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Examining the association between MAOA genotype and incarceration, anger and hostility: The moderating influences of risk and protective factors

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ABSTRACT

Findings from molecular genetic research have indicated that a polymorphism in the promoter region of the MAOA gene interacts with environmental liabilities to predict antisocial phenotypes. We use these findings as a springboard to examine whether a global protective-risk factor index moderates the effect of MAOA genotype on the probability of being incarcerated and on a measure of anger and hostility. Analysis of data from the National Longitudinal Study of Adolescent Health (Add Health) indicates that exposure to risk and protective factors in adolescence are able to moderate the effect of MAOA genotype on anger and hostility in adulthood for males. The results in relation to the probability of being incarcerated were consistently null.

1. Introduction

A long line of behavioral genetic research has examined the genetic and environmental underpinnings to virtually every measurable antisocial behavior. The results of these studies, which are based on thousands of kinship pairs, collected in different geographical regions, and at different time periods, have converged to reveal that approximately 50% of the variance in antisocial behaviors is attributable to genetic factors (Moffitt, 2005; Rhee & Waldman, 2002). As a result, there has been increasing interest in moving away from only decomposing phenotypic variance and instead focusing on identifying the specific genetic polymorphisms that are involved in explaining variance in antisocial behaviors. Extant research has indicated that the genes and gene systems that are most likely to contribute to antisocial behaviors are those that are involved in neurotransmission (Ferguson & Beaver, 2009).

Of all the genes that have been studied in relation to antisocial phenotypes, the monoamine oxidase A (MAOA) gene has produced the most consistent results. The MAOA gene is located on the X chromosome (Xp11.23-11.4) and is responsible for encoding the MAOA enzyme which degrades neurotransmitters, such as serotonin, dopamine, and norepinephrine. The MAOA gene has a polymorphism (MAOA-uVNTR) that is the result of a 30-base-pair (bp) variable number of tandem repeats upstream in the 5′ regulatory region of the gene. This polymorphism has been shown to affect the functioning of the MAOA enzyme with some of the alleles encoding a low activity MAOA enzyme and others encoding a high activity MAOA enzyme. Genotyping MAOA via PCR typically produces the following five fragment sizes: 2 repeats (2R), 3 repeats (3R), 3.5 repeats (3.5R), 4 repeats (4R), and 5 repeats (5R). A general consensus has been reached in that the 2R and 3R alleles correspond to low MAOA activity, while the 3.5R and 4R alleles correspond to high MAOA activity. The 5R allele, however, has been shown to produce both low MAOA activity (Sabol, Hus, & Hamer, 1998) and high MAOA activity (Deckert et al., 1999).

Human genetic research has examined the direct association between MAOA genotypes and antisocial behaviors, revealing that the alleles that encode the low activity MAOA enzyme confer an increased risk to antisocial phenotypes. For example, the low MAOA activity alleles have been linked to delinquent behavior in adolescents and young adults (Guo, Ou, Roettger, & Shih, 2008) as well as more serious types of violence, such as weapon use and gang membership (Beaver, DeLisi, Vaughn, & Barnes, 2010). While studies have documented the main effects that MAOA might have on violence and aggression, the most consistent evidence linking MAOA genotype to antisocial phenotypes comes from research examining gene-environment interactions. The logic underlying this line of inquiry suggests that MAOA genotype only maintains an association with antisocial phenotypes in the presence of an environmental pathogen. In the first study that tested this possibility, Caspi et al. (2002) examined the interrelationships among MAOA genotype, childhood maltreatment, and violence in a sample of males from New Zealand. Their analysis revealed that MAOA genotype was unrelated to violence for the entire sample. However, they found that MAOA genotype explained a significant amount of

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variance in violence solely among males who had been maltreated as children; MAOA was unrelated to violence for males without a history of maltreatment. Importantly, a relatively recent meta-analysis of the studies examining the MAOA-maltreatment interaction found this interaction to be statistically significant across studies (Kim-Cohen et al., 2006).

The link between MAOA genotype and antisocial behaviors and between the MAOA-maltreatment interaction and antisocial behaviors is highly complex and likely involves a long chain of intermediary processes and phenotypes. Known as endophenotypes, these intermediary processes/phenotypes are thought to fall somewhere between genotype and phenotype and are decidedly easier to identify in genetic association studies (Gottesman & Gould, 2003). Imaging genomics has provided evidence that certain neurobiological functions and structures may be endophenotypes that partially explain the association between MAOA genotype and antisocial behaviors. For example, carriers of the low MAOA activity alleles have been shown to have reduced limbic volume, amygdala hyperresponsivity, reduced prefrontal cortex reactivity, and structural changes to the orbitofrontal cortex (Meyer-Lindenberg et al., 2006). All of these neurobiological markers have been found to be, or have been posited to be, related to antisocial behaviors (Viding & Frith, 2006).

Of particular importance is that genomic-imaging research has also drawn attention to the potential for certain personality traits to be endophenotypes in the MAOA-antisocial behaviors association (Alia-Klein et al., 2009). Buckholtz and Meyer-Lindenberg (2008), for instance, showed that some of the neurobiological endophenotypes mentioned previously are associated with higher scores on the personality trait anger and hostility. Even more applicable to the current research are the studies that have examined the association between MAOA genotype and antisocial personality traits. Williams et al. (2009) found, for example, that carriers of the low MAOA activity alleles, in comparison with carriers of the high MAOA activity alleles, scored significantly higher on measures of antisocial personality traits. Similar results were reported by Yang et al. (2007) in their analysis of Korean women. Importantly, however, not all studies have detected an association between MAOA genotype and antisocial personality traits (Koller, Bondy, Preuss, Bottlender, & Soyka, 2003). In general, studies investigating the nexus between MAOA and antisocial personality traits have failed to test for the role of moderating factors. As a result, heterogeneity in these study findings could be the result of differential exposure to risk and/or protective factors, a possibility that has not been fully explored to date.

The current study builds off and extends previous research in three important ways. First, consistent with prior research, we examine whether MAOA interacts with certain factors to predict involvement in serious criminal behavior. Second, unlike existing studies, we do not focus on maltreatment as the moderating variable, but instead employ a protective-risk factor index. This index measures exposure to protective and risk factors as a continuum ranging from heavy exposure to protective factors to heavy exposure to risk factors. In this way, we are able to examine whether the presence of protective factors is able to blunt the effects of MAOA and whether the presence of risk factors is able to exacerbate the effects of MAOA (Belsky & Pluess, 2009). Moreover, this protective-risk factor index includes more than only social-environmental factors; instead, it is much more global and examines an array of environmental- and individual-level factors. As a result, we are able to explore the possibility that individual-level characteristics, such as verbal abilities, interact with genotype to affect phenotypic outcomes. Third, instead of only using a measure of antisocial behavior as the outcome of interest, we also employ a measure of anger and hostility. Because anger and hostility has previously been linked to antisocial behaviors (Gullone & Moore, 2000; Samuels et al., 2004), we propose that anger and hostility could be an intermediary phenotype that explains part of the mechanisms by which MAOA interacts with the environment to predict antisocial behaviors. We test these issues by analyzing genotypic and phenotypic data drawn from a longitudinal sample of American youths and adults.

2. Materials and methods

2.1. Participants

Participants for this study were drawn from the National Longitudinal Study of Adolescent Health (Add Health; Udry, 2003). The Add Health is a longitudinal and nationally representative sample of American youths who were enrolled in middle or high school during the 1994–1995 school year. Four waves of data have been collected thus far. The first wave of data was comprised of two different components: the wave 1 in-school survey and the wave 1 in-home survey. The wave 1 in-school survey was administered to more than 90,000 youths while they were at school. To gain in-depth information about some of the adolescents, a subsample of youths was selected to be re-interviewed at their home along with their primary caregiver. A total of 20,745 adolescents and 17,700 of their primary caregivers participated in the wave 1 in-home component of the Add Health study. About one to 2 years after the wave 1 data were collected, the second round of surveys was administered. Overall, 14,738 adolescents were included in the wave 2 of the Add Health data. Subsequently, between 2001 and 2002, the third wave of data was collected from 15,197 participants. The fourth and final round of surveys was distributed between 2007 and 2008 when most of the respondents were 24–32 years old. A total of 15,701 respondents participated in the wave 4 component of the Add Health study.

During wave 3 data collection, a subset of respondents was asked to submit samples of their buccal cells for genotyping. Respondents who had a sibling who was also participating in the Add Health study were eligible to participate. Overall, more than 2500 subjects submitted usable samples of their DNA, making the Add Health one of the largest samples in the world to include genotypic data (Harris, Halpern, Smolen, & Haberstick, 2006). After removing cases because of attrition and missing data via listwise deletion, the final analytical sample ranged between $N = 420$ and 493.

2.2. Genotyping

Add Health participants were genotyped for the MAOA-uVNTR polymorphism using a variant of a previously developed assay (Sabol et al., 1998). DNA amplification was achieved by using the following primer sequences: forward, 5′ACAGCCTGACCG-TGGA GAAG-3′ (fluorescently labeled), and reverse, 5′-GAACGTGACGCTC CATTCGGA-3′. This assay resulted in the 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. Each genotype was scored by two independent raters.

Following previous researchers analyzing the Add Health data (Haberstick et al., 2005), alleles of the MAOA gene were pooled to form two groups: a low MAOA activity group and a high MAOA activity group. The low MAOA activity group was created by pooling together the 2-repeat allele and the 3-repeat allele. The high MAOA activity group was created by pooling together the 3.5-repeat allele, the 4-repeat allele, and the 5-repeat allele. With this coding strategy employed, 17.8% of females were homozygous for the low MAOA activity allele, 43.0% were heterozygous, and 39.1% were homozygous for the high MAOA activity allele. For males, who have only one MAOA allele, 40.2% possessed the low