of the observations. Participants were included in the sample if they participated in two assessment points: at the 18 and age 25 assessments. Twin intraclass correlations were estimated and biometric models were fit to twin data using Mx full-information maximum-likelihood raw data techniques to account for missing data (Neale, Boker, Xie, & Maes, 1999).

Initially, we fit a four-variable Cholesky model which simultaneously estimated the genetic and environmental contributions to each of the gambling variables, separately for males and females at ages 18 and 25. Next, we fit a latent factor model to obtain a summary measure of gambling behavior. The factor was defined by the variance shared by the four gambling variables at each time point for males and females. We then fit a series of nested models to determine whether the A, C, and E estimates for the latent factor at the two time points could be constrained to be equal across sex and assessment age. The base for the computation of the model fit statistics was the minimized value of minus twice the log-likelihood values ($-2\ln L$) from a fully saturated model, in which means, variances, and covariances were freely estimated. This saturated model provided a baseline against which to assess the relative fit of a minimally constrained factor model which allowed estimated parameters to vary across sex and age. The fit of subsequent models was gauged relative to the minimally constrained factor model. Comparative model fit was assessed by taking the difference in $-2\ln L$, which is distributed as a χ^2 random variable under the null hypothesis of the more restrictive model. Akaike's Information Criterion (AIC $= \chi^2 - 2df$; Akaike, 1987), which considers both parsimony and goodness of fit, was also used to compare the fit of alternative models. The model with the smallest AIC is generally preferred.

Results

The Gambling Survey was administered to the twins at the second (FU2; 1990–1995) and fourth (FU4; 1996–2003) follow-up assessments. A total of 1320 twins participated in FU2, 90% ($N = 1191$; mean age = 18.1 years, range = 16.6–20.3; 46% male), of whom completed the Gambling Survey. Similarly, there were 1322 FU4 participants, and 95% ($N = 1250$; mean age = 25.2 years, range = 23.7–28.0; 48% male) completed the gambling questionnaire.

Descriptive Statistics and Twin Correlations

In total, 68% of the participants indicated that they had gambled at age 18, and the prevalence of gambling behavior increased to 76% at 25 years. However, examination of means for the four gambling variables at both ages provided little evidence of pathological gambling in the samples as a whole. The mean maximum frequency of gambling behavior is equal to 2.06 and 2.09 at age 18 and age 25, respectively; the mean typical frequency is 1.28 at both ages (2 = less than monthly). Further, the mean maximum amount lost gambling in a singled day is $1.73 at 18 years and $2.20 at 25 years (2 = $1 to $10), and the mean of the number of problem behaviors endorsed (possible range = 0 to 12) is .39 at 18 years

and .36 at 25 years. Table 1 provides means and standard deviations (*SD*) for each of the four gambling variables separately for males and females at each assessment age.

For all remaining analyses, Typical Frequency and Problem Behaviors were log-transformed to reduce positive skew. Log-transformation reduced the standardized skewness coefficient from 2.1 to 1.1 for Typical Frequency; it was less successful for Problem Behaviors, reducing skew from 3.6 to 3.0. The R-M ANOVA revealed that, for all measures, mean scores for male participants were significantly greater when compared with scores for female participants ($p < .001$). In addition, mean levels of gambling behavior were relatively stable between ages 18 and 25, with only Max Amount showing a significant increase with increasing age ($p < .001$). Nevertheless, interclass correlations (not shown in Table 1) between the gambling variables at the two ages were low to moderate (range = .31 to .45 for males and .13 to .35 for females), providing some evidence for intraindividual change over time. Table 1 also displays the MZ and DZ twin intraclass correlation coefficients, with 95% confidence intervals, for the gambling variables. In general, MZ twin correlations were moderate (range = .29 to .64 for males and .25 to .62 for females), DZ twins correlations were somewhat lower (range = .24 to .55 for males and .27 to .55 for females), and values were similar across males and females. These correlations are consistent with a moderate degree of genetic influence at ages 18 and 25.

Cholesky Estimates

Genetic (A), shared environmental (C), and nonshared environmental (E) parameter estimates and confidence intervals for each of the four gambling variables are shown in Table 2, separately for males and females at both ages. These results mirror those presented for the twin intraclass correlations. Specifically, estimates are similar for males and females, and there is evidence for genetic contributions at both ages. Also, evident in the Cholesky estimates is the waning influence of shared environment on each of the gambling variables from ages 18 (estimates range from .19 to .45) to 25 (estimates range from .03 to .17).

Factor Model

The four gambling variables were fit to a latent factor to obtain a summary measure of gambling behavior for males and females at ages 18 and 25 (see Figure 1). The initial model allowed parameters to vary for males and females and at each age. Relative to the saturated model ($-2\text{lnL} = 18244.637$, $df = 9046$), the latent factor model fit well ($\Delta -2\text{LnL} = 800.898$, $\Delta df = 474$, AIC = −147.102). Standardized factor loadings are shown in Table 3, separately by assessment age and sex. Factor loadings, which were remarkably consistent across age and sex, ranged from .48 to .94.

We then fit a series of nested factor models to determine if the standardized parameter estimates for A and C for the summary factor could be constrained across males and females. Relative to the sex- and age-variant model, these standardized estimates could be set equal across sex without a significant decrement in fit ($\Delta -2\text{LnL} = 1.688$, $\Delta df = 4$, AIC = −6.312). Next, we attempted to constrain standardized A and C across age as well as sex. Again relative to the sex- and age-variant model, this model resulted in a significant decrement in model fit ($\Delta -2\text{LnL} = 14.580$, $\Delta df = 6$, AIC = 2.580), suggesting that the relative influence of genetic and environmental factors on gambling behavior changed between ages 18 and 25. The better-fitting model was one in which A, C, and E parameters, as well as the genetic, shared and nonshared environmental correlations, were constrained across sex, but allowed to vary at each age ($\Delta -2\text{LnL} = 7.069$, $\Delta df = 7$, AIC = −6.931). For each parameter in this model, a 95% confidence interval (CI) was estimated. Genetic [A^2_{18} = .21, CI = (.01–.45); A^2_{25} = .57, CI = (.29–.70)], shared environmental [C^2_{18} = .55, CI = (.34–.74); C^2_{25} = .10, CI = (.01–.34)], and nonshared environmental [E^2_{18} = .24, CI = (.18–.31); E^2_{25} = .33, CI = (.26–.41)] contributions to common variance were all sig-

Table 1
Descriptive Statistics and Twin Intraclass Correlations With 95% Confidence Intervals for the Four Gambling Variables by Age and Sex

	Descriptive statistics				Twin correlations			
	Male		Female		Male		Female	
Variable	Mean	*SD*	Mean	*SD*	r_{MZ}	r_{DZ}	r_{MZ}	r_{DZ}
Age 18	(*N* = 552)		(*N* = 639)					
Max frequency[a]	2.32	.99	1.83	.86	.52 (.40–.62)	.37 (.21–.53)	.58 (.48–.67)	.42 (.28–.55)
Typical frequency[b]	1.39	.41	1.19	.24	.64 (.54–.72)	.55 (.39–.68)	.62 (.53–.70)	.55 (.42–.66)
Max amount[c]	1.89	.88	1.59	.78	.58 (.47–.67)	.44 (.25–.59)	.56 (.45–.65)	.53 (.38–.65)
Problem behavior[d]	.59	1.34	.21	.70	.35 (.20–.48)	.29 (.14–.45)	.49 (.36–.60)	.37 (.23–.51)
Age 25	(*N* = 605)		(*N* = 645)					
Max frequency[a]	2.30	.92	1.89	.73	.40 (.28–.51)	.25 (.07–.42)	.32 (.21–.43)	.27 (.12–.42)
Typical frequency[b]	1.36	.34	1.20	.22	.50 (.39–.60)	.40 (.22–.55)	.51 (.40–.61)	.38 (.22–.52)
Max amount[c]	2.52	1.20	1.90	.98	.54 (.43–.63)	.35 (.18–.51)	.44 (.31–.55)	.36 (.21–.49)
Problem behavior[d]	.48	.99	.26	.75	.29 (.17–.41)	.24 (.10–.39)	.25 (.11–.38)	.30 (.16–.45)

[a] Maximum frequency with which the respondent engaged in any of the listed gambling behaviors; response options ranged from *never* (1) to *weekly* (4). [b] Mean of the response frequencies for the gambling activities; response options ranged from *never* (1) to *weekly* (4). [c] Maximum amount of money lost in a single day; response options ranged from *less than a dollar* (1) to *1000 dollars or more* (5). [d] Sum of the problem gambling behaviors; response options include (1) *Yes* or (0) *No*.

Table 2
Standardized Parameter Estimates for the Four Gambling Variables

Variable	Male			Female		
	A[a]	C[b]	E[c]	A[a]	C[b]	E[c]
Age 18						
Max frequency	.24 (.03–.48)	.29 (.07–.48)	.48 (.38–.59)	.37 (.12–.58)	.22 (.03–.45)	.41 (.34–.51)
Typical frequency	.20 (.02–.47)	.45 (.19–.62)	.35 (.28–.44)	.27 (.09–.49)	.37 (.16–.54)	.36 (.29–.43)
Max amount	.24 (.02–.52)	.35 (.08–.54)	.42 (.33–.52)	.19 (.02–.46)	.40 (.15–.57)	.41 (.33–.50)
Problem behavior	.05 (.00–.30)	.29 (.06–.42)	.66 (.55–.78)	.37 (.17–.55)	.19 (.05–.35)	.44 (.34–.56)
Age 25						
Max frequency	.40 (.16–.52)	.03 (.00–.25)	.57 (.47–.68)	.30 (.07–.45)	.07 (.00–.28)	.63 (.53–.74)
Typical frequency	.40 (.11–.58)	.14 (.00–.39)	.47 (.38–.57)	.43 (.14–.61)	.13 (.00–.37)	.44 (.35–.55)
Max amount	.42 (.16–.60)	.13 (.00–.36)	.45 (.37–.55)	.30 (.03–.51)	.17 (.01–.40)	.53 (.43–.65)
Problem behavior	.19 (.03–.34)	.09 (.00–.26)	.72 (.62–.83)	.19 (.01–.44)	.17 (.00–.34)	.64 (.52–.77)

[a] A = Additive genetic influences. [b] C = Shared environmental (environments are common to twins and act to make them similar) influences. [c] E = Non-shared environmental (environments that are not common to twins and that act to make them dissimilar) influences.

nificantly different from zero at ages 18 and 25. The correlations between the genetic and shared environmental contributions at ages 18 and 25 was .54 (CI = .05–1.00) and .99 (CI = .28–1.00), respectively, suggesting considerable overlap between these influences at the two time points. Conversely, the nonshared environmental correlation, equal to .15 (CI = −.01–.30), indicated almost no association between unique environmental influences across time.

Discussion

The present study uses a longitudinal, community-based twin study design to model genetic and environmental effects on gambling behaviors from late adolescence to young adulthood. Our study adds to the small, yet growing scientific literature on genetic and environmental influences on the development of gambling. The results support the following conclusions: (a) the shared genetic and environmental contributions to a range of gambling behaviors and problems are largely similar in men and women, (b) the influence of genetic factors increases from age 18 to age 25 (21% at age 18% to 57% at age 25), (c) shared environmental influences are influential at age 18, but decrease from ages 18 to 25 (55% at age 18% to 10% at age 25), and (d) nonshared environmental influences are significant but small to moderate in magnitude at both ages.

Our findings demonstrating similarities between the sexes in the etiology of gambling frequency and problem gambling are consistent with those of recent large-scale behavioral genetic twin studies (Slutske & Richmond-Rakerd, 2014; Slutske et al., 2010) and a

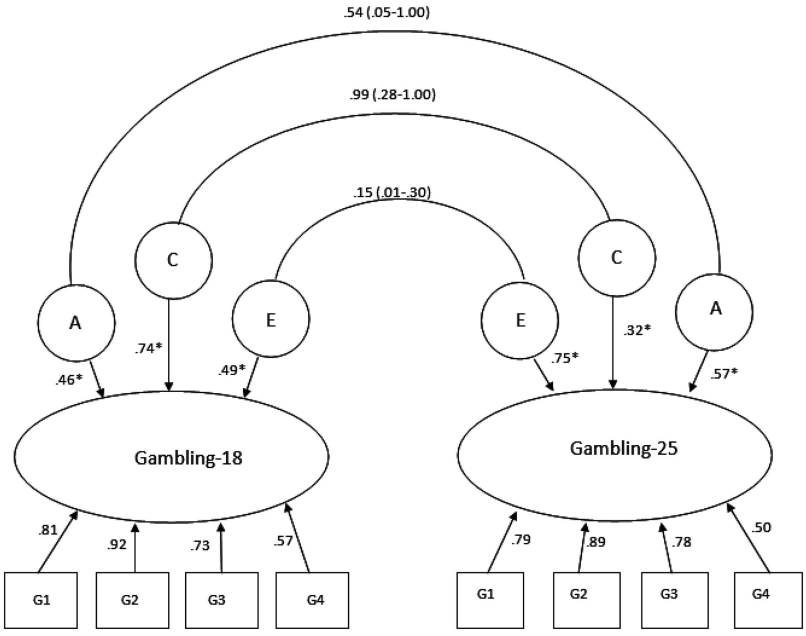

Figure 1. Path diagram of factor model of gambling from ages 18 to 25. G1, G2, G3, and G4 represent the following gambling phenotypes: Maximum frequency, typical amount gambled, maximum amount gambled, and gambling problem behavior. Factor loadings on path diagram represent an average across men and women.

Table 3
Standardized Factor Loadings for the Four Gambling Variables by Age and Sex

Variable	Age 18		Age 25	
	Male	Female	Male	Female
Max frequency	.81	.81	.79	.78
Typical frequency	.94	.90	.87	.91
Max amount	.69	.76	.83	.73
Problem behavior	.60	.53	.53	.48

sibling study of youth (Blanco, Myers, & Kendler, 2012). A few young adult twin studies have found evidence for sex differences in gambling behaviors (Winters & Rich, 1998; Beaver et al., 2010). The disparate findings in the twin literature on sex differences in the etiology of gambling may be attributable to the various developmental periods studied, retrospective reporting, types gambling measures (and severity of behaviors), regional and cohort effects, and sample composition.

On a phenotypic level, there was a moderate degree of intraindividual change in gambling behaviors over time. However, the relative importance of genetic and environmental influences explaining gambling changed substantially. The changing nature of adolescent peer relationships may influence the degree to which they engage in gambling behaviors and explain variability in the behaviors over time. This finding is novel, because few studies have attempted to directly model the changing environmental and genetic effects on gambling during this critical transition. Findings from a population based twin study indicated that for nonproblem gambling, shared and nonshared environmental factors accounted completely for nondisordered gambling behaviors (Slutske & Richmond-Rakerd, 2014). In contrast, disordered gambling was almost completely attributable to additive genetic effects, suggesting different etiologies of gambling behaviors compared with disorders (Slutske & Richmond-Rakerd, 2014). Expanding this area of research to longitudinal data, our study suggested that different nonshared environments may operate to influence gambling behaviors at age 18 and 25 (r_e = .15). Though we did not directly test specific candidate environments in our study, a possible nonshared environmental factor affecting gambling during this period is peer relationships.

Our findings suggest that the degree to which genes and environments influence gambling behaviors may shift or remain stable over time. For example, shared environmental factors influencing gambling at ages 18 and 25 were largely the same. In contrast, there was a moderate, yet significant correlation (r_a = .54) between the additive genetic factors influencing gambling at ages 18 and 25. Estimates of shared environmental influence decreased substantially over time (C_{18} = .55 to C_{25} = .10). As individuals transition from 18 to 25, the influence of genetic factors on gambling behavior became increasingly important (A_{18} = .21 and A_{25} = .57). As adolescents gain more independence during the transition to young adulthood, they may seek out and more effectively navigate environments which allow greater expression of inherited tendencies. With more time at or above the legal age limit to gamble, there may be more opportunities to gamble in peer groups or other social outlets. Also, certain specific shared environmental influences including parenting effects may be become relatively less important during this transition.

Our results are also in line with recent estimates on the heritability of gambling behaviors among youth, yet few have modeled change in the estimates over time. The findings of the current investigation (21% at age 18% to 57% at age 25) are within the range of other twin studies on gambling frequency in adolescents and young adults (e.g., 32% heritability for gambling frequency in adolescence; Blanco, Myers, & Kendler, 2012). However, most twin studies examining this stage in development had different methods or measures, which could affect the comparability of our findings to previous studies.

We found relative stability in the estimates of nonshared environmental influences. This factor remained moderate and significant from ages 18 to 25 (E_{18} = .24 and E_{25} = .33). Other studies (primarily cross-sectional) have found evidence for a nonshared environmental effect on gambling behaviors in adolescence and young adulthood (Beaver et al., 2010; see Lobo & Kennedy, 2009 for review). One study of gambling in young adulthood found evidence of shared and nonshared environmental effects (Beaver et al., 2010). Behavioral genetic studies of other addictive behaviors suggest that peer influences are influential in predicting early and escalating substance use involvement (Legrand, Keyes, McGue, Iacono, & Krueger, 2008; Legrand, McGue, & Iacono, 1999; Walden, McGue, Iacono, Burt, & Elkins, 2004). A recent twin study of youth ages 13 to 15 found evidence that although cotwin gambling did not predict gambling, delinquency was a predictor of gambling in both twins (Vitaro et al., 2014). Extending and advancing knowledge in this area will require targeted studies testing the specific effects of candidate environments in the context of genetic factors.

Differences in measurement approach and study methodology in twin studies of gambling may partially explain the range of shared environmental effects in the literature. Several adult problem gambling studies finding little, if any, shared environmental effects on disordered gambling (Lobo & Kennedy, 2009; Slutske et al., 2010). We found that shared environmental influences on gambling behaviors (but not specifically disordered gambling) decreased between age 18 (55%) and age 25 (10%), suggesting that as individuals age, the impact of shared environmental factors decreases over time. In a recent sibling and twin study, Blanco et al. (2012) found a significant and substantial effect of shared environmental factors on gambling frequency (average age = 25), whereas additive genetic factors were the only significant factor explaining disordered gambling. Our study design contrasts that of Blanco et al. (2012) in that gambling behaviors and problems were

Table 4
Footnote, Figure 1. Standardized Parameter Estimates of A, C, and E in Gambling Factor Model by Age

Age 18	Age 25
A^2 = .21	A^2 = .57
(.01, .45)	(.29, .70)
C^2 = .55	C^2 = .10
(.34, .74)	(.01, .34)
E^2 = .24	E^2 = .33
(.18, .31)	(.26, .41)

included in the measurement, and we utilized twins only in longitudinal design.

As individuals transition from late adolescence to young adulthood, increased access and new opportunities to gamble may emerge. For example, many individuals make the transition from college into the workforce, resulting in increased financial responsibilities, resources, and freedom. Friendship groups may shift from late adolescence to young adulthood. Individuals in our sample were passing through the legal age for gambling, which may offer new opportunities to gamble legally and increased financial independence. This may lead to increased risk taking around gambling. If candidate environments influencing youth gambling behaviors can be identified, we may be able to design a more effective approach to prevention and intervention efforts targeted for those at risk for gambling problems.

Although this study adds significantly to the relatively small behavioral genetic literature on gambling, there are several limitations that deserve comment. First, our study was focused on the transition from ages 18 to 25. A greater number of time points in the study would have enriched our findings, however data were only available at these assessments. Second, we did not aim to isolate specific environments or genes that impact gambling behaviors, whereas other studies have investigated environmental effects on problem gambling such as traumatic events in childhood in the context of a genetically informed design (Scherrer et al., 2007). Third, the sample was predominantly Caucasian which limits the generalizability of findings to other populations. The few genetically informative studies examining the role of candidate environments on gambling have primarily focused on adults with problem gambling, and therefore it is difficult to make direct inferences regarding the relevance to the present study findings.

The present study is one of the few to model the effects of genes and environment on gambling behavior from adolescence to young adulthood. The results suggest that the underlying etiological architecture of gambling behaviors is similar in men and women, with some age-specific effects. The study is a substantial contribution to the literature in several ways. There are few longitudinal twin studies focused on the transition to young adulthood that utilize a youth sample as compared with an adult sample (using retrospective reporting of past gambling histories). The findings highlight the shifting effects of genetic, shared environmental, and nonshared environmental influences on gambling behaviors during this critical transition. As young adults pass through this period, they may tend to seek out environments that allow for greater opportunity to express underlying genetic tendencies. The results of the study underscore the importance of identifying etiological critical periods for specific and targeted approaches to prevention and intervention (e.g., responsible gaming education, regulations on casinos, or peer prevention approaches). Future studies may benefit from identification of nonshared environments (e.g., peer gambling involvement, proximity to gambling venues, local gambling attitudes) influential in predicting gambling behaviors. Advancing research on the behavioral genetics of gambling by isolating the specific genes and environments related to youth gambling has the potential to prevent and decrease harm to those most at risk for developing gambling disorder.

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