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Potential Causal Influence of Neighborhood Disadvantage on Disordered Gambling: Evidence From a Multilevel Discordant Twin Design

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Abstract

The quality of the neighborhood in which one lives has been linked to disordered gambling (DG), but whether this reflects a causal relation has not yet been empirically examined. Participants were 3,450 Australian twins who completed assessments of past-year DG and personality and for whom census-derived indicators of disadvantage were used to characterize their neighborhood. Multilevel models were employed to estimate within-twin-pair and between-twin-pair effects of neighborhood disadvantage on DG, with the within-twin-pair effect representing a potentially causal association and the between-twin-pair effect representing a noncausal association. There was robust evidence for a potentially causal (as well as a noncausal) effect of neighborhood disadvantage on DG; in contrast, parallel analyses of past-year alcohol-use disorder (AUD) failed to find evidence of a potentially causal effect. These results support efforts focused on identifying the active ingredients contributing to the effect of neighborhood disadvantage on DG and developing interventions to limit their impact.

Keywords

gambling disorder, discordant twins, neighborhood disadvantage

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The quality of the neighborhood in which one lives has been linked to disordered gambling (DG) in the United States, Great Britain, New Zealand, and Australia (Barnes, Welte, Tidwell, & Hoffman, 2013; Pearce, Mason, Hiscock, & Day, 2008; Slutske, Deutsch, Statham, & Martin, 2015; Wardle et al., 2011; Welte et al., 2004). In Australia, the prevalence of past-year DG was nearly eight times higher among those living in the highest decile of neighborhood disadvantage (3.3%) compared to those living in the lowest decile (0.4%; Slutske et al., 2015). In the United States, those living in the highest quintile of neighborhood disadvantage reported 2.5 times as many DG symptoms as those in the lowest quintile, and the association between neighborhood disadvantage and DG remained even after a host of demographic, personality, family, and gambling-accessibility measures were entered as predictors in a multivariate analysis (Welte, Barnes, Tidwell, & Wieczorek, 2017). The authors concluded by noting that "something about poor neighborhoods . . . in itself promotes problem gambling . . . we have speculated that individuals living in disadvantaged neighborhoods have few examples of financial success by conventional means and are therefore more vulnerable to the attraction of gambling" (Welte et al., 2017, p. 339).

Despite the robustness of these findings, it may be premature to conclude that the neighborhood in which one lives is truly a measure of the environment because, like many other putative environments (Kendler &

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Baker, 2007; Plomin, DeFries, Knopik, & Neiderhiser, 2016), living in a deprived neighborhood is partially heritable (Marioni et al., 2014; Sariaslan et al., 2016; Slutske et al., 2015). In observational studies (as in life), environments are not randomly assigned to people. Rather, our environments arise in part from genetically influenced choices on the basis of our abilities, interests, talents, and proclivities, and are inherited along with our genes from our parents (Scarr & McCartney, 1983). Establishing that a risk factor is truly environmental requires more sophisticated research designs than have typically been employed in the study of DG.

This is a question that is of critical importance to public health (Kendler, 2017). Establishing that a risk factor is truly environmental is synonymous with establishing that the association between the risk factor and the outcome is causal. The discordant-twin design is a "natural experiment" or "quasi-experimental" method (McGue, Osler, & Christensen, 2010; Rutter, 2007a, 2007b) in which an unexposed twin serves as the control for an exposed cotwin in examining potentially causal associations. It is based on the knowledge that monozygotic (MZ) twins are perfectly correlated for genetic and family environmental background factors, and dizygotic (DZ) twins are perfectly correlated for family environmental background factors and correlated 0.5 (on average) for genetic factors. A comparison of DG among MZ twins who are discordant for an exposure such as neighborhood disadvantage allows one to control completely for genetic and family environmental background factors (that is, between-family risk factors), and a comparison among discordant DZ twins allows one to partially control for genetic and completely control for family environmental background factors. This type of control is more powerful than the standard statistical controls that are commonly used in the behavioral sciences because it controls for a whole host of possible confounds—including those that we know about and even those that we do not. A potentially causal effect[1] of neighborhood disadvantage on DG would be implicated when the twin living in a more disadvantaged neighborhood has a greater likelihood of DG than the cotwin living in a less disadvantaged neighborhood. On the other hand, if there are no differences between the twin living in a more disadvantaged neighborhood and the cotwin living in a less disadvantaged neighborhood in the likelihood of DG, then the association between neighborhood disadvantage and DG is more likely to be due to between-family differences that are related to both living in a disadvantaged neighborhood and DG. These two scenarios are not mutually exclusive, and neighborhood disadvantage could be both a causal factor as well as noncausally associated via between-family differences.

Although increasingly deployed in the behavioral sciences (D'Onofrio, Lahey, Turkheimer, & Lichtenstein, 2013; Lahey & D'Onofrio, 2010), this research design has been used in only one previous investigation of an important putative risk factor for DG: early age of gambling initiation (Kessler et al., 2008; Lynch, Maciejewski, & Potenza, 2004). The discordant-twin design was employed to answer the question of whether an earlier age of initiation of gambling was causally related to later regular gambling and DG (Slutske et al., 2014). There was little evidence that early gambling was causally related to later gambling involvement and disorder after shared familial risk factors were controlled. That is, the earlier-gambling twin was no more likely to proceed to gambling regularly or to develop symptoms of DG than was the later-gambling cotwin. This suggests that early gambling initiation is better thought of as an early marker of genetic and family environmental risk factors for gambling disorder. This finding has potentially important public health significance in that it suggests that efforts to delay gambling among youth may not have the intended consequence of reducing the numbers of individuals who develop a gambling disorder.

The present study represents an investigation of another putative risk factor for DG: residing in a disadvantaged neighborhood. A previous investigation found that genetic, shared environmental, and unique environmental factors explained 25%, 20%, and 55% of the variation in residing in a disadvantaged neighborhood (Slutske et al., 2015). One of the main questions of interest was the extent to which the genetic and environmental contributions to moving to or remaining in a disadvantaged neighborhood overlapped with genetic and environmental contributions to gambling involvement and disorder. The association between neighborhood disadvantage and the frequency of gambling was completely explained by overlapping familial (genetic and shared environmental) risk factors, which is inconsistent with a potentially causal association. However, the association between neighborhood disadvantage and DG among men was explained in part by nonfamilial (unique environmental) factors, which is consistent with a potentially causal association (Slutske et al., 2015).

Because comparing discordant MZ twins controls only for genetic and shared environmental background factors, there still exists the possibility that there may be individual-specific factors related to twin discordance—that is, the differential exposure of the two twins—that are also related to the outcome. Thus, when conducting a discordant-twin study, it is also important to explore the factors associated with twin discordance in exposure (McGue et al., 2010). For example, socioeconomic and personality differences may lead to one twin living in a more disadvantaged neighborhood than the other twin,

and this difference may also be related to the one twin developing a gambling disorder. Therefore, we also compared the results of models that included covariates, such as educational attainment, household income, and the "Big Three" personality traits of positive emotionality, negative emotionality, and behavioral undercontrol to unadjusted models in order to establish the robustness of the effects.

Method

Participants

Participants were selected from a sample of 4,764 members of the Australian Twin Registry Cohort II (57% female, age range = 32–43 years [mean = 37.7]). Nearly all of the participants were White (~97%), with the next most common racial category being Asian (~2%). The participants were overwhelmingly of European origin, particularly northwest Europe, and mainly the United Kingdom and Ireland (see Slutske et al., 2009 for more participant details). The participants represented a broad cross-section of the general population of Australia. Similar to most community based twin cohorts, the sample was relatively socioeconomically advantaged (median household income of approximately AU $62,500 [equivalent to U.S. $49,375]). Data from participants who were living overseas or whose state of residence was unknown were excluded. Because complete pairs were necessary for the multilevel models, the sample was limited to the 3,596 individuals whose cotwin's data were also available. Informed consent was obtained from all participants and the study was approved by the institutional review boards at the University of Missouri and the QIMR Berghofer Medical Research Institute.

Procedure

Respondents completed a structured psychiatric telephone interview conducted in 2004 through 2007 (response rate = 80.4%), during which time gambling behaviors were assessed. Interviews were conducted by trained lay interviewers who were blind to the status of the cotwin. The participants who completed the telephone interview were mailed a paper-and-pencil personality questionnaire that was usually returned within 1 month of the interview. Personality questionnaires were available for 3,106 (86%) of the participants whose data were included in this study.

Environmental-exposure measure

Neighborhood disadvantage. Neighborhood 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).[2] Census data were matched successfully for 3,450 (96%) of the complete twin pairs (970 MZ female, 632 MZ male, 692 DZ female, 404 DZ male, 752 unlike-sex DZ); this was the final analytic sample. Out of 2,515 postal codes in the 2006 Australian census, there were 1,245 different postal codes represented in the sample, with up to 23 participants residing in each (M = 2.95, SD = 2.76). Seventy-nine percent of the participants resided in a city and 21% in a rural area. The median population size of the postal code areas of the participants in this sample was 16,305 residents. Forty percent of the postal-code areas included a single participant, and 81% included fewer than 5 participants. The majority of twins and cotwins (80%) resided in a different postal-code area.

We used an index of relative socioeconomic disadvantage (IRSD) created by the Australian Bureau of Statistics (Pink, 2008a, 2008b). This index has been used in its present form since 1986, and is updated every 5 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). 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 (for example) low income, low educational attainment, unskilled employment or unemployment, single-parenthood, and subsidized living or low-rent housing (see Slutske et al., 2015 for more details about the IRSD). The IRSD was reversed so that higher scores reflected greater disadvantage. For these analyses, we used the IRSD decile, which ranged from 1 (lowest disadvantage) to 10 (highest disadvantage). The postal codes represented in this sample were slightly less disadvantaged on average than the Australian population. Although the entire range of disadvantage was represented, postal codes in the top three deciles were underrepresented and in the bottom three deciles of disadvantage were overrepresented.

Outcome measures

The main outcome measure of interest was symptoms of past-year DG. For comparison, analyses were also conducted for symptoms of past-year AUD.

Disordered gambling. Past-year symptoms of DG 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). These two measures of DG were correlated $r = .70$ ($p < .0001$). The nine DG symptoms from the 5th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–5;* American Psychiatric Association [APA], 2013)[3] and 20 items from the SOGS were summed to form the DG outcome measure (coefficient alpha = 0.85). A one-factor model provided an excellent fit to these 29 indicators (Slutske et al., 2015). Biometric modeling revealed that the familial sources of variation in liability to *DSM* DG symptoms completely overlapped with the familial sources of variation in liability to SOGS DG symptoms (Slutske, Zhu, Meier, & Martin, 2011). Using the SOGS in conjunction with the *DSM* symptom set more thoroughly describes the full DG continuum (Slutske et al., 2011). A previous study reported that genetic and unique environmental factors explained 45% and 55% of the variation in past-year DG among men, and 42% and 58% of the variation among women (Slutske et al., 2015).

Alcohol use disorder. Past-year symptoms of *DSM–IV* (APA, 1994) AUD were assessed using the World Health Organization Composite International Diagnostic Interview (Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998; World Health Organization, 1992). The seven alcohol-dependence symptoms and the four alcohol-abuse symptoms were summed to form the AUD outcome measure (coefficient alpha = 0.73).

Covariate measures

Personality dimensions. The personality questionnaire contained a modified 177-item version of the Multidimensional Personality Questionnaire (MPQ; Slutske, Cho, Piasecki, & Martin, 2013; Tellegen & Waller, 2008). The wording of some of the MPQ items was modified for use in Australia. The MPQ contains items that measure 10 lower-order factors, which in turn form three distinct superfactors (the "Big Three"): Positive emotionality includes well-being, social potency, achievement, and social closeness; negative emotionality includes stress reaction, alienation, and aggression; and constraint includes control, harm avoidance, and traditionalism. Scale scores were created by dividing the sum of the items by the number of nonmissing items; scales with more than three missing items were considered missing. These scale scores were converted to *T* scores (i.e., mean = 50, *SD* = 10) prior to inclusion as predictors in multilevel model analyses to produce more interpretable scaling of model coefficients. The internal consistency reliabilities in the present study for the three superfactors ($\alpha = 0.85$ to 0.88, mean $\alpha = 0.87$) were good. The percentage of variation in the three superfactors attributable to genetic, shared, and nonshared environment were 48%, 0, and 52% for positive emotionality, 43%, 1%, and 57% for negative emotionality, and 40%, 7%, and 53% for constraint, which is consistent with the worldwide twin literature on personality (Polderman et al., 2015).

Socioeconomic status. Participants were asked to use a respondent booklet listing 12 income ranges for response options to report their current combined household gross income before taxes. These 12 ordered categories were used as an ordinal income variable in analyses. Participants also reported their highest educational level, with the options of 8 to 10 years schooling (coded "1"); matriculation/year 12 ("2"); technical, teachers' college, technical and further education, business or secretarial college ("3"); university undergraduate training ("4"); and university postgraduate training ("5"). These ordered categories were treated as an ordinal educational-attainment variable in analyses.

Analytic plan

Descriptive analyses. Associations between study variables were estimated using a survey-data analysis procedure in SAS (SAS Institute Inc., 2009) that took into account the nonindependence of twin-pair observations.

Multilevel modeling analyses. Multilevel modeling (also known as hierarchical linear, mixed, or random-effects modeling) is an extension of conventional regression analysis that has become increasingly popular in the behavioral sciences (Snijders & Bosker, 1999). It is used to take into account and to properly model levels of nesting, or clustered observations, within a data set. In the case of data from pairs of twins, there are two levels of nesting: the individual-twin level is nested within the twin-pair level. Multilevel modeling is a flexible and powerful data analytic approach that can accommodate a continuous index of twin discordance and include covariates for the identification of potential mediators of effects.

Estimation of the multilevel models was conducted using PROC GLIMMIX for generalized linear mixed models (GLMMs) in SAS software Version 9.4. GLMM is a form of multilevel modeling for the analysis of clustered data with nonnormal dependent measures (Hedeker, 2005). A negative binomial distribution and log link function were used because the DG symptom count was skewed with a large proportion of respondents reporting zero symptoms (81.3% of analyzed sample). Both Level 1 and Level 2 variances were estimated, along with a random intercept of twin pair. Coefficients from these models were exponentiated and expressed as incidence rate ratios (IRRs).

An initial set of cotwin control models (McGue, et al. 2010) was performed to (a) test whether there was an overall association between neighborhood disadvantage

and DG symptoms, and (b) identify potential sources of confounding that might contribute to the overall effect. First, the overall association, or "individual-level" effect of exposure was tested in the full sample of twins. Next, separate models were performed that were limited to either DZ or MZ twins. In these zygosity-limited models, the neighborhood disadvantage indicator was decomposed into two variables indexing Level 1 (i.e., within-twin pair) and Level 2 (i.e., between-twin pair) sources of variance (Begg & Parides, 2003). The between-pair effect was represented using the mean score across both twins; the within-pair effect was indexed by subtracting the pair mean from each twin's own score. In this parameterization, the within-pair effect (i.e., comparison against the cotwin) represents the quasicausal unique effect of an environmental exposure, and the between-pair effect measures between-family genetic and shared environmental confounds that contribute to the phenotypic association between the environmental measure and outcome (McGue et al., 2010; Turkheimer & Harden, 2014).

The estimates of the within-pair effect among DZ twins accounts for shared environmental and partially accounts for genetic confounds. Thus, if this effect is markedly smaller than the individual-level estimate, this indicates that shared environmental or genetic factors may be inflating the overall association. The within-pair effect among MZ twins represents the most stringent test, as it fully controls for both genetic and shared environmental confounds. If this estimate is significant, this provides strong evidence for a quasicausal effect of neighborhood disadvantage exposure on DG. Comparison of the magnitude of the within-pair MZ effect to the individual-level and within-pair DZ estimates can indicate the presence and likely sources of confounding, if any, affecting the overall association (McGue et al., 2010).

Next, a series of models was tested in the pooled sample of twins, incorporating additional explanatory variables that might help account for any disadvantage effects observed in the cotwin control analyses. A base model predicted DG from the within- and between-pair neighborhood disadvantage variables, adjusting for sex and age.[4] Because neither the main effect for zygosity (IRR = 1.05, 95% confidence interval [CI] = [0.83, 1.34], p = .675) nor the Zygosity × Within-Pair Disadvantage effect (IRR = 0.95, 95% CI = [0.85, 1.07], p = .3924) was significant, these predictors were not included in the models. Subsequent models added within- and between-pair effects of (a) Big Three personality traits, (b) educational attainment, and (c) household income. A final, fully adjusted model incorporated all of these explanatory covariates simultaneously. Of particular interest was the significance of the within- and between-pair effects included in each of the models, as well as the extent to which the inclusion of the predictors reduced or completely eliminated the within- and between-pair effects of neighborhood disadvantage.

Results

Descriptive findings

Most twin pairs (69%) were discordant for their level of disadvantage; in 19% of pairs, the twins differed by one decile; in 15%, they differed by two deciles; in 11%, they differed by three deciles; in 9%, they differed by four deciles, and in 15%, they differed by five or more deciles. The average discordance was 2.09 (SD = 2.14) deciles.

The correlation between DG and neighborhood disadvantage was significant but modest (r = 0.10, p < .0001; converted to standardized mean difference [effect size]: d = 0.20). DG was significantly associated with nearly all of the covariates (see Table 1). Negative emotionality, educational attainment, and household income were significantly associated with both DG and neighborhood disadvantage, implicating these covariates as potentially accounting for the within-twin-pair (i.e., potentially causal) or between-twin-pair (i.e., family-level confounding) effects of disadvantage on DG in the multilevel models.

Multilevel models

Findings from the cotwin control analyses are depicted in Figure 1. The individual-level effect of neighborhood disadvantage was significant (IRR = 1.11, p < .001), indicating that a one-decile difference in disadvantage was associated with an 11% increase in the expected DG symptom count. The within-pair disadvantage effect among DZ twins was similar in magnitude, though not statistically significant (IRR = 1.08, p = .075). The within-pair effect in MZ twins was significant and comparable in magnitude to individual-level and DZ within-twin estimates (IRR = 1.14, p < .001). The presence of a within-pair effect among MZ twins provides strong evidence for a quasicausal effect of neighborhood disadvantage on gambling symptoms. A supplemental model in the pooled sample of MZ and DZ twins indicated that zygosity did not moderate the within-pair effect of neighborhood disadvantage, interaction IRR = 0.95, 95% CI = [0.85, 1.07], p = .392, meaning that the within-pair effect did not significantly differ among MZ and DZ twins. The fact that the magnitude of this within-pair effect was comparable to the individual-level effect despite cotwin controls is potentially consistent with a fully causal exposure effect (McGue et al., 2010).

Findings from models investigating effects of measured explanatory variables are summarized in Table 2.

Table 1. Correlations Between Study Variables

Predictor	DG	AUD	Sex	Age	Disad	PE	NE	Constraint	EA	Income
Disordered gambling										
Alcohol-use disorder	**.27**									
Sex	**.07**	**.10**								
Age	.02	–.03	–.02							
Disadvantage	**.10**	.01	.01	–.01						
Positive emotionality	–.04	–.03	**.10**	–.02	**–.15**					
Negative emotionality	**.21**	**.17**	**.08**	–.03	**.12**	**–.17**				
Constraint	**–.08**	**–.15**	**–.32**	.01	.03	**–.15**	**–.14**			
Educational attainment	**–.10**	**–.05**	–.02	–.04	**–.23**	**.19**	**–.18**	**–.10**		
Household income	**–.06**	**–.05**	**.10**	.01	**–.28**	**.23**	**–.20**	–.03	**.29**	
Mean	0.42	0.35	0.41	37.60	4.38	0.58	0.26	0.68	2.92	8.20
SD	1.46	1.00	0.49	2.30	2.66	0.15	0.15	0.14	1.32	2.22

Note: Boldface type indicates significant correlations ($p < .05$). Sex is coded 0 = female, 1 = male. DG = disordered gambling; AUD = alcohol-use disorder; Disad = disadvantaged; PE = positive emotionality; NE = negative emotionality; EA = educational attainment.

In the base model, both within-pair (IRR = 1.11, $p < .001$) and between-pair (IRR = 1.13, $p < .001$) differences in neighborhood disadvantage were significantly associated with gambling symptom count. When the base model was expanded to include personality traits as additional predictors, the quasicausal within-pair disadvantage effect remained (IRR = 1.09, $p = .006$). Additionally, significant within-pair effects for two traits indicated quasicausal influences, such that the twin reporting higher negative emotionality (IRR = 1.04, $p < .001$) and lower constraint (IRR = 0.96, $p < .001$) experienced more gambling symptoms in the past year compared to the cotwin. The between-pair effect for disadvantage also remained significant but was somewhat diminished (IRR = 1.08, $p = .015$). Between-pair negative emotionality (IRR = 1.08, $p < .001$) was significant, and thus potentially accounted for some of the familial association between living in disadvantaged neighborhoods and gambling symptoms. Neither the within-pair nor between-pair effects for positive emotionality were significant (ps > .597)

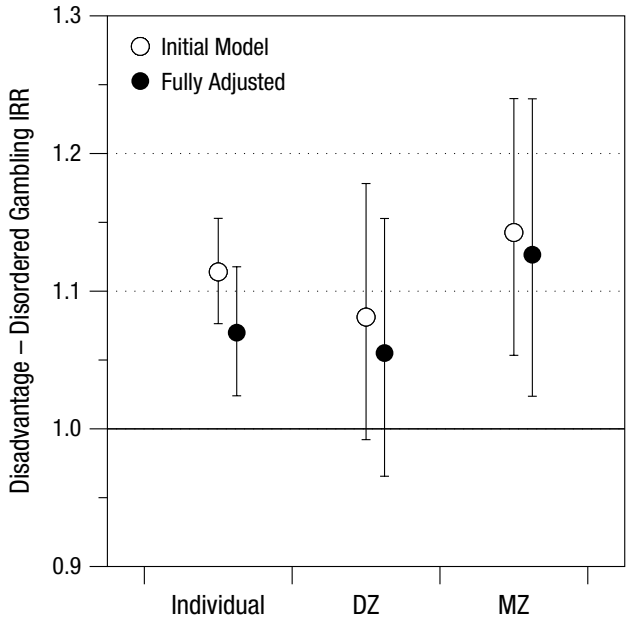

Fig. 1. Effect sizes (expressed as incidence rate ratios) and 95% confidence intervals for the association between neighborhood disadvantage and disordered gambling (DG) symptom count symptom count at the individual level and at the within-pair level in dizygotic (DZ) and monozygotic (MZ) twins. Estimates of each effect are depicted when covarying sex and age (initial models) and when fully adjusting (fully adjusted models) for all covariates listed in Table 2. The within-pair effect of neighborhood disadvantage did not significantly differ among DZ and MZ twins in the initial models ($p = .392$) or in the fully-adjusted models ($p = .3881$). Note: IRR = incidence rate ratio.

The within-pair (IRR = 1.11, $p < .001$) and between-pair (IRR = 1.09, $p = .002$) effects of neighborhood disadvantage both remained significant in the model accounting for educational attainment. Lower education potentially exerted a causal effect on gambling symptoms (within-pair IRR = 0.86, $p = .019$). Twins from families with lower average levels of educational attainment experienced more past-year gambling symptoms (IRR = 0.77, $p < .001$).

In the model accounting for household income, disadvantage effects remained significant both at the within-pair (IRR = 1.11, $p < .001$) and between-pair (IRR = 1.11, $p < .001$) levels. The within-pair effect for household income was nominally significant (IRR = 0.94, $p = .052$). Twins from families with lower incomes tended to report more past-year DG symptoms (IRR = 0.90, $p = .005$).

In the fully adjusted model, the within-pair disadvantage effect remained significant but was reduced in magnitude (IRR = 1.08, $p = .016$). Significant within-pair effects were found for negative emotionality (IRR = 1.04, $p < .001$) and constraint (IRR = 0.96, $p < .001$), suggesting

Table 2. Results From Multilevel Models Predicting Disordered-Gambling Symptom Counts

	Base model		Personality		Education		Income		Fully adjusted	
Predictor	IRR	95% CI	IRR	95% CI	IRR	95% CI	IRR	95% CI	IRR	95% CI
Sex	1.80***	[1.45, 2.24]	1.46**	[1.14, 1.87]	1.78***	[1.43, 2.21]	1.88***	[1.50, 2.35]	1.47**	[1.14, 1.90]
Age	1.02	[0.97, 1.08]	1.01	[0.95, 1.07]	1.02	[0.97, 1.07]	1.03	[0.98, 1.08]	1.02	[0.96, 1.08]
WP disadvantage	1.11***	[1.05, 1.18]	1.09***	[1.02, 1.16]	1.11***	[1.05, 1.17]	1.11***	[1.05, 1.18]	1.08*	[1.02, 1.15]
BP disadvantage	1.13***	[1.07, 1.20]	1.08*	[1.01, 1.14]	1.09**	[1.03, 1.15]	1.10***	[1.04, 1.17]	1.04	[0.98, 1.11]
WP positive emotionality			1.00	[0.98, 1.01]					1.00	[0.98, 1.02]
BP positive emotionality			1.00	[0.98, 1.01]					1.00	[0.98, 1.02]
WP negative emotionality			1.04***	[1.03, 1.06]					1.04***	[1.02, 1.06]
BP negative emotionality			1.07***	[1.06, 1.09]					1.07***	[1.05, 1.09]
WP constraint			0.96***	[0.95, 0.98]					0.96***	[0.95, 0.98]
BP constraint			0.99	[0.97, 1.00]					0.99	[0.97, 1.00]
WP educational attainment					0.86*	[0.75, 0.97]			0.90	[0.77, 1.04]
BP educational attainment					0.77***	[0.69, 0.86]			0.85*	[0.75, 0.96]
WP household income							0.94	[0.88, 1.00]	0.98	[0.91, 1.05]
BP household income							0.90**	[0.84, 0.97]	0.97	[0.89, 1.05]

Note: Because neither the main effect for zygosity (IRR = 1.05, 95% CI = [0.83, 1.34], $p = .675$) nor the Zygosity × Within-Pair Disadvantage effect (IRR = 0.95, 95% CI = [0.85, 1.07], $p = .3924$) was significant, these predictors were not included in the models.
IRR = incidence rate ratio; CI = confidence interval; WP = within-pair; BP = between-pair.
*$p < .05$. **$p < .01$. ***$p < .001$.

that these traits may be important mechanisms accounting for some of the apparent quasicausal effect of neighborhood disadvantage on DG symptoms. The between-pair disadvantage effect was not significant in the fully adjusted model (IRR = 1.04, $p = .198$). At the between-pair level, negative emotionality (IRR = 1.07, $p < .001$) and educational attainment (IRR = 0.85, $p = .011$) were the only explanatory variables associated with gambling symptoms.

Given evidence that some explanatory variables were related to DG symptoms at the within-pair level, we conducted the sequence of cotwin control models after fully adjusting for these variables. Results were similar to the initial models (Fig. 1). Disadvantage remained significantly associated with DG symptoms at the individual level (IRR = 1.07, $p = .003$). The within-pair disadvantage effect was not significant among DZ twins (IRR = 1.06, $p = .232$) but was significant among MZ twins (IRR= 1.13, $p = .016$), and the within-pair effect of neighborhood disadvantage among DZ and MZ twins did not significantly differ ($p = .3881$).

Probing the effects of negative emotionality and constraint

Additional analyses sought to further characterize the effects of negative emotionality and constraint in the personality-adjusted models by omitting these traits and substituting their corresponding facet-scale scores. All three facets of negative emotionality (stress reaction, aggression, and alienation) were significantly associated with both DG and neighborhood disadvantage (see Table S1 in the Supplemental Material available online), suggesting that they may account for the within- and between-twin-pair effects of disadvantage on DG in the multilevel models. Facets of constraint were associated with DG (control and harm avoidance) or neighborhood disadvantage (traditionalism), but not both.

In a multilevel analysis, within-pair differences in stress reaction (IRR = 1.03, $p = .006$), aggression (IRR = 1.02, $p = .009$), and control (IRR = 0.97, $p < .001$) were significant predictors of DG (Table 3). At the between-pair level, negative emotionality facets of stress reaction (IRR = 1.02, $p = .047$), alienation (IRR = 1.04, $p < .001$), and aggression (IRR = 1.03, $p = .001$), and the constraint facets of low control (IRR = 0.91, $p < .001$) and high traditionalism (IRR = 1.02, $p = .027$), were significant predictors of DG.

Probing the effects of using alternate measures of DG

All of the models were rerun substituting as the dependent variable either a *DSM–5* (APA, 2013) DG symptom count (Table S2 in the Supplemental Material) or SOGS symptom count (Table S3 in the Supplemental Material). Using the *DSM–5* measure, there was evidence for within-pair and between-pair effects in the baseline analyses that were no longer significant after inclusion of the full set of covariates. Using the SOGS measure, the within-pair effect and between-pair effects were significant in the baseline model. The within-pair effect persisted after including covariates but the between-pair effect did not.

Table 3. Results From Multilevel Models Testing Facets of MPQ Negative Emotionality and Constraint Predicting Disordered Gambling Symptom Counts

Predictor	IRR	95% CI	*p*
Sex	1.40	[1.06, 1.85]	.016
Age	1.00	[0.95, 1.06]	.945
WP disadvantage	1.09	[1.03, 1.16]	.006
BP disadvantage	1.05	[0.99, 1.12]	.081
WP positive emotionality	0.99	[0.98, 1.01]	.492
BP positive emotionality	0.99	[0.97, 1.01]	.238
Negative emotionality			
WP stress reaction	1.03	[1.01, 1.05]	.001
BP stress reaction	1.02	[1.00, 1.04]	.047
WP alienation	1.00	[0.99, 1.02]	.698
BP alienation	1.04	[1.02, 1.06]	< .0001
WP aggression	1.02	[1.01, 1.04]	.009
BP aggression	1.03	[1.01, 1.05]	.001
Constraint			
WP control	0.97	[0.95, .99]	.0004
BP control	0.96	[0.95, .98]	< .0001
WP harm avoidance	0.99	[0.97, 1.01]	.168
BP harm avoidance	1.00	[0.99, 1.02]	.669
WP traditionalism	0.99	[0.98, 1.01]	.452
BP traditionalism	1.02	[1.00, 1.04]	.027

Note: Because neither the main effect for zygosity ($p = .675$) nor the Zygosity × Within-Pair Disadvantage effect ($p = .3924$) was significant, these predictors were not included in the models. MPQ = Multidimensional Personality Questionnaire; IRR = incidence rate ratio; CI = confidence interval; WP = within-pair; BP = between-pair.

Probing the similarity of the results obtained for another addictive disorder

Analyses were conducted for AUD to discern whether the results were similar to those obtained for DG. Inspection of Table 1 reveals that AUD and DG had similar correlates except for neighborhood disadvantage, which was not significantly associated with past-year AUD symptoms ($r = .01$). Cotwin control analyses with the *DSM–IV* (APA, 2013) AUD symptom count as the dependent variable indicated that there was no effect of neighborhood disadvantage on AUD symptoms at the individual level (IRR = 1.00, 95% CI = [0.97, 1.03],

$p = .995$). The within-pair effects for disadvantage were not significant in either DZ (IRR = 1.01, 95% CI = [0.93, 1.10], $p = .749$) or MZ twins (IRR = 0.99, 95% CI = [0.99, 1.07], $p = .740$). In multilevel modeling analyses incorporating explanatory variables (Tables S4 in the Supplemental Material), there was no evidence for a within-pair effect of neighborhood disadvantage on AUD ($ps > .233$). After including covariates, however, there was a significant inverse relation between the between-pair effect of neighborhood disadvantage and AUD wherein twins from families who were living in less disadvantaged neighborhoods reported more past-year symptoms of AUD (IRR = 0.92, 95% CI = [0.87, 0.97], $p = .003$).

Discussion

The purpose of this study was to examine the relation between neighborhood disadvantage and DG using a research design that can determine whether the association is truly environmental and potentially causal. Using a discordant-twin design, levels of DG were compared within twin pairs who were discordant for neighborhood disadvantage. A potentially causal effect of neighborhood disadvantage on DG would be implicated if the twin residing in a more disadvantaged neighborhood exhibited more symptoms of DG than the cotwin living in a less disadvantaged neighborhood.

Potentially causal effect of neighborhood disadvantage

There was a significant association between the MZ within-pair difference in neighborhood disadvantage and DG that was as large as the overall association, suggesting that the relation is not confounded. However, because comparing discordant MZ twins controls only for genetic and shared environmental background factors, there still exists the possibility that there may be unique (nonshared) environmental factors related to twin discordance—that is, reasons also related to DG for why one twin is living in a more disadvantaged neighborhood than the other. Therefore, we examined whether the effect remained after controlling for educational attainment, household income, and the Big Three personality traits of positive emotionality, negative emotionality, and constraint. The evidence for a potentially causal effect of neighborhood disadvantage on DG persisted even after controlling for these covariates. These analyses also pointed to two facets of negative emotionality, stress reaction (similar to "Big Five" neuroticism, or emotional instability), and aggression (similar to Big Five low agreeableness) and a facet of constraint, low control (impulsivity), in accounting for a portion of this effect. That is, high stress reactivity, disagreeableness, and impulsivity were associated with both living in a disadvantaged neighborhood and symptoms of DG, even after accounting for overlapping genetic and shared environmental background factors.

The potentially causal effect of neighborhood disadvantage on DG contrasts with the results of parallel analyses of past-year AUD that failed to find evidence of a potentially causal effect. A national U.S. study also found a significant association between neighborhood disadvantage and DG, but not AUD (Barnes et al., 2013). This distinction provides a clue to the potential mechanisms underlying the neighborhood effect. Perhaps feelings of hopelessness that may be engendered by living in a neighborhood in which there are higher rates of unemployment (Morselli, 2017) are relieved by the hope of a potential life-transformative gambling win (Clotfelter & Cook, 1991; Downs, 2008). This false hope of a future big win is also one of the diagnostic criteria for DG: "chasing losses," wherein the solution to one's gambling problem lies in more gambling. AUD and other addictions are not characterized by such motivations and beliefs.

Noncausal effect of neighborhood disadvantage

Multilevel models are also useful in probing the noncausal, or between-family association between neighborhood disadvantage and DG. These between-family effects are the genetic and environmental factors that twins from the same family have in common. In contrast to the within-twin effects, the significant between-twin effect of neighborhood disadvantage on DG could be completely explained by individual differences in personality. In particular, all three facets of negative emotionality and two facets of Big Three constraint, low control (impulsivity), and traditionalism appeared to explain the between-twin effect. This builds on a previous article (Slutske et al., 2015) that presented evidence for a significant overlap in the genetic factors associated with moving to or remaining in a disadvantaged neighborhood and DG, but could only speculate about what was driving this genetic association. These new analyses suggest that the genetic factors that are associated with both neighborhood disadvantage and DG are genetically influenced personality traits, with the strongest contributors being alienation (distrustfulness), control (impulsivity), and aggression (disagreeableness). The gene-environment correlation observed between neighborhood disadvantage and DG could be completely explained by individual differences in personality that were related to both the quality of the neighborhood

in which a twin lived and the development of gambling pathology. In other words, in addition to a potentially causal effect of living in a disadvantaged neighborhood, individuals at genetic risk for DG were actually more likely to be exposed to this causal risk factor, primarily via genetic influences on distrustfulness, disagreeableness, and impulsivity.

The importance of the measure of DG

It is noteworthy that the evidence for a quasicausal effect of neighborhood disadvantage on gambling disorder was not firmly established when restricting the analyses to the *DSM–5* (APA, 2013) symptom set, but was robustly demonstrated with a composite measure that incorporated SOGS items. We previously advocated using this approach in etiologic research (Slutske et al., 2011), and our suggestion was put to good use in a genome-wide association study of a quantitative DG trait (Lind et al., 2013) and in a recent article in which the neighborhood effect on gambling disorder in the United States was demonstrated (Welte et al., 2017).

A concern for the future of the *DSM* diagnosis of gambling disorder is how the *DSM* can move forward in offering a dimensional approach to describing DG (Regier, Kuhl, & Kupfer, 2013). The nine *DSM–5* (APA, 2013) symptoms do not do a very good job of this, because the symptoms tap only a very narrow high end of the latent DG continuum. In a combined item-response-theory analysis of the *DSM* and SOGS symptoms, the severities (difficulties) for the *DSM–5* symptoms were uniformly high, whereas the severities for the SOGS items were more variable (Slutske et al., 2015). There were a number of items unique to the SOGS that were relatively less psychometrically severe than the *DSM* symptom set (for example, "Did you ever gamble more than you intended to?" and "Have you ever felt guilty about the way you gamble, or what happens when you gamble?"), meaning that they were endorsed by individuals lower on a latent DG continuum. These are just the sort of items that would be eliminated were the focus on the identification of items with high specificity for a categorical disorder. In contrast, for the development of a dimensional approach to describing DG, it is desirable to have items that represent a broad range of severities because this will provide better measurement of DG across the full continuum. This was not accomplished in this study, as the average item difficulty was 2.5, the lowest item difficulty was 1.0 (Slutske et al., 2015), and 95% and 83% of the participants had scores of 0 on the *DSM* and SOGS measures, respectively.[5] Although borrowing items from the SOGS (or other measures) is a good place to start in characterizing variation in DG above and below the *DSM* threshold, future efforts might focus on developing DG assessments that are informative across all levels of gambling pathology found in the general population. If we truly think that DG lies on a continuum, this is just what is needed.

Possible explanations for the potentially causal effect of neighborhood disadvantage

Exactly how neighborhood disadvantage causes DG cannot be deduced from these analyses, although there are clues from established risk factors. Neighborhood disadvantage may be causally related to DG via increased access to gambling opportunities. In the United States (Welte, Wieczorek, Barnes, Tidwell, & Hoffman, 2004), New Zealand (Pearce et al., 2008), Canada (Wilson, Derevensky, Gilliland, Gupta, & Ross, 2006), England (Wardle, Kelly, Astbury, & Reith, 2014), and Australia (Marshall & Baker, 2002), 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; Wardle et al., 2014; Wilson et al., 2006).

Other aspects of neighborhood disadvantage not related to gambling access may also be causally related to the development of DG. Neighborhood disadvantage may function as a chronic stressor or persistent strain (e.g., Boardman, 2004; Steptoe & Feldman, 2001) that may encourage gambling as a means to cope with or escape from problems (Sinha, 2008; Stewart & Zack, 2008), or by rendering one less able to regulate impulses (e.g., Duckworth, Kim, & Tsukayama, 2013; Muraven & Baumeister, 2000; Sinha, 2008). There is a robust association between adverse life events and gambling problems among adolescents (Bergevin, Gupta, Derevensky, & Kaufman, 2006; Cheung, 2016) and adults (Eitle & Taylor, 2011; Ronzitti, Kraus, Hoff, & Potenza, 2018), and the added strain associated with living in a disadvantaged neighborhood may strengthen the association between experiencing negative life events and DG. Such an interaction has been observed in which neighborhood disadvantage strengthened the association between negative life events and the subsequent onset of an episode of depression (Cutrona et al., 2005). Further support for a chronic-stress interpretation of neighborhood disadvantage comes from emerging evidence demonstrating how neighborhood deprivation "gets under the skin" (Prior, Manley, & Jones, 2018). Using data from a large United Kingdom survey,

investigators found that allostatic load (13 biomarkers from the cardiovascular, inflammatory, lipid, and glucose metabolism systems, and the hypothalamic–pituitary axis) was significantly associated with neighborhood disadvantage and partially mediated the associations between neighborhood disadvantage and mental health. Individuals residing in neighborhoods characterized as more deprived had worse allostatic load scores; that is, they showed more physiological "wear and tear" across multiple organ systems as a consequence of chronic exposure to stressful experiences (Prior et al., 2018).

In addition to increased exposure to chronic stress and strain, individuals in disadvantaged neighborhoods have less exposure to protective factors, such as access to institutional resources, mental health facilities, alternative recreational activities, or high-quality support systems (Cutrona, Wallace, & Wesner, 2006; Sampson, Morenoff, & Gannon-Rowley, 2002). In sum, disadvantaged neighborhoods provide environments that are high in risk and low in protective influences, and when combined with the increased access for gambling opportunities, could potentially create a "perfect-storm" environment that fosters the development of gambling problems. Evidence in support of this comes from a previous article demonstrating a significant interaction between neighborhood disadvantage and the density of local gambling venues in predicting DG; the association between neighborhood disadvantage and DG was stronger in states or territories in which there was a greater density of gambling venues (Slutske et al., 2015).

Limitations

This study has several limitations. Because the study was conducted in Australia, the extent to which these findings are generalizable to other settings can be questioned. Although there are important differences in the gambling milieu in Australia compared to the United States, it is reassuring that the association between gambling disorder and neighborhood disadvantage observed in Australia ($r = 0.10$) was of similar magnitude to that observed in the United States ($r = 0.08$; Welte et al., 2017), and there was a similar lack of association of neighborhood disadvantage with AUD (Barnes et al., 2013). More generally, the ubiquity of the neighborhood effect across locales suggests that the effect observed in Australia is likely to be found elsewhere in the world (Sampson, 2011).

The measures of DG and the covariates were collected between 2004 and 2007, and the measure of neighborhood disadvantage was based on 2006 census indicators; for 71% of the participants, the measure of neighborhood disadvantage was actually collected before the self-report measures. This might be a concern if the neighborhood disadvantage index evidenced substantial changes from year to year, but this was not the case. The census-derived index of neighborhood disadvantage was very stable (Pink, 2008a), and there were minimal changes in the level of disadvantage in the neighborhoods between 2004, when the earliest interviews were conducted, and 2006, when the census indicators of disadvantage were collected. It was a limitation, however, that we were not able to characterize the duration of exposure of the participants to neighborhood disadvantage.

The cross-sectional design of the study precluded establishing the temporal relation between neighborhood disadvantage and the personality covariates. The personality covariates were included to provide the most stringent test of a quasicausal association between neighborhood disadvantage and DG by accounting for potential confounding variables that were associated with DG and also may have played a role in twin discordance for neighborhood disadvantage. The rationale behind this was that personality differences between twins could have been a cause of twin discordance in neighborhood disadvantage. For example, the more emotionally unstable twin may have been more likely to move to or remain in a more disadvantaged neighborhood than the more emotionally stable cotwin. On the other hand, it is also plausible that higher levels of emotional instability may be a consequence of living in a more disadvantaged neighborhood (Kim, 2008). The latter scenario would not alter the conclusions drawn because the quasicausal effect remained even with this potentially overly stringent control.

"The idea of 'neighborhood effects' has emerged as a sharp point of contention in the social sciences. . . . Indeed, disputes have erupted across multiple disciplines over the proper level of analysis for assessing neighborhood effects" (Sampson, 2011, p. 227). There may be concerns about the use of census-defined geographic regions to define neighborhoods in the present study, but this was the only feasible approach with these national data. Also, it is not always clear what the correct level of aggregation is when using census-derived geographic entities (e.g., census blocks, block groups, census tracts, or postal code areas) because some effects may be more diffuse and others may be more localized and may vary by the specific neighborhood characteristic and outcome studied (Hipp, 2007). We have followed the lead of previous national studies conducted in Australia (Azar et al., 2015; Bayer et al., 2011) to use the postal-code area. Fortunately, a review of the literature on neighborhood effects suggests that study results tend to be robust across varying definitions of neighborhood (Sampson et al., 2002), "suggesting a

general form to neighborhood effects" (Sampson, 2011, p. 229).

Conclusions and Implications

Evidence from multivariate analyses of epidemiologic survey data led Welte et al. (2017, p. 339) to conclude that "there is something about poor neighborhoods that in itself promotes problem gambling." This article provides new evidence obtained from a quasicausal research design that supports this conclusion. Establishing that neighborhood disadvantage is a potentially causal and malleable environmental risk factor for DG is of great public health importance. It supports efforts focused on identifying the active ingredients contributing to the neighborhood effect and in the development of preventions and interventions. Some of these efforts are currently underway: Harm minimization practices are being developed to help mitigate the negative impacts of the worldwide proliferation of gambling opportunities (Blaszczynski, 2001; Ginley, Whelan, Pfund, Peter, & Meyers, 2017; A. Parke, Harris, J. Parke, Rigbye, & Blaszczynski, 2015; Planzer & Wardle, 2012; Tanner, Drawson, C. J. Mushquash, A. R. Mushquash, & Mazmanian, 2017), and there are major initiatives directed at overall health equity by improving the quality of neighborhoods and communities in which people live (Arcaya et al., 2016; Marmot et al., 2008).

However, in addition to identifying a potentially causal effect of living in a disadvantaged neighborhood, the results of this study suggest that individuals at genetic risk for DG were actually more likely to be exposed to this causal risk factor, primarily via genetic influences on individual differences in personality. This highlights the complex nature of the relation between neighborhood disadvantage and DG and the importance of taking into account the multiple contexts in which problematic gambling behavior can emerge, from genetic risk factors, to personality differences, to the neighborhood in which one lives, as well as the ways in which such contexts may interact with each other.

Action Editor

Kelly L. Klump served as action editor for this article.

Author Contributions

W. S. Slutske developed the study concept. W. S. Slutske and A. R. Deutsch contributed to the study design. Testing and data collection were performed by D. J. Statham and N. G. Martin. T. M. Piasecki and W. S. Slutske performed the data analysis and interpretation with the assistance of A. R. Deutsch. W. S. Slutske and T. M. Piasecki drafted the manuscript, and A. R. Deutsch, D. J. Statham, and N. G. Martin provided critical revisions. All authors approved the final version of the manuscript for submission.

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Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702618812700

Notes

1. The term *potentially causal* is used throughout this article because causality cannot be definitively established using observational data. For example, in the discordant-twin design, the underlying reason for twin discordance can never be known with certainty, thereby making it impossible to rule out alternative, noncausal explanations.

2. Because the census is conducted only every 5 years in Australia, it was not possible to perfectly align the year of the derivation of the neighborhood disadvantage index with the self-report data collection. Of the participants in this study, 18% and 53% were interviewed in 2004 and 2005 (prior to the 2006 census), 18% were interviewed in 2006 (concurrently with the census year from which the indicators of neighborhood disadvantage were obtained), and 11% were interviewed in 2007 (after the 2006 census). Fortunately, the census-derived index of neighborhood disadvantage was very stable—the neighborhood disadvantage index based on the 2006 census was correlated $r = .94$ with the disadvantage index based on the 2001 census data.

3. Data collection predated the release of the *DSM–5*. Because the *DSM–5* symptom set was identical to that of the *DSM–IV* with the exception of one eliminated symptom (committed illegal acts to finance gambling), we were able to create a *DSM–5* symptom set.

4. Because sex did not significantly interact with within-pair (interaction IRR = 1.09, 95% CI = [0.96, 1.23], $p = .176$) or between-pair (interaction IRR = 0.94, 95% CI = [0.86, 1.04],

$p = .260$) disadvantage variables in expanded base models, sex moderation of disadvantage effects was not explored.

5. The metric of the item difficulties are in z-score units from the z (standard normal) distribution. Higher difficulties indicate that the item is less likely to be endorsed and lower difficulties indicate that the item is more likely to be endorsed at a given level of a latent DG trait. The interpretation of an item difficulty of 1.0 is that the item has a 50% probability of being endorsed by an individual who is about one standard deviation above the mean on the latent DG trait; the interpretation of an item difficulty of 2.5 is that the item has a 50% probability of being endorsed by an individual who is about two and one half standard deviations above the mean on the latent DG trait.

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