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Test of a Potential Causal Influence of Earlier Age of Gambling Initiation on Gambling Involvement and Disorder: A Multilevel Discordant Twin Design

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The premise that an association between an earlier age of gambling initiation and the later development of disordered gambling is causal has not yet been empirically examined. The current study used a multilevel discordant twin design to examine the nature of this association. Participants were 3,546 same-sex twins (mean age = 37.7 years) from the Australian Twin Registry who completed a telephone interview that included an extensive assessment of gambling and related behaviors. Multilevel models were employed to estimate individual (within-twin-pair comparison) and family level (between-twin-pair comparison) effects, as well as the cross-level interaction between these effects. Family-level effects (genetic or environmental factors shared by family members) of age of gambling initiation robustly predicted later adult gambling frequency and disorder; the evidence for individual-level effects (unique factors not shared by family members, including a potentially causal effect of earlier age of gambling onset) was less robust. The results of this study suggest that the relation between earlier age of gambling initiation and later gambling involvement and disorder is primarily noncausal; efforts to delay the onset of gambling among young people may not necessarily reduce the number who later go on to develop gambling-related problems.

Keywords: gambling, disordered gambling, age of onset, discordant twins, multilevel modeling

There is accumulating evidence that the age at which one initiates the use of substances is related to the likelihood of later developing substance use problems. This phenomenon has been well documented with respect to the age of initiation of alcohol use and later alcohol use disorders (DeWit, Adlaf, Offord, & Ogborne, 2000; Grant & Dawson, 1997; Hingson, Heeren, & Winter, 2006), but also applies to the age of first cigarette smoked and later nicotine dependence (Breslau, Fenn, & Peterson, 1993; Everett et al., 1999), the age of initiation of illicit drug use and later drug use disorders (Grant & Dawson, 1998), and the nonmedical use of prescription drugs and later abuse or dependence on prescription drugs (McCabe, West, Morales, Cranford, & Boyd, 2007).

Fewer studies have explored links between the age of initiation and later problematic behavior for other non-substance-related addictive behaviors, such as disordered gambling; the limited evidence points to an association. Two studies based on large United States cross-sectional national surveys have examined whether an earlier age of onset of gambling participation forecasts future gambling problems. Lynch, Maciejewski, and Potenza (2004) found that young adults who had initiated gambling prior to age 18 were more likely to have experienced at least one symptom of disordered gambling than those who had initiated gambling at age 18 or later. Similarly, Kessler et al. (2008) found that the mean age of onset of gambling was significantly younger among individuals who subsequently developed pathological gambling disorder (16.7 years), compared to those who did not develop gambling problems (23.9 years).

There are a number of possible mechanisms that might explain the association between an early age of gambling onset and the development of disordered gambling. An important distinction is between causal and noncausal mechanisms. In a previous paper,

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we applied multilevel modeling of data from twin pairs to adjudicate between causal and noncausal mechanisms in explaining the inverse relation between the age of drinking initiation and the frequency and quantity of alcohol use in early adulthood (Deutsch et al., 2013). Effects at the individual level, family level, and their interaction explained the inverse relation between the age of alcohol use initiation and adult alcohol involvement. For example, the inverse relation between the age of alcohol use initiation and the frequency of drinking in early adulthood was explained in part by differences within twin pairs who were discordant for their age at first drink. The earlier-drinking twin drank more frequently in adulthood than did her later-drinking cotwin. This individual-level effect controls for genetic and shared environmental factors and therefore can be attributed to the unique or individual-specific environment; it is consistent with a possible causal influence of age at first drink on the frequency of drinking in adulthood. In addition to the within-twin-pairs effect were differences between twin pairs, that is, twin pairs who (on average) started drinking earlier drank more frequently than did twin pairs who (on average) started drinking later. This familial-level effect represents a noncausal influence of systematic genetic or environmental differences between families that are related to both earlier age of drinking initiation and more frequent drinking in adulthood. Finally, there was a significant interaction between the individual- and familial-level effects. At low levels of familial risk, individual onset of drinking was a stronger predictor of later drinking frequency than at higher levels of familial risk. The importance of the individual-level effect decreased as familial risk increased, that is, familial risk was a much stronger predictor of later drinking frequency than was the individual-level age of initiation effect when the familial risk was high.

In the present study, we used a similar multilevel twin design to attempt to adjudicate between causal and noncausal mechanisms in explaining the inverse relation between an earlier age of gambling initiation and later gambling involvement and disorder in adulthood. Initiating gambling earlier in life may set the stage for later problems through a number of causal pathways. For example, one pathway may be via peer socialization and selection effects. Individuals who initiate gambling early may associate with peers who also gamble (i.e., selection). This in turn may foster a more pro-gambling environment (socialization), which may increase normalization of gambling, interfere with the attainment of important life goals (Derevensky & Gupta, 2007; Wilber & Potenza, 2006), or even facilitate neuroadaptive changes that might occur in response to repeated exposures to gambling (Olsen, 2011; Verdejo-García, Lawrence, & Clarke, 2008; Zack & Poulos, 2009).

Noncausal mechanisms that might explain the association between earlier onset of gambling and later disordered gambling are background factors such as genetic or family environmental influences that are associated with both the uptake of gambling at a younger age and the development of disordered gambling. For example, genetic influences on individual differences in personality characteristics such as impulsivity may lead to both early gambling involvement and later disordered gambling. Gambling may also be learned through familial socialization; individuals who have parents who gamble or who have a history of gambling disorder may be exposed to more pro-gambling attitudes and these may encourage higher levels of gambling behavior (see McComb & Sabiston, 2010 for a review). Finally, there also may be interactions between causal and noncausal mechanisms. For instance, there may be situational "goads" (Sher et al., 2010) such as exposure to gambling peers, gambling advertising, or proximity to gambling venues (Abbott, 2007) that interact with genetic liability to lead to an earlier age of gambling initiation as well as increase the risk for later disordered gambling.

We focused on predicting three gambling-related outcomes in adulthood: lifetime symptoms of disordered gambling, and the frequency of gambling in the past year and during the peak gambling period. There were a number of reasons for including normative gambling involvement in addition to disordered gambling as an outcome. First, disordered gambling is relatively rare, whereas gambling involvement is common. Fully characterizing the gambling outcomes of early initiation required broadening the scope beyond disordered gambling. Second, past-year gambling frequency had the benefit of requiring minimal retrospection compared to a lifetime history of symptoms of disordered gambling, and so we could be more confident in the veracity of the results based on this outcome. Third, although some of the participants may not have passed fully through the period of risk for developing disordered gambling, frequent gambling is an important prerequisite and risk factor for later disordered gambling. Fourth, studies that have linked disordered gambling to retrospectively reported ages of gambling onset might be subject to recall bias wherein the first gambling event may be more salient and memorable to those who have experienced gambling problems in adulthood than to those who have not. The same may not be the case for normative gambling involvement. Fifth, nondisordered gambling involvement is itself an important and consequential outcome that is associated with poor physical and mental health and participation in health-risk behaviors among adolescents (Yip et al., 2011) as well as adults (Desai, Desai, & Potenza, 2007; Steenbergh, Whelan, Meyers, Klesges, & DeBon, 2008).

Childhood disruptive behavior disorders were included as covariates in the statistical models. The inclusion of such covariates is important to avoid incorrect causal inferences (see McGue, Osler, & Christensen, 2010). Studies of discordant twins can control for shared genetic and environmental factors, but they cannot control for unique environmental effects. Childhood disruptive behavior disorders were selected because there is some evidence that they may be related to both early age of gambling initiation and later gambling involvement and disorder. For example, prospective research has demonstrated that childhood conduct disorder (CD), oppositional defiant disorder (ODD), and attention-deficit hyperactivity disorder (ADHD) predict early age of initiation of alcohol, nicotine, and cannabis use (McGue, Iacono, Legrand, Malone, & Elkins, 2001; King, Iacono, & McGue, 2004), and it is likely that they may also play a role in the early uptake of gambling. A history of childhood CD (Kessler et al., 2008; Slutske et al., 2001) and ADHD (Crockford & el Guebaly, 1998) has also been linked to disordered gambling in adulthood.

The current study was guided by the following four hypotheses. First, based on previous studies regarding the relation between gambling onset and later gambling behavior, as well as research on similar relations for other addictive behaviors, we predicted that an earlier age of gambling initiation would predict higher rates of gambling behavior in adulthood (e.g., frequency of gambling and number of disordered gambling symptoms). Second, we hypothe-

sized that twins who gambled earlier than their cotwins would gamble more frequently and exhibit more symptoms of disordered gambling in adulthood compared to their cotwins (individual-level effect). Third, we also hypothesized that twin pairs who had earlier average ages of gambling initiation compared to other twin pairs would have higher levels of gambling behaviors compared to other twin pairs (familial-level effect). Fourth, cross-level interactions of individual-level and familial-level effects of age of gambling initiation on later gambling behavior were examined; we hypothesized that individual-level effects would be more pronounced within a high-risk family context.

Method

Participants

Participants were 3,623 members of the Australian Twin Registry Cohort II (for more information about participants, see Slutske et al., 2009). Only twins from same-sex pairs were included in this study. Respondents completed a structured psychiatric telephone interview conducted in 2004–2007 (mean age = 37.7 years, range = 32–43 years, response rate = 80.4%) during which gambling behaviors and adult and child psychiatric disorders were assessed. An assessment of conduct disorder was in a separate interview conducted about 8 years prior to the assessment of gambling, in 1996–2000 (see Meier, Slutske, Heath, & Martin, 2011). Of the 3,623 participants, only 1.9% (n = 77) were lifetime abstainers from gambling. These individuals were not included in this study, leaving a final sample size of 3,546 (1,156 MZ females, 898 DZ females, 830 MZ males, and 662 DZ males). From this sample, 1401 twin pairs had complete data for both twins (N = 2,802). This was the sample used for the multilevel models because they required complete twin pair data.

Procedure

Interviews were conducted by trained lay interviewers who were blind to the status of the cotwin. Retest data were collected 3.4 months (SD = 1.4, range = 1.2–9.5) after the initial interview for a small subsample (n = 166) of the twins in order to establish the reliability of the measures. An exception to this was the conduct disorder assessment, for which retest data were collected 3.7 years (SD = 0.4, range = 1.1–4.3) after the earlier interview for a small subsample (n = 215) of the twins. Informed consent was obtained from all participants and the study was approved by the Institutional Review Boards at the University of Missouri and the Queensland Institute of Medical Research.

Measures

Age of gambling initiation (AFG). Participants were asked how old they were the first time that they had engaged in 11 different gambling activities. Individuals' earliest reported age was coded as their AFG (see Richmond-Rakerd, Slutske, Heath, & Martin [2013] for the specific gambling activity that represented the first gambling experience for the participants in this study). Responses ranged from 4–41 years. Mean ages for men and women were 17.3 years (SD = 3.6) and 18.3 years (SD = 4.3), respectively; 34.5% of individuals started gambling at age 18 (the legal age to gamble throughout Australia), which was a considerably greater percentage than at any other age. The test–retest reliability of AFG was very good (r = .75). The correlation between individuals' ages at interview and their reported AFG of r = .05 indicated that older individuals did not report notably later ages of onset than did younger individuals, suggesting minimal age-related retrospective bias. Participants were also asked who was with them and where they were the first few times that they gambled.

Frequency of gambling during the past year (past year). After responding to an extensive set of questions about involvement in 11 specific gambling activities, participants were instructed that "For the remaining questions, when I refer to *gambling*, I am talking about any of the different activities that we have been discussing." Participants were asked how many days they had gambled during the last 12 months using a 14-point scale that ranged from *every day* to *never*. The scale was recoded to reflect days within a week rather than days within a year. For example, the answer "50–99 days (1 day a week)" was recoded as 1, "every day" was recoded as 7, and "2 days (2 days per year)" was coded as .02 (i.e., 2 ÷ 52, or .02 a week). Among the participants in this study, 385 (10.9%) had not gambled in the past year.

Frequency of gambling during the year of gambling the most (max year). After reporting about gambling involvement in the past 12 months, participants were asked whether "there has ever been a time when you were spending more time gambling than you have in the past 12 months?" Among the participants in this study, 1,055 (29.7%) had a 12-month period when they gambled more than in the past year. These participants were asked how frequently they had gambled "during the 12-month period in your life when you were gambling the most." The same scale measuring past-year frequency was used, and was again recoded to reflect weekly gambling frequencies. The frequency of gambling during the year of gambling the most was obtained by combining responses to the two questions about either the past year (for those responding negatively to the aforementioned question) and the 1-year period of gambling the most (for those responding affirmatively to the aforementioned question). For the 1,055 individuals whose period of gambling the most did not include the past 12 months, the average age at which this period began was 24.3 (SD = 5.4) years (24.1 among men, 24.5 among women).

Lifetime disordered gambling. Disordered gambling was assessed using the National Opinion Research Center *DSM–IV* Screen for Gambling Problems (Gerstein et al., 1999). Among the participants in the study, 443 (12.5%) had experienced at least one disordered gambling symptom in their lifetime; 239 (6.8%), 70 (2.0%), 40 (1.1%), 18 (0.5%), 28 (0.8%), 16 (0.5%), 13 (0.4%), 6 (0.2%), 7 (0.2%), and 6 (0.2%) participants had experienced from 1 to 10 disordered gambling symptoms, respectively. The disordered gambling symptom count evidenced high test–retest (r = .86) and internal consistency (α = .85) reliability and validity as indicated by significant associations with disordered gambling as measured by the South Oaks Gambling Screen (r = .68; Slutske, Zhu, Meier, & Martin, 2011). The average age of the first disordered gambling symptom was 25.9 (SD = 6.7) years (25.0 among men, 28.4 among women).

Conduct disorder symptoms. Conduct disorder was evaluated using a 15-item symptom count based on *DSM–IV* diagnostic criteria. Participants retrospectively reported on the

symptoms of CD that they had experienced before age 18. The mean number of symptoms was 0.35 (SD = 0.52) for the full sample and was 0.56 (SD = 0.60) among men and 0.21 (SD = 0.41) among women. Skewness of the variable (skewness = 2.60) required a log-transformation to approximate normality (skewness = 1.27). The internal consistency (α = .63) and the 4-year test–retest reliability (r = .75) of the CD symptom count were good. Potential age-related bias was examined by correlating participants' ages at interview with their reported number of childhood CD symptoms. The correlation of only −.02 suggested minimal age-related bias.

Oppositional defiant disorder symptoms. Oppositional defiant disorder was evaluated using an 8-item symptom count variable based on *DSM–IV* diagnostic criteria. Participants retrospectively reported on the eight symptoms of ODD that they had experienced before age 18. The mean number of symptoms was 2.28 (SD = 1.96) for the full sample and was 2.39 among men (SD = 2.01) and 2.20 among women (SD = 1.92). Skewness of the variable (skewness = 1.58) required a log-transformation to approximate normality (skewness = .85). The internal consistency reliability (α = .82) and the 3-month test–retest reliability (r = .74) of the ODD symptom scale were very good. Potential age-related bias was examined by correlating participants' ages at interview with their reported number of childhood ODD symptoms. The correlation of only −.01 suggested minimal age-related bias.

Attention-deficit hyperactivity disorder symptoms. Attention-deficit hyperactivity disorder symptoms were evaluated using an 18-item symptom count based on *DSM–IV* diagnostic criteria. Participants retrospectively reported on the nine inattention and the nine hyperactivity-impulsivity ADHD symptoms based on their behaviors when they were 6–12 years old. After endorsing a symptom, participants were asked whether this was more than other boys (girls) their age, and if they were not sure, whether the behavior occurred "often." The mean number of symptoms was 2.47 (SD = 2.78) for the full sample and was 2.93 among men (SD = 3.18) and 2.14 among women (SD = 2.39). Skewness of the variable (skewness = 2.67) required a log-transformation to approximate normality (skewness = 1.19). The internal consistency reliability (α = .89) and the 3-month test–retest reliability (r = .79) of the ADHD symptom scale were excellent. Potential age-related bias was examined by correlating participants' ages at interview with their reported number of childhood ADHD symptoms. The correlation of only .01 suggested minimal age-related bias.

Analytic Plan

The discordant-twin design is a natural experiment in which an unexposed twin serves as the control for an exposed cotwin. A comparison of outcomes such as disordered gambling observed among twins who are discordant for early gambling onset allows one to control for family environmental background factors and (completely in the case of monozygotic [MZ] twins, partially in the case of dizygotic [DZ] twins) for genetic factors. The within-twin-pair comparison "controls" for shared familial influences by comparing one twin to his or her cotwin, and is often employed to help determine causal influence between an exposure and an outcome. Statistical techniques such as multilevel mixed modeling also allow one to model effects at the familial (between-twin pairs) (Snijders & Bosker, 1999) as well as the individual (within-twin pairs) levels. These models also allow for examination of potential cross-level interactions. Such an interaction would imply a gene × environment or person × environment influence of initial onset of gambling on later gambling behavior.

Two-level models were estimated using SAS 9.3 (SAS Institute Inc, 2009) PROC GLIMMIX, a statistical procedure used for mixed models that allows for random effects. In using mixed models for clustered data (each twin pair = 1 cluster), the individual twin (within twin pair/level 1) is nested within the twin pair (between twin pair/level 2). Both level 1 and 2 variances were estimated, along with a random intercept of twin pair.

The interpretation of the level 1 (i.e., within twin pairs) and 2 (i.e., between twin pairs) parameters in these models depends upon the method used to center the level 1 predictor (Enders & Tofighi, 2007). When the level 1 predictor is group-mean centered (i.e., individual twin gambling onset minus the average onset of the twin pair), the level 1 and level 2 predictor coefficients represent the direct within-twin-pair (comparison against cotwin) and between-twin-pair (comparison against other twin pairs) effects. When the level 1 predictor is grand mean centered (i.e., individual twin gambling onset minus a constant), the level 1 predictor coefficient represents the direct within-twin-pair effect (as long as the level 2 predictor is also in the model) and the level 2 predictor coefficient represents the incremental effect of the between-twin-pair effect while controlling for level 1 effects. Therefore, variables were group-mean centered in some models to estimate direct effects and grand-mean centered in others to estimate incremental effects (see Enders & Tofighi, 2007).

A log-normal link function was used for past-year frequency and max-year frequency variables due to skewness (3.45 and 3.02, respectively). Lifetime disordered gambling symptoms were estimated using a zero-inflated Poisson model after indicating that it was a better fit than a traditional Poisson regression ($\Delta\chi^2$ = 198.31, $p < .001$) for the initial model. Zero-inflated Poisson models account for overdispersion of zeroes within count data.

Two identical sets of analyses were conducted. One set of analyses was based on the MZ and DZ twin data in order to examine overall twin effects, the other set of analyses was restricted to MZ twin data in order to allow for the most stringent tests of unique environmental causality by completely controlling for shared genetic factors. Each outcome was tested using a series of seven models (see Table 1). In the first six models the variables were group-mean centered and in the seventh model the variables were grand-mean centered. The first model tested the main effects of gender, zygosity, age, level 1 (individual gambling onset) and level 2 (twin average gambling onset) effects. Then cross-level (level 1 by level 2) and quadratic level 2 interactions were added in the second model to test for moderation effects. The third model examined interactions of gender and zygosity with the gambling onset variables (both level 1 and level 2), and significant interactions were retained for subsequent models. Gender moderated the main effect of the within-twin-pair (level 1) age of gambling

Table 1
Summary of the Series of Multilevel Regression Models Tested; In Models 1–6, Each Model Includes All of the Parameters Included in the Previous Model

Model	Model description	Main parameters of interest	Explanation and interpretation
1	main effects	Group mean centered individual gambling onset (IGO)	Level 1 within-twin-pair effect potentially causal unique environmental influence
		Grand mean centered twin average gambling onset (TGO)	Level 2 between-twin-pair effect familial effect of genetic or shared environmental influences
2	plus main effects interactions	IGO × TGO	Cross-level interaction potentially causal influence moderated by context
		TGO × TGO	Nonlinear effect of context—ensuring that the moderation of IGO by TGO is occurring similarly at all levels of TGO.
3	plus gender and zygosity interactions	gender × IGO	Level 1 potentially causal influence moderated by gender
		gender × TGO	Level 2 familial effect moderated by gender
		zygosity × IGO	Level 1 potentially causal influence moderated by zygosity
		zygosity × TGO	Level 2 familial effect moderated by zygosity
4	plus ADHD effects	individual ADHD	Level 1 control for unique environmental influence of ADHD
		twin average ADHD	Level 2 control for familial effect of ADHD
5	plus ODD effects	individual ODD	Level 1 control for unique environmental influence of ODD
		twin average ODD	Level 2 control for familial effect of ODD
6	plus CD effects	individual CD	Level 1 control for unique environmental influence of CD
		twin average CD	Level 2 control for familial effect of CD
7	incremental effects	Grand mean centered IGO	Level 1 within-twin-pair effect
		Grand mean centered TGO	Incremental between-twin-pair effect

Note. ADHD = attention-deficit hyperactivity disorder symptoms; ODD = oppositional defiant disorder symptoms; CD = conduct disorder symptoms; IGO = individual gambling onset; TGO = twin average gambling onset.

onset for both the past-year frequency and max-year frequency outcomes, and this interaction was included in subsequent models for both outcomes. There were no interactions between age of onset variables and zygosity for the past-year frequency outcome. However, zygosity moderated the relationship between the between-twin-pair (level 2) effect and max-year frequency of gambling (b = .04, p = .04), and the interaction between the between-twin-pair effect and zygosity approached significance for the disordered gambling symptom outcome (b = .09, p = .06). This interaction was included in subsequent models for both the max-year gambling frequency and the disordered gambling symptom count.

The fourth, fifth, and sixth models added level 1 and 2 ADHD effects, level 1 and 2 ODD effects, and level 1 and 2 CD effects, respectively. The level 1 and 2 ADHD, ODD, and CD variables were used in order to account for the effect of each disorder when examining both levels of age of gambling onset. We also examined the interactions between the within-twin gambling onset and the level 1 (within-twin) and level 2 (between-twin) ADHD, ODD, and CD symptoms for each of the three gambling outcomes (18 interactions altogether), and none were statistically significant (within-twin: bs = .01−.12, ps = .09−.94; between-twin: bs = .01−.02, ps = .30−.78). Therefore, these interactions were not retained in any of the models.

Finally, we returned to a variant of the first model that used a different method of centering. The seventh model tested the incremental effect of gambling onset by using a grand-mean centered level 1 variable (centered at the sample mean). Both the grand-mean-centered level 1 variable, and the level 2 variable were entered in the model along with gender, zygosity, and age in order to test if between-twin-pair differences in the age of gambling onset had an incremental effect on past-year and max-year gambling frequency and lifetime disordered gambling symptoms.

Results

Descriptive Analyses

The average age of gambling initiation was 17.86 years (SD = 4.11). Most twin pairs (80%) were discordant for age of gambling onset. Discordance was treated as a continuous variable representing the absolute difference in years of the age of onset of the first twin minus the age of onset of the second twin. Thus, twins whose ages of onset differed by one or more years were considered discordant. The average discordance was 3.25 (SD = 3.45) years.

The types of people that participants were most likely to have been with their first few times gambling were (in order of fre-

quency): friend (46.3%), cotwin (45.0%), parents (29.8%), alone/nobody (18.8%), partner (17.7%), and older sibling (15.3%). MZ twins were significantly more likely than DZ twins to have been with their cotwin (49.8% vs. 38.7%; $\chi^2 = 43.72$, $df = 1$, $p < .001$) and significantly less likely to have been with an older sibling (13.2% vs. 17.9%; $\chi^2 = 14.30$, $df = 1$, $p < .001$) the first few times they gambled. The most likely locations for the occurrence of the first few times gambling were (in order of frequency): newsagent[1] (41.2%), club[2] (23.0%), hotel/pub[3] (21.3%), race track[4] (17.3%), and casino (16.3%).

The mean past-year frequency of gambling was 0.58 (i.e., on average, individuals gambled approximately once every 2 weeks), the mean max-year frequency of gambling was 0.76 (i.e., on average, individuals gambled approximately once every week and a half during the period when they were gambling the most), and the mean number of symptoms of lifetime disordered gambling was 0.30 in adulthood. The age of gambling initiation was modestly associated with the three gambling outcomes (see Table 2 for means for and correlations between the study variables). Graphical displays of the relations between the age of gambling initiation and past-year and max-year frequencies of gambling and the lifetime disordered gambling symptom count among men and women are presented in Figures 1, 2, and 3.

Mixed Models

Past-year gambling frequency. The two-level model predicting past-year gambling frequency is presented in Table 3. Both within-twin-pair and between-twin-pair effects were significant. The within-twin-pair effect indicated a causal relation between age of gambling onset and past-year gambling frequency, such that for every year Twin 1 started gambling earlier than Twin 2, Twin 1 increased past-year gambling by approximately 2 days a year (Model 1, $b = -.02$, $p = .03$). The between-twin-pair effect indicated that there was also a familial effect (i.e., genetic or shared environmental effect) of age of gambling onset such that for every year decrease in the average age of gambling onset for the twin pair compared to other twin pairs, both twins increased in gambling frequency approximately 4 days a year (Model 1, $b = -.07$, $p = .01$). There was no cross-level interaction between level 1 and level 2 gambling onset (Model 2), and therefore there was no need to include either the cross-level interaction or the quadratic level 2 interaction in the final model. There was also a significant interaction between within-twin gambling age of onset and gender: as shown in Figure 4 (panel a), the within-twin effect was stronger for men than for women (Model 3). Finally, ADHD and ODD symptoms, but not CD symptoms, significantly predicted past-year gambling frequency. There were both within-twin and between-twin effects of ADHD, indicating that both the average symptom count of ADHD for the twin pair, and the individual symptom count of Twin 1 compared to Twin 2 predicted past-year gambling frequency (Model 4). There was a between-twin-pair (level 2) effect of ODD, indicating that as the twin-pair average ODD symptoms increased, past-year gambling frequency also increased (Model 5). Both the within-twin age of initiation effect and the interaction between the within-twin gambling age of initiation and gender became nonsignificant when accounting for childhood externalizing behaviors, indicating that underlying psychopathology may account for the relationship between individual age of gambling initiation and past-year gambling frequency. However, the between-twin-pair effect of gambling age of initiation was still significant in the full model (Model 6), indicating that underlying psychopathology could not fully explain the relation between familial effects of age of gambling initiation and gambling frequency. Finally, the between-twin-pair (level 2) effect was tested as an incremental effect (Table 1, Model 7). This effect was significant ($b = -.05$, $p = .01$) indicating that the between-twin gambling age of initiation effect was significantly stronger than the within-twin gambling age of initiation effect in predicting past-year gambling frequency.

Table 3 also shows the same model using an MZ-only sample. Although the between-twin-pair effect was significant in this model, there was not a significant within-twin-pair effect (Model 1). However, as there was not a significant interaction between the within-twin-pair effect and zygosity in the full sample ($b = -.02$, $p = .20$), this may be due to a reduced sample size rather than to a difference between the MZ and DZ twins. There was also a significant incremental between-twin-pair effect ($b = -.08$, $p = .01$), such that the between-twin gambling age of initiation effect was significantly stronger than the within-twin gambling age of initiation effect in predicting past-year gambling frequency (Table 1, Model 7).

Max-year gambling frequency. Table 4 displays the model predicting max-year gambling frequency. Both within-twin-pair and between-twin-pair effects of gambling initiation significantly predicted the max-year frequency of gambling (Model 1). There was not a cross-level effect, indicating that the within-twin-pair effect was not moderated by the between-twin-pair effect (Model 2). However, gender significantly moderated the within-twin-pair effect of gambling initiation (Model 3), such that the effect was stronger for men compared to women (see Figure 4, panel b). Zygosity also significantly moderated the effect of between-twin-pair age of gambling initiation, indicating that the between-twin effect was stronger for MZ than for DZ twins. Again, the between-twin-pair effect was significant even after controlling for ADHD, ODD, and CD symptoms, while the within-twin-pair effect (and the interaction with gender) disappeared (Models 4–6). Finally, there was also an incremental effect of between-twin-pair gambling initiation ($b = -.06$, $p = .01$), indicating that the between-twin-pair effect was significantly stronger than the within-twin-pair effect in predicting max-year gambling frequency (Table 1, Model 7).

The model using the MZ-only sample was similar to the model using the full sample. Both within-twin-pair and between-twin-pair

[1] Australian newsagents (newsstands) usually do business in well-trafficked places like city streets, airports, and train stations. They sell lottery tickets. The minimum age to purchase lottery tickets (or other lottery products such as "scratchies") in Australia is 16.

[2] A registered club in Australia serves as a place for social gatherings. They vary considerably but often have dining, lodging, sports, and other entertainment facilities. At most registered clubs one can play electronic gaming machines ("pokies" or poker machines) and place off-course bets.

[3] Australian hotel/pubs are modeled on similar establishments in Britain. Traditionally, this was considered a male-dominated venue. At hotel/pubs one typically can play electronic gaming machines ("pokies" or poker machines), keno, and place off-course bets.

[4] Australia has more horse racecourses (racetracks) than any other country in the world.

Table 2
Correlations Between Study Variables

	1	2	3	4	5	6	7	Overall mean (*SD*)
1. Age of gambling initiation	—	−.11	−.12	−.04	−.08	−.06	−.10	17.9 (4.10)
2. Past-year gambling frequency	−.12	—	.84	.22	.06	.09	.04	0.58 (0.95)
3. Max-year gambling frequency	−.16	.72	—	.37	.10	.11	.07	0.76 (1.16)
4. Lifetime disordered gambling	−.07	.31	.48	—	.09	.11	.09	0.30 (1.09)
5. ADHD	−.07	.15	.15	.19	—	.37	.22	2.47 (2.78)
6. ODD	−.07	.19	.18	.20	.46	—	.31	2.28 (1.96)
7. CD	−.13	.07	.11	.18	.27	.32	—	0.35 (0.54)

Note. Correlations above the diagonal are for women, correlations below the diagonal are for men. All correlations of $r > |.04|$ are statistically significant at $p < .02$. ADHD = attention-deficit hyperactivity disorder symptoms; ODD = oppositional defiant disorder symptoms; CD = conduct disorder symptoms.

effects of age of gambling initiation were initially significant predictors, but only the between-twin-pair effect was significant after controlling for ADHD, ODD, and CD symptoms. Finally, there was an incremental between-twin-pair effect in the MZ-only model ($b = -.08$, $p = .01$), indicating that the between-twin-pair effect was stronger than the within-twin-pair effect.

Lifetime disordered gambling symptoms. Table 5 displays the models predicting lifetime symptoms of disordered gambling. Only the between-twin-pair effect of gambling initiation was significant, indicating that for every year decrease in average twin pair age of gambling initiation, the disordered gambling symptom count increased by .09. However, twins who gambled earlier than their cotwins did not have more disordered gambling symptoms (Model 1). Again, for the full sample there was not a significant cross-level interaction effect (Model 2). There was a trend toward significance for the interaction between zygosity and the between-twin effect of age of gambling initiation (Model 3; $b = .09$, $p = .06$), indicating that the between-twin effect was stronger for MZ than for DZ twins. Again, the between-twin-pair effect was significant even after controlling for ADHD, ODD, and CD (Models 4–6). Finally, the incremental between-twin-pair effect approached significance ($b = -.06$, $p = .07$), indicating that the between-twin-pair effect was comparable to the within-twin-pair effect (Table 1, Model 7).

For the MZ-only sample, only the between-twin-pair effect was significant (Model 1). However, contrary to other models, there was a small but significant cross-level interaction effect (Model 2), such that the effect of an early initiation age of an individual twin was stronger when the average age for the twin pair was also earlier, but this effect was no longer significant when ODD was included in the model (Model 5). Furthermore, the between-twin-pair main effect disappeared when CD was included in the model

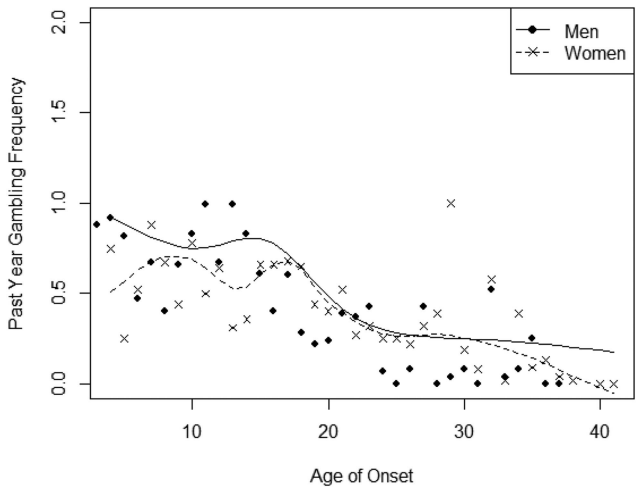

Figure 1. Smoothing spline plot representing past-year gambling frequency in adulthood as a function of age of initiation of gambling for men and women. The lines represent predicted data points as indicated by the smoothing spline parameter. Data points (dots and crosses) represent observed data points for men and women. Smoothing splines provide the best-fitting function by considering its average smoothness in conjunction with its goodness-of-fit. Goodness-of-fit is measured by residual sum of squares, while average smoothness is measured by the integral of the function's second derivative. The smoothing parameter controls the influence of smoothness on the overall best-fitting function. A smoothing spline was fit through the data to help visualize the trend in the age of gambling initiation. The smoothing parameter was chosen using the Generalized Cross-Validation (GCV) approach. The spline was weighted by number of men and women at each age of gambling initiation.

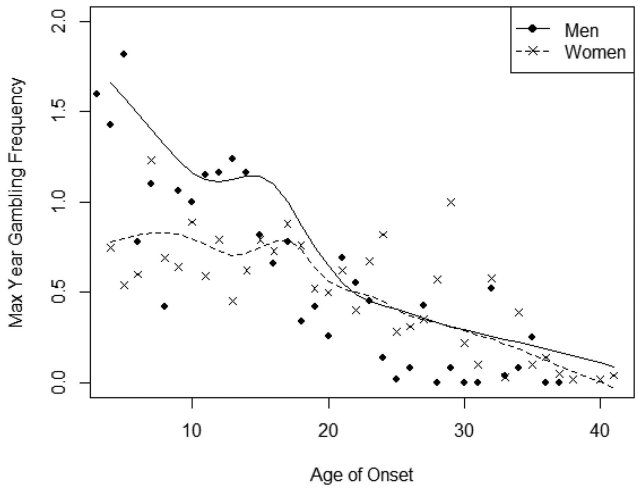

Figure 2. Smoothing spline plot representing gambling frequency during the period of maximum gambling as a function of the age of initiation of gambling for men and women. The lines represent predicted data points as indicated by the smoothing spline parameter. Data points (dots and crosses) represent observed data points for men and women.

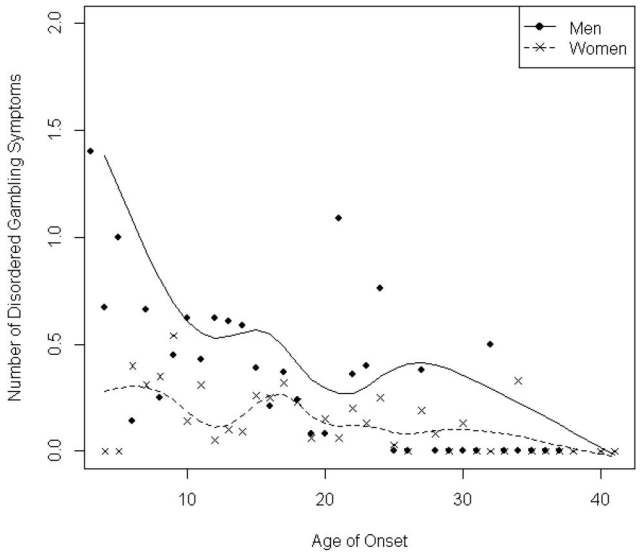

Figure 3. Smoothing spline plot representing lifetime disordered gambling symptom counts as a function of the age of initiation of gambling for men and women. The lines represent predicted data points as indicated by the smoothing spline parameter. Data points (dots and crosses) represent observed data points for men and women.

(Model 6). Finally, there was also an incremental between-twin-pair effect ($b = -.11$, $p = .01$), indicating that the between-twin-pair effect was stronger than the within-twin-pair effect (Table 1, Model 7).

Discussion

The purpose of the present study was to clarify the potentially causal nature of the relation between the age of onset of gambling and later gambling behavior using a multilevel discordant twin design. In this design, a causal effect of early gambling initiation on disordered gambling would be implicated if the earlier-initiating twin was more likely to develop disordered gambling symptoms than the later-initiating cotwin. If there was no difference between the earlier-initiating twin and the later-initiating cotwin in the development of disordered gambling symptoms, then early gambling initiation might be more aptly characterized as a marker or symptom of the vulnerability to develop disordered gambling, rather than a causal factor. Within the multilevel design, a noncausal effect of early gambling initiation on disordered gambling would be implicated if twin pairs who started gambling at an earlier age on average were more likely to develop disordered gambling symptoms than twin pairs who started gambling at a later age on average. We examined the nature of the relation with the age of gambling onset for three gambling-related outcomes: the cumulative number of lifetime disordered gambling symptoms, the past-year frequency of gambling, and the frequency of gambling during the peak period of involvement.

There was some evidence for a potentially causal effect of early gambling initiation on later gambling frequency among men, but this effect was no longer statistically significant when childhood externalizing disorders were included as covariates in the models. The inclusion of such covariates is important to avoid incorrect causal inferences (see McGue et al., 2010). Although comparisons of discordant twins can control for shared genetic and environmental factors, they still cannot control for unique environmental effects. In this case, there appeared to be unique environmental effects on childhood externalizing disorders that were common to the age of gambling initiation and later gambling frequency. For example, when twins differed in the number of childhood ADHD symptoms, it was the twin with more ADHD symptoms who initiated gambling at a younger age and went on to gamble more frequently as an adult. In other words, differences in ADHD symptoms may have been what led to twin discordance in the age

Table 3
Unstandardized and Standardized Estimates of Past-Year Gambling Frequency as Predicted by Age of Gambling Onset for the Full and the Monozygotic (MZ) Twin Samples

	Full sample					
Variable	M1	M2	M3	M4	M5	M6
Zygosity	.02 (.60)	.02 (.51)	.03 (.62)	.02 (.53)	.01 (.15)	.01 (.17)
Gender	.18** (4.43)	.16* (3.86)	.86* (21.18)	.78* (19.01)	.78* (19.06)	.70 (16.45)
Age	.03* (3.84)	.03* (3.87)	.03* (3.86)	.03* (3.75)	.03 (3.93)	.03* (3.44)
Within-twin gambling onset (Level 1)	−.02* (−2.49)	−.02* (−2.44)	−.01 (−.78)	−.01 (−.89)	−.01 (−.91)	−.01 (−.71)
Between-twin gambling onset (Level 2)	−.07** (−10.60)	−.07* (−13.83)	−.07** (−10.64)	−.06** (−10.02)	−.06** (−9.82)	.06** (−8.89)
Cross-level interaction	—	.01 (2.56)	N/A	N/A	N/A	N/A
Quadratic Level 2 interaction	—	−.01** (−5.58)	N/A	N/A	N/A	N/A
Within-twin × gender interaction	—	—	−.04* (−17.00)	−.06 (−15.98)	−.04 (−15.86)	−.03 (−13.44)
Within-twin ADHD	—	—	—	.17** (5.64)	.13** (4.27)	.13** (3.91)
Between-twin ADHD	—	—	—	.19** (7.35)	.11** (4.39)	.10* (3.66)
Within-twin ODD	—	—	—	—	.09 (1.91)	.08 (1.75)
Between-twin ODD	—	—	—	—	.23** (6.17)	.28** (7.24)
Within-twin CD	—	—	—	—	—	−.03 (−.40)
Between-twin CD	—	—	—	—	—	−.02 (−.44)

Note. Standardized estimates in parentheses. ADHD = attention-deficit hyperactivity disorder symptoms; ODD = oppositional defiant disorder symptoms; CD = conduct disorder symptoms. Because the cross-level interaction was not significant, both the cross-level and quadratic level 2 models were not included in models 3–6.
* $p < .05$. ** $p < .01$.

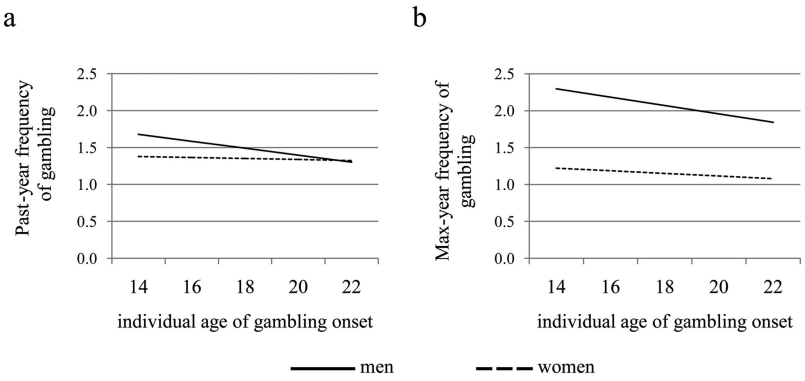

Figure 4. a) Past-year frequency of gambling as a function of within-twin gambling age of onset for men and women. (The effect of an early initiation age of an individual twin was stronger among men than among women.) b) Frequency of gambling during the year of gambling the most as a function of within-twin gambling age of onset for men and women. (The effect of an early initiation age of an individual twin was stronger among men than among women.)

of gambling initiation, and also to the differences in adult gambling frequency.

There was more consistent evidence of between-family differences explaining the associations between an earlier age of gambling initiation and later gambling involvement and disorder. This suggests that these relations were primarily due to genetic or shared environmental factors that were common to the age of gambling initiation and later gambling involvement and disorder. Between-family differences in levels of childhood externalizing disorders explained part of the association between the age of gambling onset and later gambling behavior, but this association remained even after controlling for the influence of childhood behavioral disorders. In sum, the evidence suggests that early gambling initiation is a marker of the potential to become a frequent gambler or to develop disordered gambling, rather than a direct causal influence.

We also hypothesized that the individual-level effect of an earlier age of gambling onset on later gambling involvement and disorder would be more pronounced within a high-risk family context. Whether high-risk family context was defined as a higher mean number of symptoms of externalizing disorders or a lower mean age of gambling initiation in a twin pair, there was no evidence for such a person–environment interaction. Nonetheless, a number of important environmental effects germane to gambling behavior were not included in this study, such as neighborhood socioeconomic disadvantage (Auger, Lo, Cantinotti, & O'Loughlin, 2010) and proximity to gambling venues (Pearce, Mason, Hiscock, & Day, 2008; Sévigny, Ladouceur, Jacques, &

Variable	MZ-only sample					
	M1	M2	M3	M4	M5	M6
Zygosity	—	—	—	—	—	—
Gender	.09 (1.67)	.05 (.88)	.31 (5.83)	.26 (4.94)	.26 (4.93)	.08 (1.61)
Age	.04* (3.73)	.04* (3.80)	.04* (3.73)	.04* (3.97)	.04* (3.95)	.05* (3.83)
Within-twin gambling onset (Level 1)	−.01 (−.85)	−.01 (−.66)	−.01 (−.47)	−.01 (−.44)	−.01 (−.81)	−.01 (−.60)
Between-twin gambling onset (Level 2)	−.09** (−10.24)	−.05** (−5.69)	−.09** (−10.25)	−.08** (−9.90)	−.08** (−9.64)	−.08** (−8.83)
Cross-level interaction	—	−.01 (−3.10)	N/A	N/A	N/A	N/A
Quadratic Level 2 interaction	—	−.01** (−6.81)	N/A	N/A	N/A	N/A
Within-twin × gender interaction	—	—	−.01 (−4.20)	−.01 (−3.88)	−.01 (−3.76)	−.01 (−.97)
Within-twin ADHD	—	—	—	.19** (1.31)	.16** (3.69)	.17** (3.75)
Between-twin ADHD	—	—	—	.15** (4.34)	.06 (1.77)	.04 (1.03)
Within-twin ODD	—	—	—	—	.04 (.64)	.05 (.77)
Between-twin ODD	—	—	—	—	.27** (5.67)	.29** (5.82)
Within-twin CD	—	—	—	—	—	−.06 (−.59)
Between-twin CD	—	—	—	—	—	.03 (.55)

Table 4
Unstandardized and Standardized Estimates of Max-Year Gambling Frequency as Predicted by Age of Gambling Onset for the Full and the Monozygotic (MZ) Twin Samples

Variable	Full sample					
	M1	M2	M3	M4	M5	M6
Zygosity	.04 (1.10)	.04 (.91)	.01 (.26)	.01 (.25)	−.01 (−.10)	−.01 (−.02)
Gender	.26** (6.78)	.24** (6.14)	.91** (23.80)	.86** (22.16)	.86** (22.23)	.71* (17.46)
Age	.03* (3.48)	.03* (3.64)	.03* (3.64)	.03* (3.60)	.03* (3.81)	.03* (3.56)
Within-twin gambling onset (Level 1)	−.03* (−3.95)	−.03** (−3.27)	−.02 (−2.25)	−.02 (−2.19)	−.02 (−2.16)	−.02 (−2.12)
Between-twin gambling onset (Level 2)	−.09** (−15.32)	−.03 (−4.83)	−.11** (−17.99)	−.11** (−17.40)	−.10** (−17.06)	−.10** (−15.67)
Cross-level interaction	—	−.01 (−8.87)	N/A	N/A	N/A	N/A
Quadratic level-2 interaction	—	−.01** (−6.62)	N/A	N/A	N/A	N/A
Within-twin × gender interaction	—	—	−.04* (−17.23)	−.04* (−16.82)	−.04* (−16.73)	−.03 (−12.33)
Between-twin × zygosity interaction			.04* (4.37)	.04* (4.44)	.04* (4.26)	.04* (4.30)
Within-twin ADHD	—	—	—	.16** (5.70)	.12** (4.17)	.13** (4.26)
Between-twin ADHD	—	—	—	.20** (7.92)	.12** (4.84)	.11* (4.26)
Within-twin ODD	—	—	—	—	.10* (2.32)	.11 (2.33)
Between-twin ODD	—	—	—	—	.22** (6.38)	.26** (6.79)
Within-twin CD	—	—	—	—	—	−.03 (−.37)
Between-twin CD	—	—	—	—	—	.05 (1.02)

Note. Standardized estimates in parentheses. ADHD = attention-deficit hyperactivity disorder symptoms; ODD = oppositional defiant disorder symptoms; CD = conduct disorder symptoms. Because the cross-level interaction was not significant, both the cross-level and quadratic level 2 models were not included in models 3–6.
$^{+}$ $p < .07$. * $p < .05$. ** $p < .01$.

Cantinotti, 2008; Welte, Wieczorek, Barnes, & Tidwell, 2006). The investigation of person–environment interactions in gambling behaviors incorporating more macrolevel environmental contexts (e.g., Auger et al., 2010), especially within a genetically informative design, will be an important next step.

In previous research we examined the genetic and environmental underpinnings of the age of gambling initiation (Richmond-Rakerd et al., 2013) and found significant sex differences. The percentage of variation in the age of initiation of gambling due to genetic influences was 36% among men and 6% among women, whereas the percentage of variation due to shared family environmental influences was 0% among men and 28% among women (Richmond-Rakerd et al., 2013). Thus, the between-family influences explaining the associations between earlier age of gambling initiation and later gambling involvement and disorder may be primarily genetic factors for men and shared family environmental factors for women. Another clue about the between-family effect might come from the fact that it was stronger in MZ than in DZ twin pairs for max-year frequency of gambling and disordered gambling, and MZ twins were more likely to have gambled with their cotwin than were DZ twins. This suggests that part of the between-family effect might reflect a same-age

Table 5
Unstandardized Estimates of Disordered Gambling Symptoms as Predicted by Age of Gambling Onset for the Full and the Monozygotic (MZ) Twin Samples

Variable	Full sample						MZ-only sample					
	M1	M2	M3	M4	M5	M6	M1	M2	M3	M4	M5	M6
Zygosity	.10	.10	.08	.09	.04	.05	—	—	—	—	—	—
Gender	.96**	.96**	.97**	.82**	.85**	.80**	.94**	.95**	—	.79**	.81**	.73**
Age	.04	.04	.04	.03	.03	.03	.06	.06	—	.05	.05	.05
Within-twin gambling onset (Level 1)	−.03	−.03	−.03	−.03	−.03	−.02	−.02	−.01	—	−.02	−.02	−.04
Between-twin gambling onset (Level 2)	−.09	−.15	−.13**	−.11**	−.10**	−.10**	−.13**	−.43**	—	−.40**	−.40**	−.24
Cross-level interaction	—	.01	N/A	N/A	N/A	N/A	—	.02*	—	.02*	.02	.01
Quadratic level-2 interaction	—	.01	N/A	N/A	N/A	N/A	—	−.01	—	−.01	−.01	−.01
Between-twin × zygosity interaction			.09+	.09+	.09+	.09+	—	—	—	—	—	−
Within-twin ADHD	—	—		.34**	.15	.15	—	—	—	.30*	.14	.12
Between-twin ADHD	—	—		.63**	.36**	.24*	—	—	—	.67**	.42**	.26
Within-twin ODD	—	—		—	.48**	.45**	—	—	—	—	.55**	.46**
Between-twin ODD	—	—		—	.73**	.72**	—	—	—	—	.73**	.74**
Within-twin CD	—	—		—	—	.13	—	—	—	—	—	−.02
Between-twin CD	—	—		—	—	.43*	—	—	—	—	—	.34

Note. ADHD = attention-deficit hyperactivity disorder symptoms; ODD = oppositional defiant disorder symptoms; CD = conduct disorder symptoms. Because the cross-level interaction was not significant, both the cross-level and quadratic level 2 models were not included in models 3–6.
* $p < .05$. ** $p < .01$.

Variable	MZ-only sample					
	M1	M2	M3	M4	M5	M6
Zygosity	—	—	—	—	—	—
Gender	.20* (3.97)	.16 (3.10)	.57 (11.52)	.54 (10.67)	.54 (10.88)	.27 (5.24)
Age	.03* (3.12)	.03* (3.38)	.03* (3.12)	.03* (3.19)	.04* (3.42)	.04* (3.24)
Within-twin gambling onset (Level 1)	−.03* (−2.51)	−.02 (−1.76)	−.02 (−1.84)	−.02 (−1.74)	−.02 (−1.67)	−.02 (−1.69)
Between-twin gambling onset (Level 2)	−.11** (−14.33)	−.02 (−2.72)	−.11** (−14.33)	−.11** (−14.02)	−.10** (−13.75)	−.10 (−12.91)
Cross-level interaction	—	−.01 (−9.54)	N/A	N/A	N/A	N/A
Quadratic level-2 interaction	—	−.01** (−7.67)	N/A	N/A	N/A	N/A
Within-twin × gender interaction	—	—	−.02 (−7.64)	−.02 (−7.51)	−.02 (−7.62)	−.01 (−2.15)
Between-twin × zygosity interaction	—	—	—	—	—	—
Within-twin ADHD	—	—	—	.20** (4.77)	.17** (3.86)	.19** (4.14)
Between-twin ADHD	—	—	—	.16** (4.94)	.08 (2.32)	.04 (1.28)
Within-twin ODD	—	—	—	—	.10 (1.57)	.10 (1.63)
Between-twin ODD	—	—	—	—	.25** (5.56)	.28** (5.98)
Within-twin CD	—	—	—	—	—	−.01 (−.08)
Between-twin CD	—	—	—	—	—	.03 (.58)

peer/sibling influence that is more pronounced among MZ twin pairs.

An unanticipated finding was the key role played by symptoms of ODD in childhood. ODD symptoms explained the between-family effect for all three outcomes even when ADHD was also included in the model. Childhood history of ODD symptoms also explained the within-twin-pair effect for disordered gambling. In fact, a history of childhood ODD appeared to be a better predictor of later gambling involvement and disorder than the age of gambling initiation, and the within-twin effect was consistent with a potentially causal relation. This finding aligns with previous research in which we demonstrated that two components of negative emotionality, alienation and aggression, were the aspects of personality that were the most strongly associated with the genetic risk for disordered gambling (Slutske, Cho, Piasecki, & Martin, 2013) and a prospective study linking disordered gambling in adulthood to behavioral observations of being willful, emotionally labile, and negativistic as a 3-year-old child (Slutske, Moffitt, Poulton, & Caspi, 2012). The link between childhood ODD and adult gambling behavior clarifies the relation between negative emotionality and disordered gambling in adulthood, suggesting that it may not be completely a consequence of gambling difficulties contributing to negative emotionality. Rather, having a negative disposition as a child may set the stage for later gambling involvement and difficulties.

Limitations

The study has a number of limitations. First, age of gambling initiation and childhood histories of ADHD, ODD, and CD were based on retrospective reports. Although we demonstrated adequate test–retest reliabilities, obtaining contemporaneous assessments within the context of a prospective study would have been preferable. Second, the age of gambling onset was based on the onset of any form of gambling, and different results may have been obtained if specific potentially high-risk gambling activities had been the focus. Third, the outcomes of the first gambling activities were not taken into account. There is some evidence that large wins early in the gambling career are relevant in shaping future gambling activities (Sharpe, 2002). Fourth, the majority of the participants were Caucasians of Northern European ancestry, so it is not clear the extent to which these results will apply to other racial groups. Fifth, it is unclear how the results of this Australian twin study will generalize to other countries. Sixth, an implicit assumption of the analytic design is that the initial exposures to gambling were equivalent among discordant and concordant twin pairs. There are a number of plausible scenarios in which this assumption may not be correct. For instance, the earlier-gambling twin from a discordant pair may have been introduced to gambling by a boyfriend, an earlier-gambling pair of twins may have been introduced to gambling by their parents. The former may have a more enduring effect and influence later gambling behavior if the boyfriend becomes the spouse, whereas the latter may have a more transitory effect as the twins establish their independence away from their parents.

Conclusions

A number of initiatives have been proposed to prevent disordered gambling through educational or policy initiatives (Williams, Simpson, & West, 2007). For example, it is generally thought that delaying the initiation of gambling among youth, either through educational programs warning young people about the dangers of gambling or by legally restricting access to gambling, will reduce the number of individuals who develop a gambling disorder. This assumption is premised on a causal theory of the association between the age of gambling initiation and disor-

dered gambling—a theory that has never before been empirically evaluated.

The results of this study suggest that universal initiatives to delay the onset of gambling to reduce the numbers of individuals who develop gambling problems may not be effective. They may not be effective because there appears to be a much stronger impact of between-family differences linking the earlier uptake of gambling to later frequent and problematic gambling. This is not to say that preventing young people from gambling is misguided—it is likely to have many benefits. The purpose of this investigation is to draw attention to the possibility that it may not have the intended consequence of reducing the numbers in the population afflicted with a gambling disorder.

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