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Local Area Disadvantage and Gambling Involvement and Disorder: Evidence for Gene-Environment Correlation and Interaction

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Previous research has demonstrated that local area characteristics (such as disadvantage and gambling outlet density) and genetic risk factors are associated with gambling involvement and disordered gambling. These 2 lines of research were brought together in the present study by examining the extent to which genetic contributions to individual differences in gambling involvement and disorder contributed to being exposed to, and were also accentuated by, local area disadvantage. Participants were members of the national community-based Australian Twin Registry who completed a telephone interview in which the past-year frequency of gambling and symptoms of disordered gambling were assessed. Indicators of local area disadvantage were based on census data matched to the participants' postal codes. Univariate biometric model-fitting revealed that exposure to area disadvantage was partially explained by genetic factors. Bivariate biometric model-fitting was conducted to examine the evidence for gene-environment interaction while accounting for gene-environment correlation. These analyses demonstrated that: (a) a small portion of the genetic propensity to gamble was explained by moving to or remaining in a disadvantaged area, and (b) the remaining genetic and unique environmental variation in the frequency of participating in electronic machine gambling (among men and women) and symptoms of disordered gambling (among women) was greater in more disadvantaged localities. As the gambling industry continues to grow, it will be important to take into account the multiple contexts in which problematic gambling behavior can emerge—from genes to geography—as well as the ways in which such contexts may interact with each other.

General Scientific Summary

Previous studies have shown that there are genetic risk factors that increase the chance of developing a gambling problem. This study found that where one lives, such as in a disadvantaged neighborhood or close to a gambling venue, also increases the chances of developing a gambling problem. These 2 types of risk factors can work together to increase risk for gambling problems. For example, some of the same genetic risk that is related to developing a gambling problem is also associated with living in a high risk environment, such as in a disadvantaged neighborhood or close to a gambling site.

Keywords: gene-environment interaction, gene-environment correlation, neighborhood disadvantage, gambling, disordered gambling, natural experiment

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There is marked international (Pryor, 2008; Volberg & Williams, 2013) and intranational (Productivity Commission, 2010; Volberg, 2001) variation in the level of gambling involvement and the percentage of people who develop gambling-related problems. Research is relatively consistent in demonstrating that local exposure to gambling venues is associated with higher rates of gambling and gambling-

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related problems (Abbott, 2007; Vasiliadis, Jackson, Christensen, & Francis, 2013), suggesting that some of these differences might be explained by differential access to gambling opportunities. For example, in a national United States survey, living within 10 miles of a casino, compared with not living near a casino, was associated with more than double the rate of disordered gambling in the past year (Welte, Wieczorek, Barnes, Tidwell, & Hoffman, 2004).

Local Area Disadvantage and Gambling Involvement and Disorder

In addition to differential access to gambling opportunities, differences in contextual factors such as area disadvantage may explain regional variation in gambling behaviors (Barnes et al., 2013; Martins et al., 2013; Pearce et al., 2008; Welte et al., 2004). Studies conducted in the United States (Barnes et al., 2013; Welte et al., 2004) and New Zealand (Pearce et al., 2008) used national survey data coupled with census information to examine the relation between neighborhood disadvantage and gambling involvement and problems. In both studies, census indicators such as the proportion of households in the area in which the adults were living on public assistance, were unemployed, or were of low income were combined to characterize the relative neighborhood disadvantage of the participants. In the United States study, for example, each standard deviation increment in neighborhood disadvantage was associated with eight additional gambling occasions and a 69% increased odds of exhibiting problem gambling in the past year (Welte et al., 2004)—similar results were obtained in the New Zealand study.

Despite the empirical evidence, there have not been any theories advanced that specifically focus on the role that local area disadvantage plays in gambling involvement and disorder. One potential explanation is that local area disadvantage is related to the availability of gambling opportunities. In the United States (Welte et al., 2004), New Zealand (Pearce et al., 2008), Canada (Wilson et al., 2006), England (Wardle et al., 2014), and Australia (Marshall & Baker, 2002; Productivity Commission, 1999), there is a greater density of gambling outlets in relatively disadvantaged neighborhoods. There is consistent evidence that access to electronic gambling machines (variously known as slot machines, pokies, fruit machines, or VLTs in different countries), in particular, is greater in disadvantaged than in more advantaged neighborhoods (Marshall & Baker, 2002; Pearce et al., 2008; Productivity Commission, 1999; Wardle et al., 2014; Wilson et al., 2006). Therefore, one way that local area disadvantage may be related to gambling involvement and disorder is via increased access to gambling opportunities and the social contagion (Christakis & Fowler, 2013) of living in an area surrounded by individuals who routinely gamble.

Gene-Environment Interaction

When examining the relations between contextual factors such as gambling availability or local area quality and gambling behavior, the role of individual vulnerabilities are rarely considered. An exception is a study of young adults in which the personality trait of impulsivity was significantly associated with an earlier age of gambling initiation among individuals from relatively deprived neighborhoods, but not among those from relatively advantaged neighborhoods (Auger et al., 2010). This study illustrates a person-environment interaction whereby a personal vulnerability is only expressed within a particular environmental context, or conversely, a particular environmental context only impacts on individuals who possess a personal vulnerability. With genetically informative data, such as data collected from pairs of monozygotic and dizygotic twins, one can move beyond person-environment interactions; the personal vulnerability can be parsed into genetic and environmental components and one can examine potential *gene-environment interactions* (Dick, 2011; Manuck & McCaffery, 2014; Shanahan & Hofer, 2005). For example, in conditions that are more facilitative, such as having easier access to gambling opportunities, genetic predispositions to gamble may be more likely to be actualized. Similarly, in conditions in which there are limited social controls (Sampson & Groves, 1989; Sampson, Morenoff, & Gannon-Rowley, 2002) or chronic stress (Agnew, 1992; Boardman, 2004; Steptoe & Feldman, 2001), such as in relatively impoverished neighborhoods, genetic predispositions to gamble may also be more likely to be expressed. Both scenarios predict that the heritability of gambling behavior will be higher in contexts that are more facilitative or lower in social controls, compared with contexts that are less facilitative or higher in social controls.

The behavioral genetic research on gambling has not yet progressed to examining gene-environment interaction (or gene-environment correlation), but there are two noteworthy examples from the substance use literature. Among 18.5-year-old Finnish twins, the heritability of alcohol consumption was significantly higher among those residing in urban than in rural areas, and this appeared to be explained in part by the relative proportions of young people living in these areas (Dick, Rose, Viken, Kaprio, & Koskenvuo, 2001). Another example comes from a large national U.S. sample of 12- to 21-year-old twins and siblings in which the contribution of genetic and environmental influences to regular smoking varied as a function of the state of residence (Boardman, 2009). This appeared to be explained in part by state-to-state variation in cigarette taxes and restrictions on the locations of cigarette vending machines and billboards, with the heritability of regular smoking lower in states with more restrictions. In sum, living in an urban region appeared to facilitate the expression of the genetic propensity to drink via exposure to more same-age peers among Finnish young adults, and state-level social controls appeared to dampen the genetic propensity to smoke among U.S. adolescents and young adults.

Gene-Environment Correlation

A critical issue when studying gene-environment interactions is accounting for *gene-environment correlation* (Duncan et al., 2014; Eaves, Silberg, & Erkanli, 2003; Kendler, 2011). A gene-environment correlation occurs when one's genetic make-up influences exposure to a high-risk environment (Kendler & Eaves, 1986; Rutter, 2006), such as a disadvantaged neighborhood. It is important to account for gene-environment correlation because, for example, the overrepresentation of individuals at higher genetic risk for disordered gambling in more disadvantaged areas can lead to results that mimic the effects of a gene-environment interaction. To our knowledge, there are no examples of studies of gene-environment correlation in which the high-risk environment was a

social context such as area of residence. There is an extensive sociological literature, however, describing the importance of the phenomenon of selection bias in the study of neighborhood effects (Goodnight et al., 2012; Leventhal & Brooks-Gunn, 2000; Sampson et al., 2002), where it is recognized that where one lives is in part determined by one's own characteristics. The previous gene-environment interaction investigations of Dick, Rose, Viken, Kaprio, and Koskenvuo (2001) and Boardman (2009) did not examine gene-environment correlation; data from twin pairs that were living in different municipalities (Dick et al., 2001) or states (Boardman, 2009) were excluded. In order to detect active gene-environment correlations (Scarr & McCartney, 1983), one must have data on twins that have "actively" selected different environments.

The Present Study

The present study was an examination in a large Australian twin sample of the extent to which genetic contributions to individual differences in gambling involvement and disorder contributed to being exposed to, and were also accentuated by, local area disadvantage. Australia provides a unique setting within which to explore these questions because it is characterized by generally high rates of gambling but with considerable regional variation. Based on the sociological research implicating selection effects on neighborhood of residence, we hypothesized that there would be a genetic influence on living in a disadvantaged area that would overlap with the genetic influences contributing to individual differences in gambling involvement and disorder (i.e., an active gene-environment correlation). Given the geographic association between the density of gambling outlets and relative local area disadvantage we hypothesized that genetic influences on gambling involvement and disorder would be more pronounced in regions characterized by greater disadvantage (i.e., a gene-environment interaction).

Method

Participants

Participants were selected from a sample of 4,764 members of the Australian Twin Registry Cohort II (mean age = 37.7 years). Although the participants were somewhat more advantaged than the general population of Australia, they represented a broad cross-section. For example, the sample included individuals without postsecondary education (38.0% [vs. 52.0%; Callender, Schofield, Shrestha, & Kelly, 2012]), who were unemployed (2.3% [vs. 5.2%; Pink, 2009]), and who were on public assistance (5.7% [vs. 7.3%; Pech & Landt, 2001]). (For more information about the participants, see Slutske et al., 2009.) Respondents completed a structured psychiatric telephone interview conducted in 2004–2007 (response rate = 80.4%) during which gambling behaviors were assessed. Of the 4,764 participants, 2.1% (n = 101) were lifetime abstainers from gambling and an additional 2.0% (n = 93) had been living overseas in the past year. These individuals were not included in this study, leaving a final sample size of 4,570 (1,957 men, 2,613 women). There were data available for 827 complete monozygotic (MZ) and 966 dizygotic (DZ) twin pairs and from 294 unpaired MZ and 690 unpaired DZ twins.

Procedure

Interviews were conducted by trained lay-interviewers who were blind to the status of the cotwin. 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

Local area disadvantage. Local area socioeconomic disadvantage was obtained by matching the participants' postal codes to information provided by the Australian Bureau of Statistics using data from the 2006 Australian census (Pink, 2008a).[1] Out of 2,515 postal codes in the 2006 Australian census, there were 1,339 different postal codes represented in the sample, with up to 27 participants residing in each (M = 3.41, SD = 3.36); 79% of the participants resided in a city and 21% in a rural area. Thirty-six percent of the postal code areas included a single participant, and 75% included fewer than five participants.[2] The majority of twins and cotwins (84%) resided in a different postal code area.

An index of relative socioeconomic disadvantage (IRSD) created by the Australian Bureau of Statistics was used (Pink, 2008a, 2008b). This index has been used in its present form since 1986, and is updated every five years subsequent to each new Australian census. The construct of relative socioeconomic disadvantage was defined in terms of "people's access to material and social resources, and their ability to participate in society" (Pink, 2008a, p. 17). It has been used extensively in Australian public health research and is also routinely used in other published research (e.g., Bayer et al., 2011; Laslett et al., 2012).

The IRSD is based on the results of a principal components analysis of 17 census-derived indicators and takes into account the proportion of households in the specified geographic area characterized by (e.g.) low income, low educational attainment, unskilled employment or unemployment, single-parenthood, subsidized living or low rent housing (see Appendix A for the full list of indicators and their factor loadings; see Supplemental Materials for more information about the development and validity of the IRSD.). The IRSD index was reversed so that higher scores reflected greater local area disadvantage. The postal codes represented in this sample were slightly less disadvantaged on average (d = −0.25) than the Australian population. Although the entire range of area disadvantage was represented, postal codes in the top three deciles were underrepresented, and in the bottom three deciles of disadvantage were overrepresented (See Table S1 in Supplemental Materials). Local area disadvantage was significantly associated with personal household income, r = −.25, p < .0001 and with rural (vs. urban) residence, r = .27, p < .0001.

[1] The measure of local area disadvantage was extremely stable over a span of five years. The same results were obtained when data from the 2001 census were used. The indexes of local area socioeconomic disadvantage from the 2001 and 2006 censuses were correlated r = .94 and the difference for each participant was only d = 0.01 when using the disadvantage score obtained in 2001 versus 2006.

[2] The average population size of the postal code areas was 13,180 (SD = 11,944); 48% of the postal code areas had a population size of less than 10,000, and only 2% had a population size of greater than 50,000.

Gambling. For each of 11 different gambling activities, 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*. Following the questions about specific gambling activities, participants were asked how many days they had participated in any gambling activity, and where they were when they gambled in the last 12 months.

Two different indexes of gambling involvement were examined in the present study: the frequency of electronic gambling machine (EGM) and any gambling participation in the past year. We elected to focus on the specific activity of electronic gambling machines because this form of gambling accounts for the majority of gambling expenditures in Australia (Delfabbro, 2012),[3] has been linked to neighborhood disadvantage (Marshall & Baker, 2002), is considered by many to be an especially virulent form of gambling (e.g., Fiorito, 2006), and has been associated with progression to disordered gambling (Hodgins et al., 2012; Slutske et al., 2015). Among the participants in this study, 45% and 89% had gambled at least once on an electronic gambling machine or on any gambling activity in the past year, respectively. The gambling outcomes were log-transformed prior to conducting analyses to minimize the skewness and kurtosis of their distributions (any gambling: skewness = −0.09, kurtosis = −1.03; electronic machine gambling: skewness = 1.50, kurtosis = 1.57).

Disordered gambling. Past-year symptoms of disordered gambling were assessed using the National Opinion Research Center *DSM* Screen for Gambling Problems (Gerstein et al., 1999) and the South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987). The nine *DSM–5* disordered gambling symptoms (American Psychiatric Association, 2013), 15 items from the SOGS (five SOGS items had to be omitted because they were endorsed by two or fewer participants), and an additional item related to the versatility of gambling involvement (a count of the number of different gambling activities in the past year) were combined into a disordered gambling measure by extracting a single common factor from a confirmatory factor analysis using the Mplus software program (Muthén and Muthén, 1998-2004). Mplus is especially well-suited for this purpose because it allows for the proper factor analysis of dichotomous data (by using tetrachoric and biserial correlations). A similar approach has previously been used to create a lifetime disordered gambling factor (Lind et al., 2013; Slutske et al., 2013b).

The results of the factor analysis are presented in Appendix B (Additional details can be found in the Supplemental Materials). The factor loadings represent the strength of the association between the item and the latent disordered gambling factor. The factor loadings for all nine of the *DSM–5* symptoms were uniformly high ($\lambda = 0.86$ to 0.95). The factor loadings for the 15 SOGS items were also high ($\lambda = 0.59$ to 0.93). An important point to bear in mind is that the estimated factor score was weighted by the factor loadings so that better items contributed more to the factor score than did the weaker items.

The item thresholds (also shown in Appendix B) are required whenever categorical items are factor analyzed. The item thresholds can be interpreted in the same way as item difficulties are interpreted in an IRT analysis. The metric of the item thresholds are in *z*-score units from the z (standard normal) distribution. Higher thresholds indicate that the item is more "difficult" and lower thresholds indicate that the item is less "difficult." The thresholds for all nine of the *DSM–5* symptoms were uniformly high ($\tau = 1.93$ to 2.60). The thresholds for the 15 SOGS items were more variable ($\tau = 1.03$ to 3.02). For the development of a quantitative trait, it is desirable to have items that represent a broad range of difficulties because this will provide better measurement of the trait across the full continuum and will also lead to a latent trait that more closely approximates a normal distribution. By supplementing the symptoms from the *DSM–5* with items from the SOGS and with the gambling versatility index we were able to improve the psychometric properties of our disordered gambling latent factor above what could have been achieved using the *DSM–5* symptoms alone.

The different measures of disordered gambling were combined to create a variable more amenable to biometric modeling, especially the investigation of gene-environment interactions (see Slutske et al., 2011). A recent simulation study has demonstrated that the use of measures that are not normally distributed can lead to falsely detecting a gene-environment interaction when it does not exist (Van Hulle & Rathouz, 2015). The use of a *DSM–5* disordered gambling symptom count was inadequate because only 5% of the participants had a nonzero score and even after log transformation the distribution was extremely skewed (5.25) and kurtotic (29.69). In contrast, the disordered gambling factor score more closely approximated a normal distribution (skewness = 1.30, kurtosis = 1.49).

The disordered gambling factor evidenced construct validity. As expected, it was significantly correlated with a *DSM–5* disordered gambling symptom count ($r = .59$). It was also significantly associated with the frequency of any gambling ($r = .59$) and electronic machine gambling ($r = .66$), and the amount of money ($r = .55$) and time ($r = .41$) spent in a typical day of gambling in the past year. The disordered gambling factor score was used in all of the analyses; past-year diagnoses of *DSM–5* disordered gambling are presented for descriptive purposes only.

Data Analysis

Two types of analyses were conducted. The first set of analyses examined the association between the three past-year indexes of gambling involvement (frequency of any gambling, electronic machine gambling, and disordered gambling) and local area disadvantage at the individual level using linear regression. The data were treated as clustered, with the family unit (twin pair) and the geographic unit (postal code) serving as the clusters. Analyses were conducted using SAS survey data analysis procedures (SAS Institute, Inc., 2013).

The second set of analysis was conducted at the level of the twin pair using the Mplus program (Muthén & Muthén, 2010). The analyses were conducted in three steps. In Step 1, univariate biometric model-fitting was conducted to partition the *variation* in local area disadvantage and the three gambling outcomes, considered individually, into additive genetic, shared environmental, and unique environmental influences (Neale & Cardon, 1992). The partitioning of variation in local area disadvantage provided a clue to the processes

[3] There are about 198,000 electronic gambling machines in Australia, about 6% are found in casinos, and the remaining 94% are in clubs and pubs. All of the eight states and territories in Australia have at least one casino (13 casinos in total), and all but one has non-casino electronic gambling machines in 5,683 different non-casino venues (Productivity Commission, 2010).

involved in selection into a disadvantaged area of residence. Of particular interest in this study was the overlap in the genetic and environmental sources of variation associated with selection into a disadvantaged area of residence with the sources of variation associated with the gambling outcomes (described in the following paragraph).

In Step 2, bivariate Cholesky models were fit to partition the *covariation* between local area disadvantage and each of the three gambling outcomes into additive genetic, shared environmental, and unique environmental influences in order to estimate genetic and environmental correlations (Neale & Cardon, 1992). Figure 1 illustrates how the Cholesky model was used to estimate the genetic (paths $ad^{*}ac$) and unique environmental (paths $ed^{*}ec$) covariation between local area disadvantage and a gambling outcome. The gene-environment correlation, for example, between local area disadvantage and a gambling outcome was paths $ad^{*}ac$ divided by the square root of the total genetic variation for local area disadvantage (path *ad* squared) times the total genetic variation for the gambling outcome (paths $ac + au$ squared). This is an estimate of the correlation between the genetic factors associated with selection into a disadvantaged area of residence with the genetic factors associated with a gambling outcome.

The Cholesky model also allowed for the separation of the genetic and environmental influences on the gambling outcome that were shared with local area disadvantage (paths *ac* and *ec*, respectively) from the genetic and environmental influences that were unique to the gambling outcome (paths *au* and *eu*, respectively). The former two paths included the gene-environment correlation, whereas the latter two paths are of particular interest within the context of a gene-environment interaction. The evaluation of gene-environment interaction was based on genetic and environmental influences on the gambling outcome that were not confounded with genetic and environmental contributions to exposure to local area disadvantage.

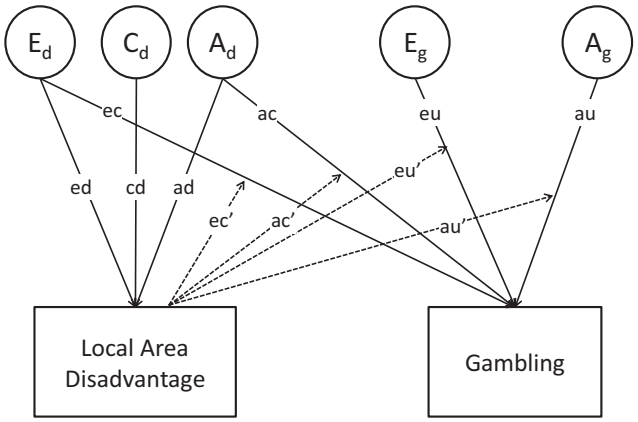

Figure 1. Bivariate Cholesky model (solid paths) with interaction terms (dashed paths) used to partition the genetic (Ad), shared environmental (Cd), and unique environmental (Ed) variation in local area disadvantage, and the genetic (Ag) and unique environmental (Eg) variation in gambling (any gambling, electronic machine gambling, or disordered gambling), into portions that are common to local area disadvantage and gambling (ac, ec) and unique (au, eu) to gambling. The dashed paths au' and eu' are interpreted for each individual as (local area disadvantage score)*au and (local area disadvantage score)*eu. The covariation between local area disadvantage and gambling explained by genetic ($ad^{*}ac$) and unique environmental influences ($ed^{*}ec$) is also estimated in this model. To simplify presentation, the model for only one twin from a pair is shown.

In Step 3, the bivariate Cholesky models were modified to include gene-environment interactions and environment-environment interactions (Purcell, 2002; dashed paths *ac*', *ec*', *au*', and *eu*' in Figure 1). When the results of Step 3 indicated that there was a significant gene-environment or environment-environment interaction, an alternate model (Rathouz et al., 2008; Van Hulle, Lahey, & Rathouz, 2013) was fit to probe the interaction (shown in Figure S1 in Supplemental Materials).

In the univariate biometric model-fitting, data from unlike-sex and same-sex twin pairs were included in order to examine sex differences. For the remaining bivariate models that included local area disadvantage and a gambling outcome, the analyses were restricted to data from same-sex twins. This was because it was not clear that the processes linking local area disadvantage to the genetic and environmental propensity to gamble or to develop gambling problems would be the same in men and women.

Results

Individual-level Analyses

Associations between local area disadvantage and gambling involvement and disorder. Local area disadvantage was significantly associated with the frequency of any form of gambling, $r = .10$; $t = 6.25$, $p < .0001$, playing electronic gambling machines, $r = .16$; $t = 9.62$, $p < .0001$, and disordered gambling ($r = .10$; $t = 6.47$, $p < .0001$; see Figure 2); note that both the standard deviations as well as the means increased with increasing local area disadvantage (See Table S4 in Supplemental Materials). Figure 2 shows that the overall prevalence of a past-year disordered gambling diagnosis was very low, at 1%, but was nearly eight times higher among those living in the highest decile of local area disadvantage (3.3%) compared with those living in the lowest decile (0.4). The associations between local area disadvantage and gambling involvement and disorder did not significantly differ for men and women, $t = 0.45$ to 1.12, $p = .26$ to $.65$. There were not significant nonlinear associations between local area disadvantage and the frequency of any gambling, $t = -1.93$, $p = .054$; playing electronic gambling machines, $t = 0.58$, $p = .56$; or experiencing disordered gambling, $t = 0.60$, $p = .55$.

Personal household income was not significantly associated with the frequency of any form of gambling ($r = -.03$; $t = -1.58$, $p = .11$), but was inversely associated with the frequency of playing electronic gambling machines ($r = -.07$; $t = -3.69$, $p = .0002$), and disordered gambling ($r = -.05$; $t = -2.68$, $p = .007$). After controlling for personal household income, however, local area disadvantage was still significantly associated with the frequency of any form of gambling, $t = 5.79$, $p < .0001$; playing electronic gambling machines, $t = 8.81$, $p < .0001$; and symptoms of disordered gambling, $t = 5.97$, $p < .0001$.

Among nine different possible locations, only gambling at a hotel or pub (odds ratio [OR] = 1.09; $\chi^2 = 53.27$, $df = 1$, $p < .0001$) or a club (OR = 1.08; $\chi^2 = 32.02$, $df = 1$, $p < .0001$) were significantly associated with local area disadvantage; each decile of disadvantage was associated with an 8%–9% increase in the

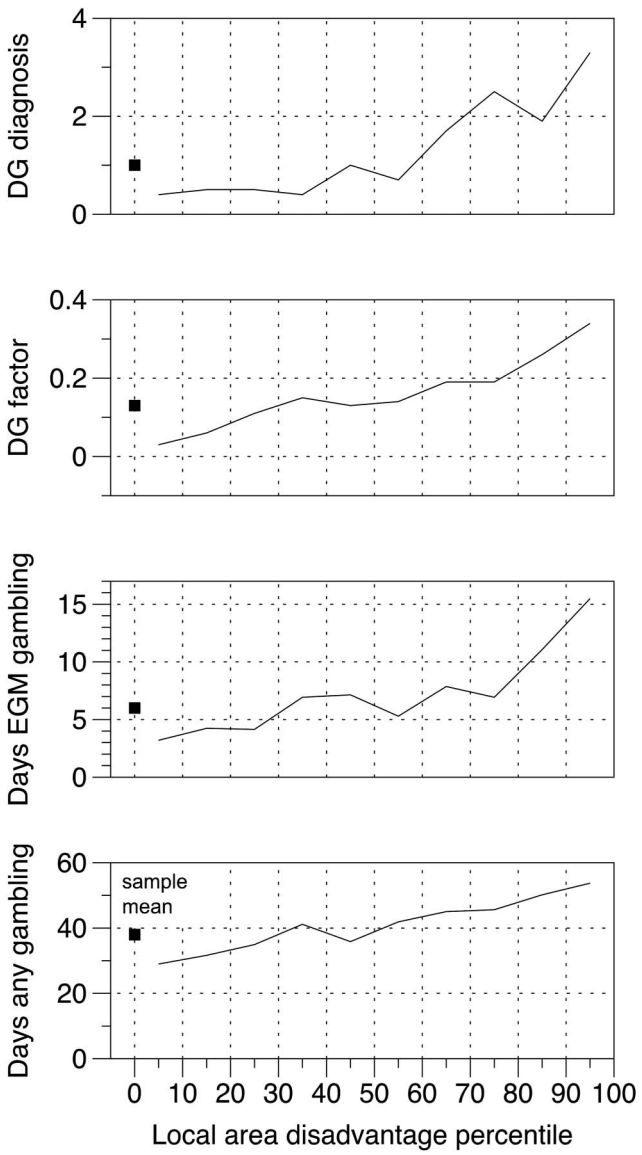

Figure 2. Mean number of days gambled and disordered gambling in the past year by level of local area disadvantage. Values in the top panel (DG diagnosis) represent percentages, values in the remaining three panels represent means. In each panel, the solid square represents the overall sample percentage (top panel) or mean (bottom three panels). DG = disordered gambling, EGM = electronic machine gambling. See Supplemental Material Table S3 for the numerical values for the means and the standard deviations.

odds of gambling at a hotel/pub or club. Note that hotel/pubs and clubs are the main venues in which electronic machine gambling occurs in Australia.

Twin-Level Analyses

Genetic and environmental contributions to local area disadvantage and gambling involvement and disorder (Step 1). Inspection of twin correlations provided preliminary information about the contribution of genetic and environmental influences to individual differences in the three gambling outcomes and local area disadvantage (upper portion of Table 1). The results of univariate biometric model-fitting indicated that the estimates of shared environmental effects were not significantly greater than zero, and dropping shared environmental influences did not significantly worsen model fit for variation in the past-year frequency of any gambling ($\Delta\chi^2 = 2.14$, $df = 2$, $p = .34$). There were also no sex differences. In the model that dropped shared environmental influences and equated parameter estimates for men and women, genetic and unique environmental factors explained 47% and 53% of the variation in the frequency of any gambling.

For the past-year frequency of playing electronic gambling machines, the estimates of shared environmental effects were significantly greater than zero among women but not among men. A reduced model that dropped shared environmental influences significantly worsened model fit ($\Delta\chi^2 = 11.52$, $df = 2$, $p = .003$). There was evidence for sex differences in the contributions of the three sources of variation ($\Delta\chi^2 = 90.95$, $df = 3$, $p < .0001$); individual differences in the frequency of electronic machine gambling was best explained by genetic (55%) and unique environmental (45%) factors among men, and by genetic (25%), shared environmental (31%), and unique environmental (43%) factors among women (see Table 2).

For past-year disordered gambling, dropping shared environmental influences did not significantly worsen model fit ($\Delta\chi^2 = 0.80$, $df = 2$, $p = .67$), and there was evidence for sex differences in the proportion of variation attributed to the three sources of variation ($\Delta\chi^2 = 77.58$, $df = 3$, $p < .0001$). In the model that dropped shared environmental influences, genetic and unique environmental factors explained 45% and 55% of the variation in past-year disordered gambling among men, and 42% and 58% of the variation in past-year disordered gambling among women (see Table 2).

For variation in local area disadvantage, neither genetic ($\Delta\chi^2 = 8.66$, $df = 2$, $p = .01$) nor shared environmental influences ($\Delta\chi^2 = 6.46$, $df = 2$, $p = .04$) could be dropped without significantly worsening model fit. There was no evidence for sex differences; in the model that equated parameter estimates for men and women, genetic, shared environmental, and unique environmental factors explained 25%, 20%, and 55% of the variation in local area disadvantage, respectively. Individual differences in selection into a disadvantaged living area was due in nearly equal parts to familial factors (both genetic and environmental) and factors unique to the individual.

Overlap of genetic and environmental contributions to local area disadvantage, gambling, and disordered gambling: Evidence for gene-environment correlation (Step 2). The significant cross-trait twin correlations between local area disadvantage and the three gambling outcomes implicated familial factors contributing to their associations (lower portion of Table 1). The observation that the MZ correlations were nearly as large as the within-twin correlations in most cases suggests that the associations, although modest, were almost completely explained by familial factors. The smaller unlike-sex than same-sex DZ twin cross-trait correlations (especially for electronic machine gambling) supported the concern that the processes linking local area disadvantage to the genetic and environmental propensity to gamble or to develop gambling problems might not be the same in men

Table 1
Twin Correlations for Past-Year Gambling Involvement and Disorder and Local Area Disadvantage

	Monozygotic		Dizygotic		
	Men (N = 319 pairs)	Women (N = 479 pairs)	Men (N = 211 pairs)	Women (N = 339 pairs)	Opposite sex (N = 376 pairs)
	Within-trait correlations				
Any gambling	**.50**	**.45**	**.27**	**.31**	**.16**
Electronic gambling machines	**.55**	**.59**	**.20**	**.45**	**.25**
Disordered gambling	**.42**	**.42**	**.23**	**.25**	**.16**
Local area disadvantage	**.47**	**.45**	**.30**	**.33**	**.22**
	Cross-trait correlations between past-year gambling and local area disadvantage[a]				
Any gambling	**.09**	**.08**	.08	.04	.06/−.11[b]
Electronic gambling machines	**.14**	**.14**	**.14**	**.11**	.08/−.02[b]
Disordered gambling	.01	**.09**	.06	.07	.05/−.03[b]

Note. Correlations in bold are significantly greater than zero at $p < .05$. Sample sizes represent the number of complete twin pairs, individual twins from 1,120 incomplete pairs were also included in analyses.
[a] The within-twin correlations of any gambling, electronic machine gambling, and disordered gambling with local area disadvantage were $r = .11$, $r = .16$, and $r = .10$, respectively, among men, and were $r = .09$, $r = .15$, and $r = .11$, respectively, among women. [b] First correlation is female twin gambling with male twin local area disadvantage, the second is male twin gambling with female twin local area disadvantage.

and women and the decision to conduct separate analyses for men and women in the bivariate models.

Bivariate biometric models were fitted to the twin data to decompose the covariation between local area disadvantage and the three past-year gambling outcomes, considered individually, into genetic and environmental components (Figure 1 without the dashed paths). Based on the results of the univariate models, all three sources of variation (genetic, shared environment, and unique environment) were included to explain variation in local area disadvantage in all of the bivariate models. For the three gambling outcomes, genetic and unique environmental factors were included to explain variation. (Although there was evidence for significant shared environmental contributions to variation in electronic machine gambling among women, a Cholesky with gene-environment interaction model that included all three sources of variation was computationally impracticable.)

The main results of interest from the bivariate Cholesky models are presented in Table 3. There were significant genetic correlations between local area disadvantage and any gambling and electronic machine gambling for both men and women. There was also a significant unique environmental correlation between local area disadvantage and disordered gambling among men, which might be a clue to a possible causal relation between local area disadvantage and disordered gambling. The results of the Cholesky model-fitting also confirmed that nearly all of the covariation between local area disadvantage with any gambling, electronic machine gambling, and disordered gambling was explained by genetic factors (the latter only among women), and that this shared genetic influence associated with local area disadvantage explained a small proportion of the genetic variation (4%–12%) and none of the unique environmental variation in these gambling outcomes (not shown).

Moderation of genetic and environmental contributions to gambling and disordered gambling by local area disadvantage: Evidence for gene-environment interaction (Step 3). For each of the three gambling outcomes, the fit of the interaction model was

Table 2
Proportion of Variation in Past-Year Gambling Involvement and Disorder and Local Area Disadvantage Explained by Additive (A), Shared Environmental (C), and Unique Environmental (E) Influences

	Men			Women		
Variable	A	C	E	A	C	E
Any gambling	.50	.02	.48	.31	.15	.54
95% CI	[.22, .78]	[.00, .27]	[.40, .55]	[.09, .53]	[.00, .34]	[.47, .61]
Electronic gambling machines	.55	.00	.45	.25	.31	.43
95% CI	[.48, .62]	[.00, .02]	[.38, .52]	[.07, .44]	[.15, .48]	[.38, .49]
Disordered gambling	.41	.04	.56	.32	.09	.59
95% CI	[.10, .71]	[.00, .31]	[.47, .64]	[.08, .56]	[.00, .30]	[.52, .66]
Local area disadvantage	.34	.13	.53	.20	.24	.56
95% CI	[.05, .63]	[.00, .39]	[.45, .61]	[.00, .42]	[.05, .44]	[.49, .63]

Note. A = additive genetic influences; C = shared environmental influences; E = unique environmental influences; CI = confidence interval. The results are stratified by sex to be consistent with the bivariate analyses reported elsewhere.

Table 3
Genetic and Environmental Associations Between Past-Year Gambling Involvement and Disorder With Local Area Disadvantage

	Genetic and environmental correlations				Proportions of covariation explained by genetic and environmental factors			
	Men		Women		Men		Women	
Past-year gambling outcome	r_G	r_E	r_G	r_E	$prop_G$	$prop_E$	$prop_G$	$prop_E$
Any gambling	**.22**	.03	**.25**	.03	**.85**	.15	**.85**	.15
95% CI	[.04, .40]	[−.07, .14]	[.02, .48]	[−.06, .11]	[.38, 1.00]	[.00, .62]	[.37, 1.00]	[.00, .63]
Electronic gambling machines[a]	**.30**	.03	**.35**	.07	**.91**	.09	**.80**	.20
95% CI	[.11, .49]	[−.08, .13]	[.12, .58]	[−.02, .15]	[.56, 1.00]	[.00, .44]	[.56, 1.00]	[.00, .44]
Disordered gambling	.05	**.12**	.21	.07	.24	.76	**.63**	.37
95% CI	[−.13, .23]	[.01, .22]	[−.01, .42]	[−.02. .15]	[.00, 1.00]	[.00, 1.00]	[.18, 1.00]	[.00, .82]

[a] Because there was evidence for significant shared environmental influences for electronic machine gambling among women, the bivariate model was re-run including shared environmental influences and estimating r_G, r_C, and r_E. In this model r_G =.12; r_C =.34, and r_E =.08, and none significantly differed from zero. r_G = correlation between genetic influences; r_C = correlation between shared environmental influences; r_E = correlation between unique environmental influences; $prop_G$ = the proportion of the covariation with local area disadvantage attributable to genetic factors; $prop_E$ = the proportion of the covariation with local area disadvantage attributable to unique environmental factors; CI = confidence interval. Correlations in bold are significantly greater than zero at $p < .05$.

compared with a similar main effects model in which the interaction paths were set to zero (Figure 1 with and without the dashed paths; Table 4). For the past-year frequency of any gambling, there was no evidence for gene-environment interaction in either men or women. There was evidence, however, for significant gene-environment interactions in both men and women for the frequency of electronic machine gambling and for disordered gambling. These results were largely confirmed using an alternate parameterization of the gene-environment interaction effects (See Supplemental Materials). The parameter estimates from the main effects and interaction models are presented in Table 5.

Several findings presented in Table 5 are worth highlighting. First, by comparing the estimates for *ac* and *ec* to *au* and *eu*, it is clear that (with the exception of disordered gambling among men) the majority of the genetic and environmental influences on the gambling outcomes were distinct from genetic and environmental influences contributing to exposure to local area disadvantage. Second, the significance of the *ac* parameter for any gambling and electronic machine gambling for men and women in the interaction models confirmed that the gene-environment correlations uncovered in the main effects models were still observed when gene-environment interactions were included in the model. Third, the parameters for the main effect and interaction models for disordered gambling among men were quite discrepant from each other compared with disordered gambling among women and for the other two gambling outcomes.

The unstandardized estimates of genetic and unique environmental variation in electronic machine gambling at different levels of local area disadvantage are presented in Figure 3. Both genetic and unique environmental variation were larger with increasing levels of local area disadvantage. Among men, the estimates of genetic and unique environmental variation were 2.3 and 1.7 times greater, respectively, in the highest quintile than in the lowest quintile of local area disadvantage; among women the estimates of genetic and unique environmental variation were 1.6 and 4.5 times greater, respectively, in the highest quintile than in the lowest quintile of local area disadvantage. Because both genetic and environmental variation were increasing, the standardized estimates did not show the same relations with level of local area disadvantage as the unstandardized estimates. When the estimates were standardized, the heritabilities (the proportion of variation explained by genetic factors) increased slightly for men exposed to the highest versus the lowest level of local area disadvantage, whereas the heritabilities decreased substantially for women exposed to the highest versus the lowest level of local area disadvantage (see Table 6). The unstandardized estimates provide a more straightforward interpretation in this situation (when the variation in the trait is changing with the level of the environmental moderator), whereas the standardized estimates obscure potentially important differences in variances (Young-Wolff et al., 2011).

The unstandardized estimates of genetic and unique environmental variation in disordered gambling at different levels of local area disadvantage among women are presented in Figure 4 (results for men are not included in Figure 4 because the interaction model did not provide a significantly improved fit compared with the main effects model). The results were similar to those for electronic machine gambling in that the estimates of both genetic and unique environmental variation were larger with increasing levels of local area disadvantage. The unstandardized estimates of genetic and environmental variation were 2.1 and 1.8 times greater, respectively, in the highest quintile than in the lowest quintile of local area disadvantage. Because the magnitudes of genetic and environmental variation were increasing nearly equally, the estimate of the heritabilities at the lowest and highest levels of local area disadvantage did not differ much (0.48 vs. 0.53) even though the overall amount of variation in disordered gambling nearly doubled.

Post hoc analyses probing the local area disadvantage effect. In order to potentially disentangle the effects of gambling outlet density[4] and local area disadvantage, we compared the associ-

[4] The density of local gambling venues in each state was calculated from the number of non-casino venues presented in a 2010 report (that used data from a variety of sources on the number of venues in 2008-9; Productivity Commission, 2010) and the 2006 population of each state obtained from the Australian census. Density was the number of venues divided by the population, and then transformed to represent venues/per 10,000 individuals.

Table 4
Model Fits for Bivariate Main Effect and Interaction Models of Past-Year Gambling Involvement and Disorder and Local Area Disadvantage

	Men				Women			
	Model fit (log-likelihood)		Difference test[a]		Model fit (log-likelihood)		Difference test[a]	
Past-year gambling outcome	Main effects model	Interaction model	χ^2	p	Main effects model	Interaction model	χ^2	p
Any gambling	−2732.21	−2730.24	3.94	.41	−4330.03	−4326.87	6.32	.18
Electronic gambling machines	−2814.85	−2804.57	20.56	.0004	−4161.78	−4123.16	77.24	<.0001
Disordered gambling	−2828.96	−2823.10	10.12	.038	−4195.33	−4184.17	22.32	.0002

[a] Likelihood ratio chi-square difference tests have four degrees of freedom. A significant difference in model fit indicates a significant interaction effect.

ations between disadvantage and the three gambling outcomes as a function of the state/territory-level density of local gambling outlets (See Supplemental Materials for more details). Of particular interest was Western Australia, which has *no* local electronic gambling machine venues, compared with the remaining seven states and territories that have on average about three venues for every 10,000 residents. When multilevel regression models were fit using the density of local gambling venues as a continuous Level-2 indicator, there was a significant cross-level interaction between local area disadvantage and the density of local gambling venues in predicting gambling involvement and disorder (any gambling: $t = 4.24$, $df = 4415.30$, $p < .0001$; electronic machine gambling: $t = 3.23$, $df = 4447.68$, $p = .001$; disordered gambling: $t = 3.04$, $df = 3881.62$, $p = .002$). Figure 5 illustrates that the association between local area disadvantage and the three gambling outcomes was stronger in contexts in which there was a greater density of gambling venues. The simple slopes at one standard deviation below and above the mean on local gambling venue density were 1.87 ($p = .0001$) and 2.96 ($p < .0001$) for the frequency of any gambling, were 0.38 ($p = .02$) and 1.18 ($p < .0001$) for the frequency of electronic machine gambling, and were 0.02 ($p = .0193$) and 0.05 ($p < .0001$) for disordered gambling, respectively. The calculation of regions of significance (Preacher, Curran, & Bauer, 2006) revealed that there was a significant association between local area disadvantage and the frequency of any gambling for the entire range of gambling venue densities. The association between local area disadvantage and the frequency of electronic machine gambling was significant for venue densities of greater than 0.32, and the association between local area disadvantage and disordered gambling was significant for venue densities of greater than 0.30. Note that the only state or territory in Australia with a gambling venue density as low 0.32 is Western Australia.

The pivotal role that accessibility to electronic gambling machine venues appeared to play in the local area disadvantage effect prompted the question of whether the association between local area disadvantage and the frequency of any gambling or disordered gambling was driven by the frequency of electronic machine gambling. The overall correlations between local area disadvantage and the frequency of any gambling and disordered gambling were 0.10 and 0.11 before, and were 0.03 and 0.00 after controlling for the frequency of electronic machine gambling, and this did not differ for men and women. This strongly implicates the frequency of electronic machine gambling as the unifying behavior underlying the three seemingly distinct outcomes in the present study and lends additional support to the notion that the density of electronic gambling machine venues is a necessary condition by which area disadvantage contributes to gambling involvement and disorder.

Discussion

Previous research has demonstrated that exposure to area disadvantage (Pearce et al., 2008; Welte et al., 2004) and genetic risk factors (Blanco et al., 2012; Eisen et al., 1998; Slutske et al., 2009; Slutske et al., 2010) are associated with gambling involvement and disordered gambling among adults. The present study weaves together these two strands of research by finding evidence that: (a) the genetic risk for these gambling outcomes is associated with exposure to local area disadvantage, and (b) after controlling for their shared genetic and environmental risk, exposure to area disadvantage exacerbates the genetic risk associated with gambling involvement and disordered gambling (or conversely, that the genetic risk associated with gambling involvement and disordered gambling is associated with greater sensitivity to the deleterious effects of being exposed to living in a disadvantaged area). In other words, the results suggest that the genetic risk for excessive and disordered gambling makes one more likely to be exposed to settings in which there is greater disadvantage, *and* that the genetic risk for excessive and disordered gambling is more likely to be actualized within settings in which there is greater disadvantage.

Genetic Influences on Gambling and Sensitivity to Local Area Disadvantage

Because of the geographic confounding between gambling outlet density and local area disadvantage, it is difficult to tease apart the "active ingredient" involved in the gene-environment interaction observed. An incisive approach for "pulling apart" the effects of two environmental risk factors that usually tend to go together—such as gambling outlet density and local area disadvantage—is via a natural experiment where one has been eliminated (Rutter et al., 2001). A comparison of Western Australia with the remainder of Australia provided such a natural experiment. Western Australia has no local gambling venues whereas the remainder

Table 5
Parameter Estimates From Bivariate Main Effect and Interaction Models of Past-Year Gambling Involvement and Disorder and Local Area Disadvantage

	Men			Women		
	Main effects model	Interaction model		Main effects model	Interaction model	
Parameter	estimate	estimate	90% CI	estimate	estimate	90% CI
			Any gambling			
ac	**.16**	**.17**	[.04, .29]	**.19**	**.21**	[.07, .35]
ec	.02	.01	[−.06, .07]	.01	.03	[−.03, .08]
au	**.69**	**.68**	[.61, .75]	**.66**	**.65**	[.58, .72]
eu	**.69**	**.69**	[.64, .73]	**.71**	**.71**	[.67, .74]
ac'	—	−.06	[−.19, .07]	—	−.11	[−.22, .00]
ec'	—	.06	[−.02, .15]	—	−.01	[−.07, .06]
au'	—	.04	[−.03, .12]	—	.00	[−.07, .07]
eu'	—	.02	[−.04, .07]	—	.04	[−.01, .08]
			Electronic gambling machines			
ac	**.26**	**.26**	[.12, .40]	**.27**	**.28**	[.13, .42]
ec	.03	.02	[−.05, .09]	.04	**.05**	[.01, .10]
au	**.75**	**.74**	[.66, .82]	**.67**	**.66**	[.59, .73]
eu	**.73**	**.73**	[.68, .77]	**.58**	**.57**	[.54, .60]
ac'	—	.02	[−.13, .16]		.04	[−.08, .16]
ec'	—	.01	[−.10, .11]		−.06	[−.12, .01]
au'	—	.09	[.01, 17]		.04	[−.02, .11]
eu'	—	.06	[−.01, .12]		**.15**	[.10, .19]
			Disordered gambling			
ac	.05	.63	[−.01, 1.0]	**.16**	**.16**	[.05, .27]
ec	**.09**	.04	[−.05, .12]	.05	**.07**	[.02, .12]
au	**.69**	.13	[−1.0, 1.0]	**.57**	**.57**	[.51, .62]
eu	**.80**	**.76**	[.70, .81]	**.68**	**.68**	[.64, .71]
ac'	—	.06	[−2.0, 2.1]	—	−.02	[−.12, .08]
ec'	—	.06	[−.06, .19]	—	−.03	[−.09, .03]
au'	—	−.35	[−.71, .02]	—	.06	[.01, .12]
eu'	—	.04	[−.02, .09]	—	.05	[.01, .09]

Note. The parameters listed in the first column correspond to eight of the ten paths depicted in Figure 1 (paths specific to local area disadvantage [ad, cd, and ed] are not shown). CI = confidence interval. Estimates in bold are significantly greater than zero at $p < .05$, underlined estimates are significantly greater than zero at $p < .10$. Ninety-percent confidence intervals are presented for the interaction models to indicate the precision of the estimates.

of Australia has many; there was no association between local area disadvantage and the frequency of electronic machine gambling and disordered gambling in Western Australia, but significant associations for the remainder of Australia. When the state-level density of gambling venues was used in regression analyses as a continuous indicator, there was a significant interaction between gambling venue density and local area disadvantage in predicting the three gambling outcomes. The relation between local area disadvantage and gambling involvement and disorder was stronger in regions in which there was a greater density of gambling venues. This result is consistent with the hypothesis that local area disadvantage has an impact on gambling involvement and disorder primarily within settings where there is also ample availability of electronic gambling machines. Consistent with this were post hoc analyses revealing that the relation between local area disadvantage and disordered gambling (and the frequency of any gambling) was completely explained by the frequency of electronic machine gambling. It has been suggested that residential areas that are high in both disadvantage and electronic gambling machine availability may be gambling vulnerability "hot spots" (Rintoul et al., 2013). Perhaps easier access coupled with area disadvantage was the active ingredient in the gene-environment interaction observed in the present study.

Genetic Influences on Gambling and Exposure to Local Area Disadvantage

Local area disadvantage, along with residential urbanicity (Whitfield et al., 2005) and "walkability" (Duncan et al., 2012), can now be included among the list of environmental exposures that are genetically influenced (Kendler & Baker, 2007).[5] The best-fitting biometric model suggested that genetic factors explained 25% of the variation in exposure to local area disadvantage, with 20% and 55% of the remaining variation explained by shared and unique environmental influences, respectively. This estimate is surprisingly similar to the overall

[5] In contrast to results obtained in Australia (Whitfield et al., 2005), Willemsen et al. (2005) did not obtain evidence for genetic influences on residential urbanicity in the Netherlands.

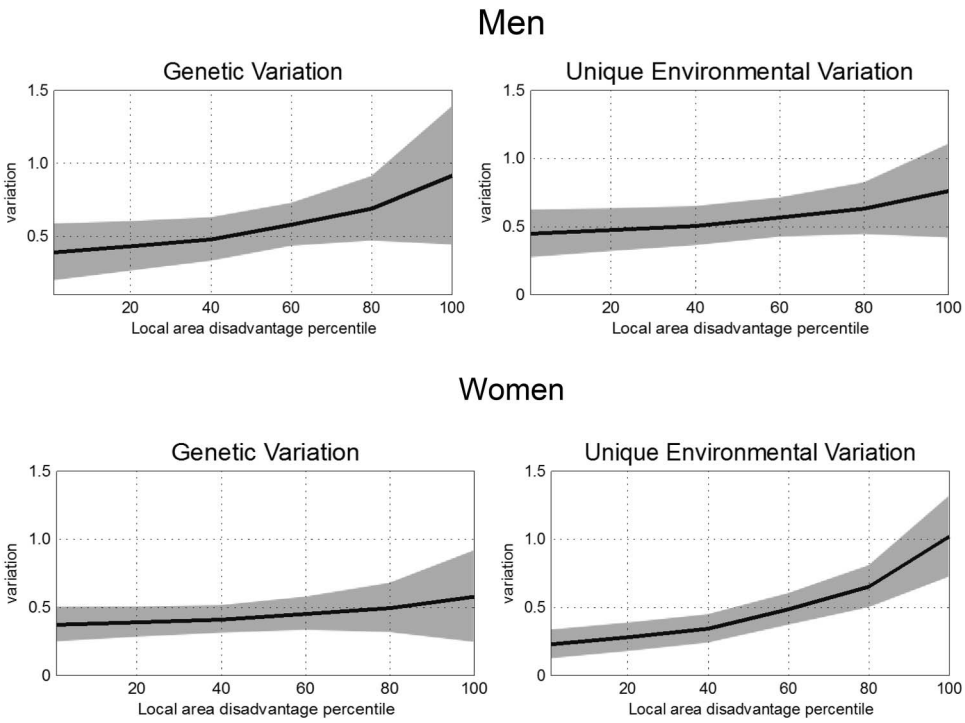

Figure 3. Unstandardized estimates of genetic and unique environmental variation in the frequency of electronic machine gambling at different levels of local area disadvantage. Estimates were derived from the residual portion of unique variation in electronic machine gambling that was unrelated to variation in levels of local area disadvantage (paths *au* and *eu* in Figure 1). Shading represents 95% confidence bands around estimates.

estimate obtained for more proximal measures of the environment such as peer influences, marital quality, and stressful life events (Kendler & Baker, 2007).

Of greater relevance to this study is that the genetic factors associated with exposure to a disadvantaged area were correlated with the genetic factors associated with the frequency of being involved in any gambling activity or electronic machine gambling in the past year. In fact, this gene-environment correlation was the primary explanation for the modest associations observed between local area disadvantage and the frequency of gambling involvement. These results suggest that a small portion of the genetic propensity to gamble is explained by moving to or remaining in a disadvantaged local area. This is either because there are overlapping sets of genes that are associated with both the frequency of gambling and exposure to local area disadvantage, or that exposure to local area disadvantage is a genetically influenced environmental risk factor for the frequency of gambling. A third interpretation, that the frequency of gambling is a genetically influenced environmental risk factor for exposure to local area disadvantage, is less likely because of the temporal precedence of local area disadvantage (measured in 2006, but strongly correlated [r = .94] with disadvantage measured in 2001) over past-year gambling frequency (measured between 2004 and 2007). There was no evidence of a direct causal influence of local area disadvantage on the frequency of gambling—the only evidence consistent with such an effect was for disordered gambling among men.

Table 6
Unstandardized and Standardized Estimates of the Variation in Past-Year Electronic Machine Gambling Explained by Additive Genetic (A) and Unique Environmental (E) Influences at High and Low Levels of Local Area Disadvantage

Level of local area disadvantage	Men		Women	
	A	E	A	E
Unstandardized estimates				
Lowest quintile	0.39	0.45	0.37	0.23
95% CI	[.19, .58]	[.27, .62]	[.24, .50]	[.12, .33]
Highest quintile	0.92	0.76	0.58	1.02
95% CI	[.44, 1.39]	[.42, 1.11]	[.24, .92]	[.72, 1.32]
Standardized estimates				
Lowest quintile	0.47	0.53	0.62	0.38
95% CI	[.31, .62]	[.38, .69]	[.50, .75]	[.25, 0.51]
Highest quintile	0.55	0.45	0.36	0.64
95% CI	[.35, .74]	[.26, .65]	[.21, .51]	[.49, .79]

Note. A = additive genetic influences; E = unique environmental influences; CI = confidence interval.

Limitations

There are at least six major limitations of this study. First, observing gene–environment and environment–environment in-

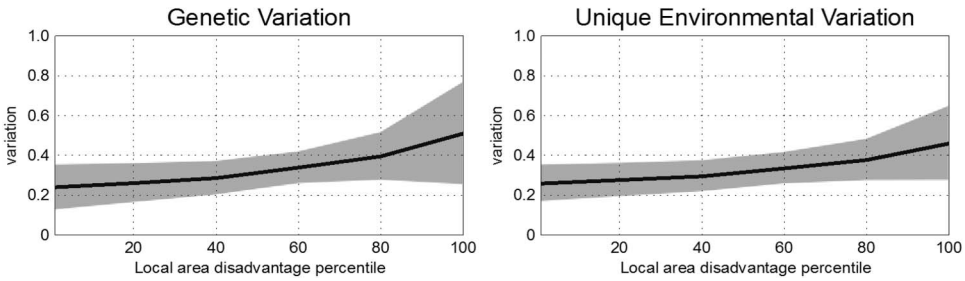

Figure 4. Unstandardized estimates of genetic and unique environmental variation in disordered gambling at different levels of local area disadvantage among women. Estimates were derived from the residual portion of unique variation in disordered gambling that was unrelated to variation in levels of local area disadvantage (paths *au* and *eu* in Figure 1). Shading represents 95% confidence bands around estimates.

teraction jointly has been shown to occur when the measures are skewed or heteroscedastic (Purcell, 2002). In other words, the interactions observed may be more statistical than substantive in nature. Efforts that were taken to minimize this problem included creating a composite disordered gambling factor that was more suited for testing gene-environment interactions and by transforming all of the gambling outcomes to approximate normality.

Second, although it is a common practice to use census-derived geographic entities in neighborhood research, the most commonly used geographic units are the smaller census blocks, block groups, or census tracts, rather than larger postal code areas (Pickett & Pearl, 2001; Sampson et al., 2002). There may have been considerable variability in local area disadvantage within the geographic units demarcated by postal code areas, which may have resulted in an under- or overestimate of the association between local area disadvantage and gambling outcomes. Alternatively, it has been suggested that the correct level of aggregation to use might vary by the specific neighborhood characteristic and the outcome studied; the finding of significant associations between local area disadvantage at the postal-code-area level of aggregation and the gambling outcomes suggest that the effects of disadvantage on the gambling outcomes may have been more diffuse rather than localized (Hipp, 2007). Unfortunately, because it was not possible to assign participants to geographic units that were the Australian equivalent of census blocks, block groups, or census tracts, this could not empirically evaluated.

Third, the use of a global measure of local area disadvantage precluded parsing the local area effect into potentially important components. This limitation is offset by the increased reliability of using a composite index of disadvantage rather than a single indicator (Caspi et al., 2000; South & Crowder, 1999). Fourth, the temporal relation between local area disadvantage and gambling outcomes could not be definitively established because of the cross-sectional design; however, the near-perfect 5-year stability of the disadvantage indicator from 2001 to 2006 suggests that disadvantage preceded the gambling outcomes. Fifth, the participants were middle-aged adults, mostly of Northern European descent. The extent to which these results can be generalized to other ethnic/racial groups or developmental periods is an open question for future research. Sixth, the results of this Australian study may not be applicable to other countries. Most countries have not (yet) had the level of community infiltration of gambling that characterizes much of Australia (Young, 2010).

Conclusions and Future Directions

Despite limitations, this study represents a major step forward. It is the first demonstration of the importance of genetic contributions to living in a disadvantaged local area, the first to examine the association between local area disadvantage and gambling behaviors within a genetically informed study design, and the first demonstration of the role of gene-environment interaction in excessive and disordered gambling. Local area disadvantage was related to the genetic risk for gambling involvement and disorder in two ways: It was a genetically influenced environmental risk factor (via an active gene–environment correlation), and it moderated the genetic risk (via a gene–environment interaction). These results may provide an explanation for failures to obtain replicated findings in the search for susceptibility genes for excessive and disordered gambling (Slutske, 2013) and suggests a way forward, that is, by taking environment into account in the gene search. Similarly, this may provide an explanation for failing to obtain evidence for family environmental influences in standard twin models of risk for disordered gambling (e.g., Blanco et al., 2012; Slutske et al., 2010). When such influences interact with genetic factors, they are subsumed under the "A" component in biometric "ACE" models of twin data.

Building upon the previous research demonstrating that local area disadvantage was associated with an earlier age of initiation of gambling among more impulsive young adults (Auger et al., 2010), future genetically informed studies should examine the influence of local area disadvantage at different stages in the gambling career. This will require studying individuals at different developmental stages, from adolescents first initiating gambling to older pensioners with "disposable time and disposable income" (McNeilly & Burke, 2002, p. 75). Genetically informed longitudinal studies can track participants as they move into or out of disadvantage. They will provide a better understanding of the gene-environment correlation effects by examining (a) how environment may be simultaneously a selection effect (i.e., individuals are genetically predisposed to select themselves into certain environments conducive to gambling behavior) and a causal effect (i.e., environmental influ-

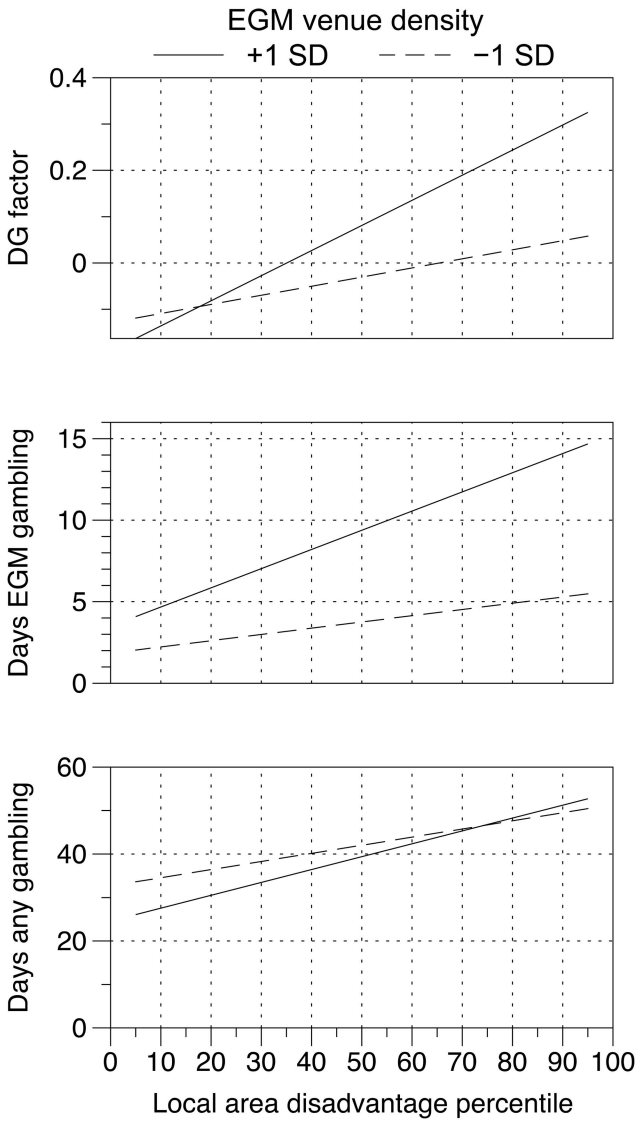

Figure 5. Model-based estimates of the mean past-year frequencies of any gambling and electronic machine gambling and the disordered gambling factor score as a function of local area disadvantage, at one standard deviation above and below the mean of state/territory-level density of local electronic gambling machine venues. Means shown in the Figure for days any gambling and days EGM gambling are based on raw data to facilitate interpretation, analyses reported in the text were conducted using log-transformed variables. DG = disordered gambling; EGM = electronic machine gambling.

ences subsequently lead to initiation or reinforcement of gambling behavior) via a feedback loop or cascade mechanism; and (b) the genetically influenced individual characteristics that may explain selection into and away from a disadvantaged environment.

In addition to studying individuals at different developmental stages, it will be important to better understand potential sex differences in the processes linking local area disadvantage to the genetic and environmental propensity to gamble or to develop gambling problems. In the present study, the significant gene–environment correlation and interaction between local area disadvantage and disordered gambling observed among women was not observed among men; instead there was a significant correlation and interaction between local area disadvantage and the latent sources of unique environmental risk for disordered gambling among men (Results described in the Supplemental Materials). It is not clear what might contribute to this unique environmental risk for disordered gambling among men, but an intriguing clue comes from a previous study in this sample in which there were significant unique environmental correlations between the personality traits of neuroticism and (low) self-control and disordered gambling among men but not among women (Slutske et al., 2013a). Perhaps men higher in neuroticism and lower in self-control are more likely to remain in or move to a disadvantaged area, and are also more vulnerable to the effects of living in such an environment. A logical next step for future research would be to examine whether the personality traits associated with living in a disadvantaged area differ in men and women.

Another priority for future research will be to determine whether these results will generalize to other countries, such as the United States. Although the gambling landscape differs considerably between Australia and the United States, they are similar in that there is also considerable regional variation in the rates of gambling (Welte et al., 2002) and access to gambling opportunities (American Gaming Association, 2013) in the U.S. There are a number of states (Oklahoma, Montana, Louisiana, and of course, Nevada) that have per capita densities of electronic gambling machines that are comparable with those found in Australia. Previous nationally representative research in the United States has demonstrated that neighborhood disadvantage and proximity to a casino are associated with higher rates of problem gambling (Welte et al., 2004), and there is emerging evidence of gambling vulnerability "hot spots," that is, areas close to casinos in which there is greater neighborhood disadvantage (i.e., Philadelphia; Conway, 2012). Whether exposure to such environments in the U.S. are correlated with or moderate the genetic risk for gambling involvement and disorder as they appear to do in Australia awaits genetically informed research based in the U.S.

Finally, more in-depth examinations of specific aspects of disadvantaged areas (e.g., residential instability, shortage of jobs) and geocoding of the proximity and density of gambling venues will help to isolate the active ingredients from the "environment" side of the gene-environment interaction. Future gene-environment interaction studies incorporating measured candidate genes (Slutske, 2013)—rather than latent genetic influences estimated from a twin model—will be required to isolate the active ingredient on the "gene" side of the gene-environment interaction.

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Appendix A

Results of a Principal Components Analysis of the Index of Relative Socioeconomic Disadvantage (IRSD) From the 2006 Australian Bureau of Statistics Socio-Economic Index for Areas (SEIFA)

Census variable, % . . .	Factor loadings
. . . Occupied private dwellings with no Internet connection	0.85
. . . Employed people classified as Labourers	0.76
. . . People aged 15 years and over with no post-school qualifications	0.76
. . . People with stated annual household equivalised income between $13,000 and $20,799 (approx. 2nd and 3rd deciles)	0.76
. . . Households renting from a Government or Community organisation	0.70
. . . People (in the labour force) unemployed	0.70
. . . Families that are one parent families with dependent offspring only	0.67
. . . Households paying rent who pay less than $120 per week (excluding $0 per week)	0.67
. . . People aged under 70 who have a long-term health condition or disability and need assistance with core activities	0.61
. . . Occupied private dwellings with no car	0.57
. . . People who identified themselves as being of Aboriginal and/or Torres Strait Islander origin	0.52
. . . Occupied private dwellings requiring one or more extra bedrooms (based on Canadian National Occupancy Standard)	0.52
. . . People aged 15 years and over who are separated or divorced	0.51
. . . Employed people classified as Machinery Operators and Drivers	0.51
. . . People aged 15 years and over who did not go to school	0.44
. . . Employed people classified as Low Skill Community and Personal Service Workers	0.44
. . . People who do not speak English well	0.33
Eigenvalue	6.62
% variance explained	39.00

Note. From: Pink (2008B). Socioeconomic Indexes for Areas—Technical Paper 2006, Australian Bureau of Statistics.

(*Appendices continue*)

Appendix B

Derivation of the Past-Year Disordered Gambling Factor Score Based on Extraction of a Single Factor From an Exploratory and Confirmatory Factor Analysis of Data Obtained From Australian Adults

Past-year item[a]	Standardized factor loadings	Thresholds[b]
DSM1 (needs to increase amounts of money gambled)	0.89	1.95
DSM2 (restless or irritable when attempting to control)	0.88	2.42
DSM3 (unsuccessful efforts to control)	0.95	2.48
DSM4 (preoccupied with gambling)	0.95	2.43
DSM5 (gambles to escape from problems or mood)	0.82	1.93
DSM6 (chasing losses)	0.92	2.08
DSM7 (lies to conceal extent of involvement)	0.88	2.19
DSM8 (relationships or career suffered)	0.86	2.56
DSM9 (relied on others for money)	0.90	2.60
SOGS4 (chasing losses)	0.86	2.73
SOGS5 (lying about winning)	0.80	1.93
SOGS6 (felt had a problem)	0.93	2.31
SOGS7 (gambled more than intended)	0.86	1.03
SOGS8 (others criticized gambling)	0.88	2.11
SOGS9 (felt guilty about gambling)	0.90	1.80
SOGS10 (didn't think could stop)	0.90	2.29
SOGS11 (hid signs of gambling)	0.77	2.15
SOGS13 (gambling-related money arguments)	0.90	2.44
SOGS14 (failed to repay loans)	—	—
SOGS15 (gambling-related school or work absences)	0.63	2.90
SOGS16a (borrowed from household money)	0.78	2.29
SOGS16b (borrowed from spouse)	0.59	2.13
SOGS16c (borrowed from other relatives)	0.81	2.52
SOGS16d (borrowed from banks, finance companies . . .)	—	—
SOGS16e (obtained credit card loans)	0.67	2.37
SOGS16f (borrowed from loan sharks)	—	—
SOGS16g (cashed in shares, bonds . . .)	—	—
SOGS16h (sold personal or family property)	0.70	3.02
SOGS16i (written a bad check)	—	—
Gambling versatility (0–11)	0.49	—[b]
Eigenvalue	17.18	
% variance explained	68.84	

Note. Factor loadings and thresholds are from a confirmatory factor analysis, the eigenvalue and % variance explained are from an exploratory factor analysis. DSM = Diagnostic and Statistical Manual of Mental Disorders, SOGS = South Oaks Gambling Screen.

[a] The DSM and SOGS item numbers correspond to the diagnostic criterion numbers or scale item numbers provided in the original publications. [b] Thresholds are only applicable to categorical items; all but one item (gambling versatility) are categorical. The mean gambling versatility score was 2.77 (*SE* 0.04). — indicates that item was excluded because it was endorsed by only 0–2 respondents.

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