All in the Family: Gene x Environment Interaction Between DRD2 and Criminal Father Is Associated With Five Antisocial Phenotypes

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Criminal Justice and Behavior 2009; 36; 1187
DOI: 10.1177/0093854809342884

ALL IN THE FAMILY

Gene × Environment Interaction Between DRD2 and Criminal Father Is Associated With Five Antisocial Phenotypes

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A range of Gene × Environment interactions is associated with antisocial phenotypes, and the evidence is clear that the etiology of antisocial behavior is strongly heritable and that environmental liabilities are important. However, the precise ways that genetic and environmental pathogens interact to predict antisocial behavior are underspecified. The present study shows that the interaction between a polymorphism in a dopamine receptor gene (DRD2) and a criminal father predicts five antisocial phenotypes among African American females ($n = 232$) in the National Longitudinal Study of Adolescent Health. Genetic risk (as measured by the A1 allele) and a criminal father interacted to predict serious and violent delinquency at Wave 1, serious and violent delinquency at Wave 2, and number of police contacts. The current investigation represents the first study to show Gene × Environment interactions in the prediction of antisocial phenotypes using criminal justice system status as an environmental pathogen.

Keywords: DRD2; delinquency; violence; family; Gene × Environment interaction; genetics

Family environments and parent–child interactions are intimately associated with externalizing and antisocial behaviors in children. A host of factors contribute to delinquency and maladaptive behaviors (Beaver & Wright, 2007; Deater-Deckard, 2003; Jaffee et al., 2005; Nagin, Pogarsky, & Farrington, 1997; Patterson, 1982; Petrill, Plomin, DeFries, & Hewitt, 2003; Sayre-McCord, 2007) and are partly responsible for the intergenerational

AUTHORS' NOTE: *This research uses data from Add Health, a program project designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris and funded by a grant (P01-HD31921) from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies. Special acknowledgment is due to Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 West Franklin Street, Chapel Hill, NC 27516-2524; e-mail: addhealth@unc.edu. No direct support was received from Grant No. P01-HD31921 for this analysis. An earlier version of this study was presented at the annual meeting of the American Society of Criminology, November 2008. Correspondence may be addressed to Matt Delisi, e-mail: delisi@iastate.edu.*

CRIMINAL JUSTICE AND BEHAVIOR, Vol. 36 No. 11, November 2009 1187-1197
DOI: 10.1177/0093854809342884

transmission of criminal behavior—factors such as family size, birth order, abuse, neglect, maltreatment, low maternal age, low parental interest in the child, weak parental supervision, family instability, chaotic home environments, child antisocial behavior, and others. As noted by Deater-Deckard (2003), "the development of externalizing problems and conduct disorder is intimately linked to problems in parent-child interaction" (p. 252).

Farrington and Welsh (2007) recently explicated the literature on family factors and crime and generated six global explanations for why offending tends to run in families. First is intergenerational continuity in exposure to multiple risk factors, in which offending is part of a larger cycle of deprivation and antisocial behavior. Second is phenotypic assortment, which argues that antisocial people choose partners who are similar to themselves in terms of antisocial attitudes, traits, and behaviors. Third is differential association, where children learn crime from parents, siblings, or both. Fourth is the idea that criminal parents place their children in environmental situations that are conducive to offending. Fifth is labeling, which occurs when criminal justice systems disproportionately target youths with criminal parents. The sixth explanation is that "the effect of a criminal parent on a child's offending is mediated by genetic mechanisms" (p. 59). Several commentators have noted either the empirical strength of the genetic basis for the crime–family link or the diverse ways that social scientists have attempted to deny it (cf. Brennan & Mednick, 1990; Fishbein, 1990; Freese, 2008; Rowe, 1986; Rowe & Osgood, 1984; Walters, 1992; Walters & White, 1989). As noted by Farrington, Barnes, and Lambert (1996), "most American criminologists were trained as sociologists and are concerned to avoid any suggestion that offending might be genetically transmitted" (p. 47).

Fortunately, scientists have generally moved beyond squabbling about nature "or" nurture bases of behavior, and contemporary research has shown that upward of 40% to 80% of the variance in various externalizing behaviors, antisocial traits, and antisocial behavior is attributable to genetic factors (Button, Scourfield, Martin, Purcell, & McGuffin, 2005; Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Frick, & Plomin, 2007; Wright, Beaver, DeLisi, & Vaughn, 2008). For instance, given data from a high-risk sample of 1,116 pairs of 5-year-old twins, Arseneault et al. (2003) examined childhood antisocial behaviors based on reports from mothers, teachers, examiner–observers, and twins' self-reports. They found, in a pervasive measure of antisocial behaviors across settings and reports, that 82% of variation was attributable to genetic factors.

Since 2003, when the mapping of the human genome was complete, scientists have identified many candidate genes that confer susceptibility to various behavioral and psychiatric problems. As such, there is great potential today to specify the mechanisms of the gene–environment interplay that underscores the family–crime relationship. As noted by Butcher and Plomin (2008),

> it is likely that the DNA differences responsible for this heritability have such small or subtle effects that even more powerful strategies will be needed to detect them. Identifying genes associated with environmental measures will be worth the effort because they will foster research on an active model of experience in which individuals select, modify, and create environments on the basis of their genetic proclivities (p. 370).

An important candidate gene for serious antisocial behavior is the dopamine receptor gene (DRD2), which codes for the D2 receptor and is found throughout the body but especially in the striatum, pituitary gland, amygdala, caudatus, putamen, and other brain regions

(Marino et al., 2004). Located at 11q22-23, DRD2 has a polymorphic TaqI restriction endonuclease site approximately 2,500 base pairs downstream (3′ untranslated region) from the coding section of the gene (Grandy et al., 1989). The site of the TaqI restriction endonuclease is referred to as the TaqIA site to keep it distinct from the TaqIB restriction site also found on the DRD2 gene. The minor TaqIA (A1) allele has a point mutation, C → T (TCGA to TTGA), that erases the TaqI site, whereas the A2 allele has the TaqI site intact. The A1 allele of the DRD2 gene is considered the risk allele and is a contributor to the reward deficiency syndrome, which typifies people who need high levels of excitement and stimulation to activate their reward system in the same capacity as those with normally functioning reward systems.

DRD2 has pleiotropic effects on a host of antisocial and maladaptive phenotypes (Noble, 2003), including antisocial personality (Ponce et al., 2003), attention-deficit/hyperactivity disorder (Bobb, Castellanos, Addington, & Rapoport, 2006; Comings et al., 2000), alcoholism (Blum et al., 1990), conduct disorder (Beaver et al., 2007), serious and violent delinquency (Guo, Roettger, & Shih, 2007), heroin dependence (Xu et al., 2004), criminal victimization (Beaver et al., 2007), and increased frequency of alcohol use (Guo, Wilhelmsen, & Hamilton, 2007).

METHOD

PARTICIPANTS AND PROCEDURES

Data are derived from the National Longitudinal Study of Adolescent Health (Add Health) that contains participants (n = 2,574) who provided buccal cells at Wave 3 for genotyping in the 3′ untranslated region of the DRD2 polymorphism is the site of TaqIA, which was genotyped as a single-nucleotide polymorphism. The analytical sample was limited to 232 African American females, for three reasons: First, no prior research has examined gene–environment interactions on antisocial phenotypes among African American females; second, focusing on one racial/sex group avoids population stratification effects; and, third, no significant effects were observed for other racial/sex groups. Geneticists working at the Institute for Behavioral Genetics at the University of Colorado originated an single-nucleotide polymorphism assay by employing Taqman© Assays by Design™ for SNP Genotyping Service (Applied Biosystem, Foster City, CA). To genotype the DRD2 TaqIA polymorphism, the following primers and probes were used (note the bold italic font in Probes 1 and 2, which indicates the point of mutation): forward primer, 5′-GTGCAGCTCACTCCATCCT-3′; reverse primer, 5′-GCAACACAGCCATCCTCAAAG-3′; Probe 1, 5′VIC-CCTGCCT***T***G ACCAGC-NFQMGB-3′; and Probe 2, 5′-FAM-CTGCCT***C***GACCAGC-NFQMGB-3′. Two independent observers scored the genotype results, where the T-probe signal corresponded to the TaqIA (A1) allele and the C-probe signal corresponded to the TaqIB (A2) allele (Haberstick & Smolen, 2005).

MEASURES

Delinquency scales. During Wave 1 interviews, respondents were asked 11 questions about their involvement in various forms of serious delinquency—for example, how frequently in the past 12 months they had (a) hurt someone badly enough to need medical

TABLE 1: Descriptive Statistics for the Antisocial Phenotype Measures

Measures	M	SD	*Skewness*
Wave 1			
Serious Delinquency Scale	1.23	2.44	3.23
Violent Delinquency Scale	0.93	1.91	3.46
Wave 2			
Serious Delinquency Scale	0.754	1.61	2.94
Violent Delinquency Scale	0.585	1.36	3.06
Police contacts	0.078	0.364	5.85

attention, (b) used or threatened to use a weapon, (c) sold drugs, and (d) taken part in a group fight. Responses for most items were coded such that 0 = *never*, 1 = *once or twice*, 2 = *three or four times*, and 3 = *five or more times*. However, two items were coded dichotomously: whether the respondent had shot or stabbed someone and whether the respondent had pulled a knife or a gun on someone (0 = *no*, 1 = *yes*). To take into account the serious nature of these items, previous researchers suggested that they be recoded such that 0 = *no* and 3 = *yes* (Guo, Roettger, et al., 2007). Responses to these 11 items were summed together to create the Wave 1 Serious Delinquency Scale ($\alpha = .67$). The same items were available at Wave 2, and thus, an identical Wave 2 Serious Delinquency Scale was created ($\alpha = .63$). Table 1 contains the descriptive statistics for the Serious Delinquency Scales and the other outcome measures employed in the analyses.

In line with prior research (Beaver, 2008; Guo, Roettger, et al., 2007), a Violent Delinquency Scale was created to examine whether genetic factors had consistent effects across various antisocial phenotypes. We developed the Violent Delinquency Scale by selecting seven items from the serious delinquency scale that tapped physical violence. For example, items pertaining to physical fighting and assault were included in the Violent Delinquency Scale. Once again, the Wave 1 Violent Delinquency Scale ($\alpha = .58$) and the Wave 2 Violent Delinquency Scale ($\alpha = .59$) comprised identical items.

Number of police contacts. To measure contact with the criminal justice system, we included a variable in the analyses indicating number of police contacts. During Wave 3 interviews, respondents were asked to indicate the number of times during their life they had been stopped and questioned by the police for something other than a minor traffic violation. Responses to this item were coded continuously, where the value indicated the total number of police of contacts.

Criminal father. Children born to parents who are criminal are likely to receive a genetic propensity for antisocial behaviors, as well as environmental risk factors that predispose them to such behaviors (Farrington & Welsh, 2007). To take this into account, we included a single-item variable that measures whether the respondent's biological father had ever been incarcerated (0 = *no*, 1 = *yes*).

ANALYSIS

The analysis for this study was conducted in a number of steps. First, prior research has revealed that genetic polymorphisms often exert their strongest effect on behavioral

phenotypes when they are paired to a criminogenic environment. As a result, we anticipated that the DRD2 polymorphism would not have a statistically significant main effect on any of the outcome measures. Instead, we hypothesized that the DRD2 polymorphism would interact with the criminal father variable to predict variation in the antisocial outcome measures. To test for this interaction, we created a multiplicative interaction term between DRD2 and criminal father. Second, this interaction term was included in five models: a model predicting each of the Serious Delinquency Scales, each of the Violent Delinquency Scales, and the police contacts variable. As Table 1 shows, all five outcome measures are highly skewed; so, all the models were estimated using negative binomial regression. All models controlled for the respondent's age (measured in years).

The DNA subsample contains some nested observations, where more than one sibling from the same household was selected for inclusion in the data. This process necessarily violates the assumption of independence in observations, which can artificially deflate standard errors and result in biased tests of statistical significance for the coefficients. We corrected for this problem in two ways. First, one twin from each monozygotic pair was randomly removed from the analytical sample (Haberstick & Smolen, 2005). Second, all the models were estimated using Huber–White standard errors.

RESULTS

As shown in Table 2, neither DRD2 nor criminal father predicted serious delinquency at Wave 1; however, their interaction did ($b = 1.00$, $z = 2.03$, $p = .043$), as displayed in Figure 1. Similar effects emerged for violent delinquency, with null effects for DRD2 and criminal father but a significant effect with their interaction at Wave 1 ($b = 1.16$, $z = 2.19$, $p = .028$; see Figure 2). Longitudinal effects were assessed for serious delinquency and violent delinquency occurring at Wave 2. As presented in Table 3 and displayed in Figure 3, the interaction between DRD2 and criminal father moderately predicts serious delinquency ($b = 1.35$, $z = 1.73$, $p = .084$).

For violent delinquency at Wave 2, independent effects ($b = .571$, $z = 2.61$, $p = .009$) and interactive genetic effects ($b = 2.45$, $z = 5.39$, $p = .000$) emerged. Figure 4 presents the interaction between DRD2 and criminal father. Finally, as shown in Table 4, DRD2 significantly predicted number of police contacts ($b = .863$, $z = 2.13$, $p = .033$), as did the interaction between DRD2 and criminal father ($b = 2.25$, $z = 2.04$, $p = .042$), which is shown in Figure 5.

DISCUSSION

Before delving into the discussion, we should address two important limitations of the current study. First, prior reports of Gene × Environment interactions for antisocial phenotypes have not been uniformly replicated across various samples (cf. Blum et al., 1990; Blum et al., 1995; Caspi et al., 2002; Gelernter, Goldman, & Risch, 1993; Huizinga et al., 2006; Kim-Cohen et al., 2006); thus, it is critical that additional research attempt to replicate the current findings. Second and concomitantly, the current findings are limited to

TABLE 2: Independent and Interactive Effects of DRD2 and Criminal Father on Wave 1 Serious and Violent Delinquency

	Wave 1 Serious Delinquency				*Wave 1 Violent Delinquency*			
Measures	b	SE	z	p	b	SE	z	p
DRD2	–0.075	.17	–0.44	.661	0.123	.18	0.68	.499
Criminal father	–0.143	.38	–0.38	.706	–0.358	.41	–0.88	.379
DRD2 × Criminal Father	1.00	.49	2.03	.043	1.16	.53	2.19	.028
Age	–0.158	.08	–1.88	.061	–0.153	.09	–1.78	.075

Note. Huber–White standard errors.

TABLE 3: Independent and Interactive Effects of DRD2 and Criminal Father on Wave 2 Serious and Violent Delinquency

	Wave 2 Serious Delinquency				*Wave 2 Violent Delinquency*			
Measures	b	SE	z	p	b	SE	z	p
DRD2	0.261	.25	1.04	.298	0.571	.22	2.61	.009
Criminal father	–1.25	.56	–2.22	.026	–1.75	.46	–3.78	.000
DRD2 × Criminal Father	1.35	.78	1.73	.084	2.45	.45	5.39	.000
Age	–0.249	.10	–2.40	.022	–0.280	.11	–2.61	.009

Note. Huber–White standard errors.

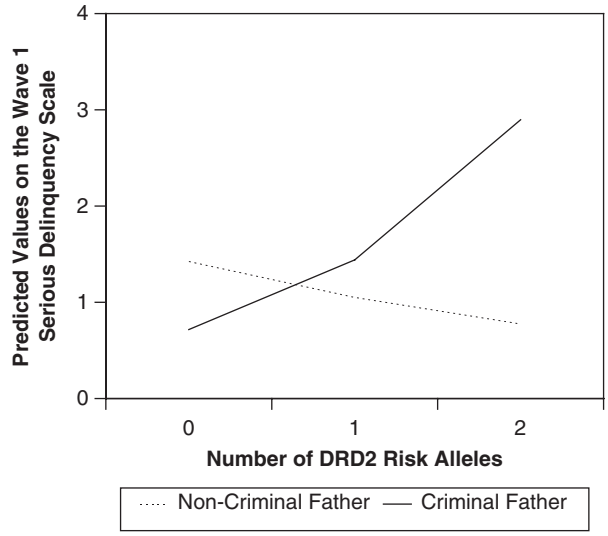

Figure 1: Having a Criminal Father Moderates the Effect of DRD2 on Wave 1 Serious Delinquency

African American females (n = 232) selected from the Add Health data. Null findings (not shown) were found for African American males, Caucasian males, and Caucasian females. To increase the generalizeability, additional studies with diverse racial, ethnic, and gender groups are needed to further specify the molecular and environmental bases that indicate that crime runs in families.

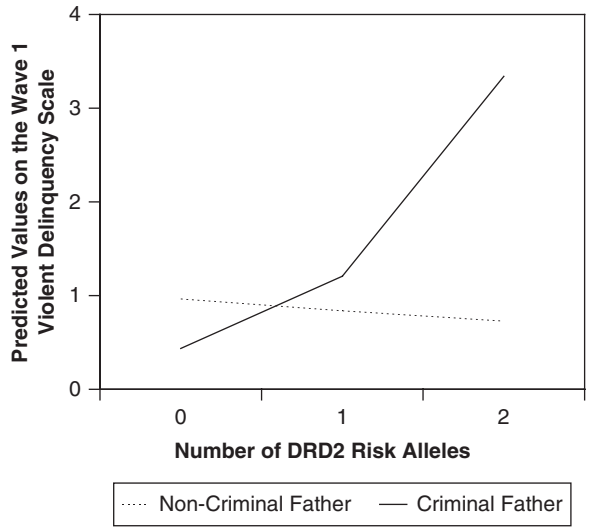

Figure 2: Having a Criminal Father Moderates the Effect of DRD2 on Wave 1 Violent Delinquency

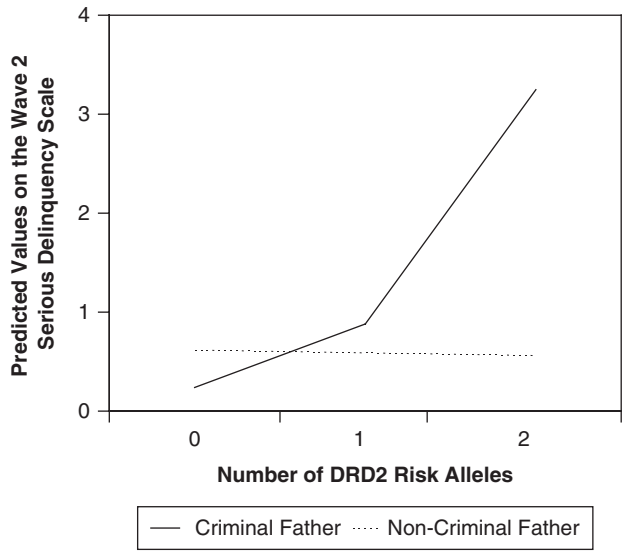

Figure 3: Having a Criminal Father Moderates the Effect of DRD2 on Wave 2 Serious Delinquency

Despite these limitations, the current effort is the first criminological study to our knowledge to detect a Gene × Environment interaction in the prediction of antisocial phenotypes using criminal justice status as an environmental pathogen. Data from Add Health indicate that African American females with (a) risk alleles for a polymorphism in the DRD2 gene and (b) criminal fathers were significantly at risk for antisocial behavior in terms of serious delinquency, violent delinquency, life-course-persistent offender status, and police contacts.

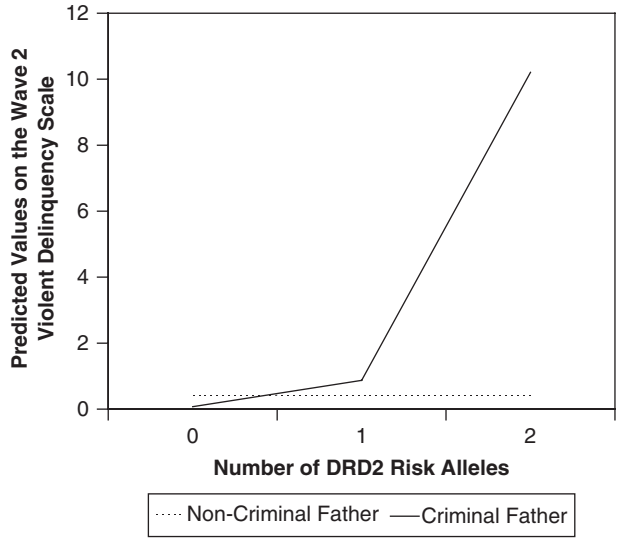

Figure 4: Having a Criminal Father Moderates the Effect of DRD2 on Wave 2 Violent Delinquency

TABLE 4: Independent and Interactive Effects of DRD2 and Criminal Father on Number of Police Contacts

	Number of Police Contacts			
Measures	b	SE	z	p
DRD2	0.863	0.40	2.13	.033
Criminal father	–1.71	1.25	–1.37	.171
DRD2 × Criminal Father	2.25	1.11	2.04	.042
Age	0.082	0.21	0.39	.694

Note. Huber–White standard errors.

These effects were consistently replicated across waves and across measures. The current findings add to a burgeoning literature that has shown Gene × Environment interactions between DRD2 and diverse environmental conditions—such as delinquent peer networks, religious beliefs, family risk, martial status, and marital stability—in the prediction of diverse phenotypic outcomes, including victimization, violent delinquency, early onset offending, and attention-deficit/hyperactivity disorder (see Beaver, 2009, p. 96).

Why does DRD2 interact with criminal father to robustly predict antisocial conduct? One speculation is that the DRD2 × Criminal Father interaction can be regarded as a homozygous disadvantageous state whereby exposure to a criminal father and to residual ecological pathogens express the underlying liability to aggressive antisocial behavior (Guo et al., 2007). Another interpretation is that respondents in the current analytical sample suffer from what Jaffee, Moffitt, Caspi, and Taylor (2003) refer to as a double whammy, in that genetic risk (e.g., TaqIA polymorphism in the DRD2 gene) and environmental pathogen (e.g., having a criminal father) are transmitted from father to daughter. Jaffee et al.

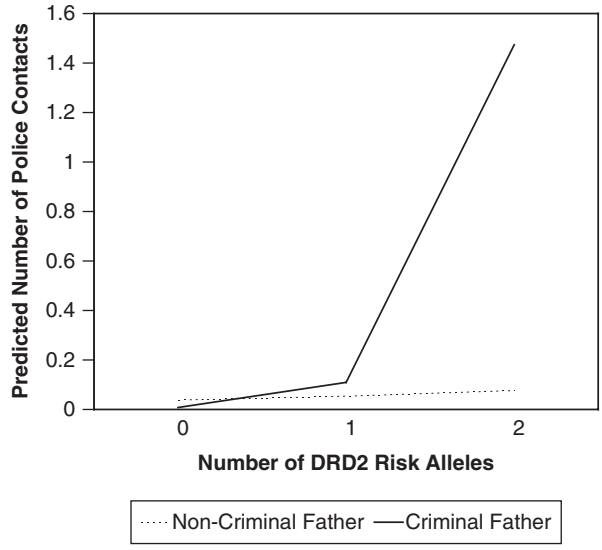

Figure 5: Having a Criminal Father Moderates the Effect of DRD2 on Number of Police Contacts

found that children with criminal fathers were at increased risk for conduct disorder and that risk was amplified when children spent more time with their fathers.

Research on the intergenerational transmission of antisocial behavior suggests a clustering of criminal and analogous forms of behavior within families but, traditionally, without specifying how heredity and environmental conditions interact to predict crime (Farrington & Welsh, 2007). In contrast, behavioral and molecular genetics research is increasingly demonstrating the profound importance of genes and gene–environmental interactions to the family–crime link (Arseneault et al., 2003; Butcher & Plomin, 2008; Deater-Deckard, 2003; Jaffee et al., 2003; Jaffee et al. 2005; Wright & Beaver, 2005; Wright et al., 2008). The current study is the first criminological study to utilize criminal justice status—that is, having a criminal father—as an environmental pathogen that interacts with a measured genetic polymorphism to predict antisocial phenotypes.

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