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The role of gene–gene interaction in the prediction of criminal behavior

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Abstract

A host of research has examined the possibility that environmental risk factors might condition the influence of genes on various outcomes. Less research, however, has been aimed at exploring the possibility that genetic factors might interact to impact the emergence of human traits. Even fewer studies exist examining the interaction of genes in the prediction of behavioral outcomes. The current study expands this body of research by testing the interaction between genes involved in neural transmission. Our findings suggest that certain dopamine genes interact to increase the odds of criminogenic outcomes in a national sample of Americans.

Given the complexities of behavior, especially violence and aggression, most scholars have long recognized the importance of, and need for, multifaceted explanations – including both biological and environmental factors – when predicting behavioral phenotypes [8,14,32,33,40]. Judging from one extant body of research, for example, there appears to be mounting evidence suggesting that gene–environment interactions – whereby genetic risk factors moderate (or are moderated by) environmental pathogens – are relevant for understanding the etiology of psychopathology [40,53]. Questions remain, though, regarding the chance nature of these findings as well as the ability to replicate earlier results consistently using independent data sources [32,48]. Despite some uncertainty, scholars continue investing tremendous amounts of time and effort testing for the presence of gene–environment interactions [2,28,29].

Though gene–environment interaction studies are now relatively commonplace, the possibility that the influence of genes on human outcomes – ranging from diseases to behavior – may depend on other genes through a complicated arrangement of gene–gene interactions is only recently beginning to gain traction [13,46,51]. What should be considered, moreover, is that failure to detect the influence of gene–gene interactions may bias findings from studies intended to detect gene–environment interactions [26]. Put differently, the presence of epistasis (i.e., an interaction between genetic loci) might underlie both the positive and the even null findings of studies attempting to isolate the presence of gene–environment interactions [58].

The examination of gene–gene interactions becomes all the more important given that, despite linkages with a variety of psychopathologies [51], evidence regarding the role of gene–gene interactions in the prediction of overt criminal behaviors is less well established. Beaver et al. [3] uncovered evidence that a gene–gene interaction predicted variation in antisocial behaviors among subjects drawn from the National Longitudinal Study of Adolescent Health (Add Health). Given the general lack of evidence in this area, however, efforts are needed to replicate this finding using independent samples. The current study represents a step in this direction by offering an analysis of gene–gene interactions in the prediction of antisocial behavior among an independent sample of respondents drawn from the National Youth Survey Family Study (NYSFS).

1. Dopamine and the neurochemical origins of antisocial behavior

Most studies examining polymorphisms related to antisocial behaviors have centered on genes involved in

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brain functioning, especially the task of neurotransmission [4,15,17,36,40]. Genes associated with neurological functioning (and brain structure) have elicited tremendous scrutiny and the results, to this point, appear somewhat promising [10,41]. Functional polymorphisms in the dopaminergic system, specifically, have been linked to various pathological outcomes ranging from fairly benign incarnations such as gambling [12], approach/avoidance behaviors and novelty seeking [20,21] to more adverse outcomes like addiction [6]. Two genes in particular, the D2 receptor polymorphism (DRD2) and the D4 receptor polymorphism (DRD4), appear to offer insight concerning the link between variation at the molecular level and behavioral variation at the phenotypic level ([23,38,49]; however, see also [31,34,45]).

The human dopaminergic system regulates a number of physiological processes ranging from motor coordination to functioning in the reward centers of the brain [34,35,57]. Located on the long arm of the eleventh chromosome (11q22-23) [22,30,42] DRD2 is a functional polymorphism that encodes for the production of the dopamine receptor D2. The *TaqI*A restriction fragment length polymorphism, to be specific, corresponds to two alleles, A1 and A2. These alleles are of great interest given their capacity to yield altered production of D2 receptors [43,44]. The A1 allele of DRD2 has been linked to reduced D2 receptor binding across the striatum [54], coupled also with a general reduction in the density of the D2 receptors[1] [44]. DRD4, the gene responsible for encoding the D4 receptor, is located along the short arm of chromosome 11 (11p.15.5) [37]. The DRD4 gene contains a 48 base pair (bp) sequence which is repeated between two to eleven times [9]. The 4-repeat allele is quite common (in terms of global representation), with the 7-repeat allele following in a close second ([9]; for additional information regarding differences in protein structures, see also [52,55]).

2. DRD2, DRD4, and antisocial behavior

A growing body of extant research has linked both the A1 allele of DRD2 and the 7-repeat allele of DRD4 to a variety of antisocial outcomes. The A1 allele, for example, correlates with impulsive behaviors such as gambling [11], deleterious outcomes such as substance abuse [5], as well as overt forms of criminal behavior [3] and even violent victimization [56]. Boutwell and Beaver [7] uncovered evidence that individuals homozygous for the A2 allele on DRD2 were overrepresented in subjects who abstained entirely from criminal behavior. In other words, the A2 allele may either insulate against antisocial behavior or fail to correlate with behavior entirely, while the A1 allele corresponds to a diminished probability of abstaining behavior. Studies examining the 7-repeat allele of DRD4 report similar relationships [7]. This becomes all the more interesting given that the 7-repeat allele of DRD4 has been linked to increased novelty seeking in both human and non-human animals [21], as well as overt displays of aggression and violence across species that carry homologous genes (for a general review see [57]).

Emergent evidence suggests that the interaction between the 7-repeat allele of DRD4 and the A1 allele of DRD2 (i.e., a gene–gene interaction) may increase the likelihood of pathological and antisocial behaviors above and beyond the main effects exhibited by the individual polymorphisms [18]. Prior research has demonstrated such an interaction in the prediction of criminal behavior. For example, Beaver and colleagues [3] examined the interaction of DRD2 and DRD4 in the prediction of antisocial conduct using participants drawn from the Add Health. The findings revealed evidence that DRD2 and DRD4 interacted to significantly increase conduct problems—respondents carrying the A1 allele on DRD2 and the 7-repeat allele on DRD4 had higher probabilities of antisocial behavior compared to all other respondents. To date, there has been no effort, of which we are aware, to replicate this research. The current study, then, is intended to examine the findings uncovered by Beaver et al. [3] using an independent sample of males also residing in the United States.

3. Methods

In order to test whether a gene–gene interaction was implicated in the origins of criminogenic behaviors, we examined subjects drawn from the National Youth Survey Family Study (NYSFS) (Table 1.). The NYSFS constitutes a national sample of youth residing in the United States [25]. Detailed explanation of sampling procedures and overall sample composition have been outlined elsewhere [19,25,39]. Briefly, data collection was initiated in 1976 and included 1725 youth between the ages of 11 and 17. Currently, the NYSFS has collected 11 waves of data from the original respondents. During wave 10 (in the year 2002)

Table 1
Descriptive statistics for NYSFS males.

	Mean	SD	Min	Max
DRD2	0.44	0.61	0	2
DRD4	0.38	0.57	0	2
Major Theft	0.09	0.28	0	1
Burglary	0.14	0.34	0	1
Gang Fight	0.05	0.23	0	1
Physical Assault	0.11	0.31	0	1
Composite Measure	0.27	0.45	0	1
Age	40.38	2.06	36	45
Race	0.79	0.40	0	1

[1] As Neville and colleagues [42] note, the *Taq*I1A polymorphism corresponds to a substituted amino acid downstream of DRD2 and located within the ANKK1 gene. As such, this location might also provide a pathway and explanation for the association between this particular gene and various antisocial phenotypes.