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Exploring the association between the 2-repeat allele of the MAOA gene promoter polymorphism and psychopathic personality traits, arrests, incarceration, and lifetime antisocial behavior

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ABSTRACT

A line of research has revealed that a polymorphism in the promoter region of the MAOA gene is related to antisocial phenotypes. Most of these studies examine the effects of low MAOA activity alleles (2-repeat and 3-repeat alleles) against the effects of high MAOA activity alleles (3.5-repeat, 4-repeat, and sometimes 5-repeat alleles), with research indicating that the low MAOA activity alleles confer an increased risk to antisocial phenotypes. The current study examined whether the 2-repeat allele, which has been shown to be functionally different from the 3-repeat allele, was associated with a range of antisocial phenotypes in a sample of males drawn from the National Longitudinal Study of Adolescent Health. Analyses revealed that African-American males who carried the 2-repeat allele were, in comparison with other African-American male genotypes, significantly more likely to be arrested and incarcerated. Additional analyses revealed that African-American male carriers of the 2-repeat allele scored significantly higher on an antisocial phenotype index and on measures assessing involvement in violent behaviors over the life course. There was not any association between the 2-repeat allele and a continuously measured psychopathic personality traits scale. The effects of the 2-repeat allele could not be examined in Caucasian males because only 0.1% carried it.

1. Introduction

A significant amount of behavioral genetic research has examined the genetic basis to antisocial behaviors (Moffitt, 2005). The results of these studies, best summarized by a number of meta-analyses, indicate that approximately 50% of the variance in measures of antisocial phenotypes is attributable to genetic factors (Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002). More recent research has begun to investigate genetic polymorphisms that might be partially responsible for producing variation in antisocial phenotypes (Caspi et al., 2002). Genes involved in neurotransmission have been identified as the most promising candidate genes for antisocial behaviors and traits (Ferguson & Beaver, 2009). Although studies have identified an association between polymorphisms in a number of neurotransmission genes and various antisocial behaviors, these associations are often plagued by the inability for follow-up studies to replicate the original findings. An important exception to the replication problem appears to be for a functional polymorphism in the monoamine oxidase A (MAOA) gene.

The MAOA gene has been mapped to the X chromosome at location Xp11.23–11.4 (Levy et al., 1989) and codes for the production of the MAOA enzyme that catabolizes certain neurotransmitters, such as dopamine and serotonin (Shih, Chen, & Ridd, 1999). The MAOA gene has a polymorphism in the promoter region that is the result of a 30 base pair (bp) variable number of tandem repeats (VNTR) in the regulatory region of the gene. This polymorphism has been shown to be functional as different alleles correspond to the production of MAOA enzymes with different activity levels (Sabol, Hus, & Hamer, 1998). Recognizing differences in transcriptional efficiency, researchers commonly pool the alleles into two groups: those that correspond to low MAOA activity and those that correspond to high MAOA functioning. In most studies, the 2-repeat allele and the 3-repeat allele are pooled together to form the low MAOA activity genotype and the 3.5-repeat allele, 4-repeat

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allele, and 5-repeat allele are pooled together to form the high MAOA activity genotype (Caspi et al., 2002).

The polymorphism in the promoter region of the MAOA gene has been the source of a considerable amount of research examining whether different alleles are associated with antisocial phenotypes. In a landmark study, Caspi et al. (2002) reported a link between low MAOA activity alleles and antisocial behaviors, but only among males who had been maltreated in childhood. The results of a meta-analysis seemed to confirm the association between MAOA and antisocial outcomes for maltreated males (Kim-Cohen et al., 2006). Although most of this research has revealed that MAOA only has significant effects when paired to a criminogenic environment, there is some evidence to indicate that the low MAOA activity alleles may have effects independent of environmental factors for some antisocial behaviors (Beaver, DeLisi, Vaughn, & Barnes, 2010).

Guo and his colleagues (2008) provided evidence that the MAOA gene is related to delinquent behavior in a sample of adolescents and young adults independent of environmental factors. Unlike the vast majority of research examining the effects of the MAOA gene, Guo et al. examined the effects of the 2-repeat allele against the effects of the 3-repeat allele and 5-repeat allele and against the effects of the 3.5-repeat allele and the 4-repeat allele. The results of their analysis indicated that carriers of the 2-repeat allele were at a statistically significant greater risk for engaging in serious and violent delinquency in adolescence and early adulthood. The effects were particularly marked for males. Guo et al. also conducted a functional analysis of the alleles and found that the 2-repeat allele, in comparison with 3-repeat and 4-repeat alleles, had the lowest level of promoter activity.

The results of the study by Guo et al. suggest that pooling together the 2-repeat and 3-repeat alleles may be incorrect and that the 2-repeat allele should be examined in isolation because of its functional significance. Besides this single study, though, research has yet to fully explore this possibility and thus whether the 2-repeat allele is truly a marker for antisocial phenotypes remains to be determined. The current study examines this possibility by testing for an association between the 2-repeat allele and psychopathic personality traits, the odds of being arrested, the odds of being incarcerated, and lifetime antisocial behavior in a sample of American males.

2. Materials and methods

2.1. Participants

Data for this study were drawn from the National Longitudinal Study of Adolescent Health (Add Health; Harris, 2009). The Add Health is a four-wave prospective study of a nationally representative sample of American youth who were enrolled in middle or high school in 1994–1995. The first ($N = 20{,}745$) and second ($N = 14{,}738$) waves of data were collected when most of the respondents were adolescents. The third wave of data was collected in 2001–2002 when the subjects were young adults ($N = 15{,}197$). The fourth wave of data was collected in 2007–2008 when the subjects were 24–32 years of age ($N = 15{,}701$). More details about the data can be gathered by consulting previously published reports (Harris, Tucker Halpern, Smolen, & Haberstick, 2006; Harris et al., 2003; Resnick et al., 1997).

At wave 3, a subsample of subjects was genotyped for the MAOA-uVNTR. Respondents who had a sibling who was also participating in the Add Health study were eligible for inclusion in the DNA subsample. In total, 2574 subjects submitted usable buccal cells that were genotyped, making the Add Health one of the largest samples in the world that includes genotypic and phenotypic information. Genotyping was carried out in a coordinated effort between the Add Health team and the Institute of Behavioral Genetics in Boulder, Colorado (Harris et al., 2006). As discussed below, the analytical sample was based on *N*s ranging between 167 and 174 African-American males. In the statistical analyses, missing cases were removed using listwise deletion techniques.

2.2. Genotyping procedures

Subjects were genotyped for the MAOA-uVNTR polymorphism using a variant of the assay developed previously (Sabol et al., 1998). Primer sequences were as follows: forward, 5′-ACAGCCTGACCGTGGAGAAG-3′ (fluorescently labeled), and reverse, 5′-GAACGTGACGCTCCATTCGGA-3′. This assay produced PCR products of 291 (2-repeat allele), 321 (3-repeat allele), 336 (3.5-repeat allele), 351 (4-repeat allele), and 381 (5-repeat allele) base pairs. The genotypes were scored independently by two different raters. Subjects with the 2-repeat allele were placed into one group and subjects with the 3-repeat, 3.5-repeat, 4-repeat, and 5-repeat alleles were pooled together into another group. Because most research examining the effects of MAOA has focused only on males and because MAOA is X-linked, the current study includes only males in the analytical sample.

2.3. Measures

Four main outcome measures were employed in the current study. The first outcome measure was a psychopathic personality traits scale. Prior researchers analyzing these data have developed a 23-item five-factor model-based psychopathic personality traits scale (Beaver, Barnes, May, & Schwartz, 2011) based on wave 4 data, where higher values represent more psychopathic personality traits. This same scale was used in the current study (Cronbach's $\alpha = 0.81$). In addition, two criminal justice measures were also included as outcome variables. First, respondents were asked at wave 4 whether they had ever been arrested during their life (0 = no, 1 = yes). Second, respondents were asked at wave 4 whether they had ever been incarcerated during their life (0 = no, 1 = yes). The last outcome measure was a composite antisocial phenotype index that was constructed by combining scores on the psychopathic personality traits scale, the arrest measure, and the incarceration measures. Specifically, respondents who scored 1.5 standard deviations above the mean on the psychopathic personality traits scale were assigned a value of one (1) and all other scores were assigned a value of zero (0). Then scores on the dichotomized psychopathic personality traits variable were summed together with the binary arrest variable and the binary incarceration variable, which produced a composite antisocial phenotype with scores ranging between zero (0) and three (3).

Last, a dichotomous race variable was included in the analyses. At wave 1, interviewers were asked to indicate the race that best describes each respondent. The data were initially analyzed using respondents who were characterized as being either Caucasian or African-American.

3. Findings

Since prior research has revealed that the distribution of the 2-repeat allele varies by race (e.g., Reti et al., 2011; Widom & Brzustowicz, 2006), the analysis begins by examining the allelic distributions by race. As Table 1 shows, the 2-repeat allele was carried by 0.1% of Caucasian males and by 5.2% of African-American males. To check the consistency of these estimates, all of the analyses were recalculated using self-reported race instead of