MAOA genotype and longitudinal delinquency among males in the United States: the moderating role of parental incarceration and parental closeness

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Abstract

The similarity of patterns of delinquency among parents and children has been noted in decades of research. Unfortunately, data limitations and the complex nature of genetic inheritance from parents to children have made it difficult to identify the source of familial patterns of delinquency as genetic or environmental. Using data from the National Longitudinal Study of Adolescent Health, we address this issue by making use of the novel transmission of the 2R allele in the MAOA gene with detailed information about delinquent behaviors among males paired with data on their biological mother and father's incarceration histories. As with previous research, we show an increased risk of delinquent behaviors among carriers of the 2R allele compared to those without this allele. Importantly, this association is completely independent of maternal and paternal incarceration histories eliminating the possibility of passive gene-environment correlation. This association is also consistent regardless of parental incarceration history (e.g., no evidence for a social trigger gene-environment interaction) but the risk of the 2R allele is significantly reduced for those who report a close relationship with their fathers (e.g., evidence for the social control gene-environment model). Taken together, these results suggest that parental behaviors and environments associated with parental incarceration do not moderate the influence of 2R on delinquency, while lack of father closeness does. We discuss the importance of considering social and familial contexts (mass incarceration, rise of single-parent households) when considering gene-environment interactions predicting delinquency in the context of modern U.S. society.

Introduction

Researchers have long noted that delinquent behaviors and criminal histories are concentrated within families (Robinson 1932; Robbins 1966; Rowe and Farrington 1996; Sampson and Laub 1993; Thornberry 2005). Ellis (1982) suggests that roughly 40% of those involved in delinquent behaviors have least one parent that has a criminal history. The comparison of delinquent behaviors among siblings and twins suggests that some of the family patterns of delinquency may be due to genetic factors shared by family members (Dilalla and Gottesman 1989; Wright 2008). However, given that family members share environmental factors and have a common genetic architecture, it is often difficult to identify the source of familial patterns of problem behavior as genetically or environmentally situated (Rowe and Farrington 1996; Farrington et al 2007).

Recent evidence suggests that a rare polymorphism within a gene linked to the production of the enzyme monoamine oxidase (the 2R variant of the MAO-A gene) is associated with an increased risk of delinquent behavior among males (Guo et al 2008a, Guo et al 2008b). This risky allele is notable because it is located on the X chromosome. Therefore, males (who are XY) with the 2R allele must have inherited it from their biological mother, while women inherit copies from the biological father and biological mother (Eisenberger et al 2007). Thus, unlike most genes associated with delinquency, such as DAT1 and DRD2, where a child may inherit a risk allele from either biological parent, the MAOA gene is hemizygote among males, thus we may evaluate how the link between maternal and child delinquency is due to this risky allele and the source of the 2R-deliqeuncy association -- delinquency associated directly with the 2R allele would be transmitted through the environment by fathers and by some combination of genes and environment from mothers.

The simultaneous measurement of both parents and children is notably lacking in the study of intergenerational delinquency (Thornberry 2009; Wakefield and Wildeman 2011). This is further confounded when you consider the lack of any study with *both* genetic *and* behavioral data from *both* parents *and* children. Identity by descent is the gold standard of genetic association studies and a pure assessment of this status is only possible with genetic information from both parents and the child (e.g., a trio). Therefore matching genetic information about each parent and their delinquent behaviors with the behaviors of their

offspring is very difficult. While datasets such the Framingham Heart Study have examined three generations to examine links between genetic traits and health outcomes, the lack of comparable multiple-generation studies in criminology make studying intergenerational transmission of delinquency quite rare, making the study of the transmission of genetic and environmental sources of delinquency beyond the scope of current research (Farrington, Coid & Murray 2009). For genes which have been associated with antisocial behavior, such as dopaminergic transporters and receptors, teasing out the role of genetic propensities in intergenerational delinquency is difficult when genetic and behavioral information is not known about both the mother and father.

While lacking the 'gold standard' of parent and child genes/behaviors, the National Longitudinal Study of Adolescent Health is one of the only existing studies which enable one to examine these processes formally. There are several aspects of this study which enable advancement of research in these areas. First, in in-home studies in 2008-2009, ~15700 respondents were asked about incarceration histories for their biological mother and father. Second, detailed information about engaging in delinquent behaviors is provided for all respondents from early adolescence (Wave 1) to adulthood (Wave 4). Third, at Wave IV interviews, saliva samples were collected from respondents and genotyped for several candidate genes, including MAOA for research. Fourth, because the allele frequency is significantly higher among African-Americans and the link between the 2R allele and delinquency is the most meaningful among males, we take advantage of the fact that genetic and behavioral data are available for roughly 1,200 African-American male respondents. And finally, identifying environmental factors that may moderate the link between the 2R allele and delinquent behaviors requires detailed information about social risks and resources within the adolescents' home and we use important measures of maternal and paternal closeness to capture some of these complicated processes. Together with the significance of this research question, the Add Health data provide a valid, reliable, and nationally representative characterization of the link between this risk allele and delinquent behaviors.

Families, delinquency, and gene-environment interplay

Examining family processes as antecedents to the 2R-delinquency association is important for several reasons. First, it is possible that *both* family risk factors and genetic risk factors are necessary in order to observe a link between 2R and problem behaviors among males. Recent work by Delisi et al. (2009) has applied the concept of a criminological environment that accounts for the concentrations of offending within families; the presence of a 'criminal' mother or father[1] interacts with genetic propensities to produce delinquency and criminal justice involvement within families. Delisi et al (2009, pp. 1994) argue that genetic propensities associated with DRD2 and having a 'criminal' father creates a 'double whammy' which leads to antisocial behaviors among black women. This association is described as a gene-environment interaction (GxE) in which the effect of the each component (e.g., genetic and environmental) depends on the respective level of the other. As such, family patterns of delinquency indicated by parental incarceration may be a fundamental precursor to the link between 2R and delinquency in the 2nd generation. Shanahan and Hofer (2005) call this GxE model a 'social trigger' model because the social environment triggers the expression of genes related to problem behaviors. This follows similar research used in prior work examining parental incarceration (Roettger and Swisher 2011) using Add Health data, with other studies have demonstrated it remains an important component in reducing delinquency (Demuth and Brown 2004; Booth, Scott, and King 2010).

Second, it is also possible that family factors may control genetic tendencies for delinquent behavior. While an almost limitless number of factors (familial, peer, school, etc) may influence delinquency, the closeness to parents to is well-established factor that reduces the likelihood of delinquency (Booth, Scott, and King; Demuth and Brown 2004; Johnson 1987). For example, the analysis by Booth et al (2010) also suggests attachment to both a non-biological or biological parent significantly reduce delinquency, while work by Johnson (1987) suggests that closeness to the biological father is much more likely to reduce delinquency than closeness to the biological mother. For general social support, Simons et al. (2011) find that a

[1] While we use the term 'criminal' adopted by Delisi et al (2009) in the literature review, it is important to note that we cannot test the implied social labeling/symbolic interactionist perspective implied. The Add Health data lack several key variables such as criminal offending by parents and social psychological measures for measuring how parental incarceration influences children. We adopt 'mother/father incarceration' to more descriptively fit the variables we use in analysis.

supportive social environment moderates genetic propensities to predict aggression. Similarly, Guo et al. (2008a) provide strong evidence that family social resources limit the association between genetic risks and problem behaviors. Using factor analysis among sibling pairs, Beaver (2012) finds that both (a) parental criminality and (2) a lack of maternal attachment and disengagement predict delinquency ('family environment') among children ($p<0.05$), with parental incarceration not mediated by lack of attachment or disengagement. Beaver's analysis, like Delisi et al (2009), suggests that a 'criminal' mother or father results in an 'environmental pathogen' that uniquely predicts delinquency, with parental incarceration distinct from a supportive familial environment. But, more importantly, this research shows that same risky path can be significantly controlled with socially supportive familial environments.

Finally, gene-environment correlation (rGE) is a situation in which the frequency of an allele is correlated with environmental factors that make it difficult to identify the association between the particular allele and some behavioral outcome as causal in nature. In our case, if the 2R allele is linked to delinquent behaviors, then mothers with this risk allele may be more likely than those without the allele to have engaged in risky behaviors themselves. Thus, their children may have been raised in an environment that was more conducive to these behaviors because of the common genetic risk. This form of rGE is called passive gene-environment correlation because children inherit both common genetic and environmental characteristics from their parents. Because gene-environment interaction models assume that genotype and environmental exposure are independent of one another, it is often the case that presence of rGE may bias GxE estimates; what may appear to be GxE may have much more to do with rGE (Jaffee and Price 2007). As such, it is important to examine the association between this risk allele and problem behaviors while adjusting for maternal reports of criminal histories.

Analytic strategy

The MAOA 2R genotype, while rare, provides an opportunity to examine if the direct effects of a genotype associated with delinquency may be explained by social and/or genetic variables related to parental incarceration or parental closeness. Due to lack of inheritance of the MAOA 2R genotype among sons from the father, any moderation of the effect of 2R by father incarceration (FI) or father closeness (FC) would be

due to the non-genetic (environmental) issues related to the father. In contrast, a moderating effect of mother's incarceration (MI) or maternal closeness (MC) on the effect of 2R genotype would suggest that a combination of genetic and environmental effects result in delinquency. In other words, it would be difficult to differentiate between rGE and GxE if the interaction was only among maternal factors. However, the presence of an interaction among paternal factors and 2R genotype and not for 2R genotype and maternal factors provides strong evidence for the social moderation of genetic risk rather than confounding joint inheritance of both factors. In other words, joint comparisons of interactions for both maternal and paternal factors can yield further insights into genetic and non-genetic sources of transmission. While noting the caution in making causal inference due to error, we would interpret results as follows: 1) Non-significance of father interaction and significance of mother interaction would suggest that the expression of 2R arises from genetic components attributable to parental incarceration (e.g., evidence of passive rGE); 2) Significance of both father and mother interactions would suggest that both genetic and non-genetic components are central to the link between 2R and delinquent behavior (e.g., evidence for rGE and GxE); 3) Significance of father interaction but not mother interaction suggests that social factors moderate the link between 2R and delinquency (e.g., evidence for GxE); and 4) non-significance of both father and mother interactions provide evidence that rGE and GxE are largely irrelevant with respect to this genotype and these measures of the environment. If evidence is found for the GxE model only, then significant interactions for parental attachment vs. parental incarceration will lend support for the social control vs. the social trigger model, respectively.

Data and Methods

Data are taken from the National Longitudinal Study of Adolescent Health (Add Health). The Add Health in-home sample consists of 20,700 respondents enrolled in grades 7-12 at Wave I. Follow-up interviews were conducted in 1996, 2001-2002, and 2007-2008, with approximately 14,700 (71%), 15,200 (73%), and 15,700 (75.5%) of respondents, respectively, completing interviews at Waves II, III, and IV. Answers to sensitive questions in Add Health, including youth offending and arrest, were obtained using audio-CASI technology to increase reliability of self-reports (Harris et al. 2009). For this study, we examine

males who had completed interviews at Waves I and IV, and (2) had complete data for variables used in analysis. Our requirement of completion of the Wave I and IV interviews arises from using multiple items from the Waves I & IV questionnaires, including the timing of both mother and father's incarceration. Our resulting sample contains 5594 respondents and 21432 observations which meet these criteria.

We use list-wise deletion in analysis, which is generally known to produce consistent, but inefficient estimates relative to multiple imputation (Allison 2001). The relative rarity of the 2R genotype within the U.S. population and unique nature of the inheritance process for the MAOA gene create issues which are not observed in the existing data, violating the missing at random assumption necessary for analysis.

Measures

[Table 1 About Here]

Delinquency. We use an eight-item measure to measure delinquency. These items consist of the eight items of the subscale used by Guo et al (2008a) and Roettger and Swisher (2011). A similar scale is used by DeLisi et al (2009). These items are a set of violent and non-violent actions which may lead to arrest and incarceration over the twelve months prior to interview. These items include deliberately damaging other's property, stealing something worth more than $50, stealing something worth less than $50, selling drugs, threat or use of weapon to take something from someone, participate in a group fight, burglary of a home or other building, or getting into physical fight. Each item was measured as a collapsed frequency count such that ' 0'=did not occurring in prior 12 months; '1'= occurred 1-2 times in prior twelve months; '2'=occurred 3-4 times in prior twelve months; '3'= occurred 5 or more times in prior twelve months. At each wave, the eight items are summed to create an aggregate score. The Cronbach's alpha for the scale at Wave I is 0.78, 0.77 at Wave II, 0.72 at Wave III, and 0.67 at Wave IV. It is important to note we use self-reports of delinquency. Self-reports have been found to be reliable measures of delinquency when official measures of delinquency, such as arrest and incarceration frequently undercount the extent of illegal behaviors respondents have participated in (Hindelong 1981; Thornberry and Khron 2000).

Mother/Father Incarceration. To measure incarceration histories of the biological mothers and fathers, we rely on self-reports by respondents during Wave IV interviews. Respondents were asked "Has your biological mother/father ever spent time in jail or prison?" If respondents answered 'yes', they were next asked "How old were you when your biological father went to jail or prison (the first time)?" Responses ranged from "not yet born" to "31 years old". In order to address issues of potential temporal ordering, we analyze parental incarcerations which began at ages prior to Wave I interviews, when respondents were in childhood or adolescence. Similar to previous studies examining parental incarceration and deviance with Add Health data, we have found that age when the biological parent is incarcerated is relative insensitive to when the biological parent is first or last incarcerated (Roettger and Swisher 2011; Swisher and Roettger forthcoming).

Mother/Father Closeness. At Wave I, respondents were asked how close they felt to their resident and non-resident parents and residential, but non-biologically related mother and father (i.e., step-parent foster parent, adoptive parent, etc.). Responses ranged from 'not at all' to 'extremely close'. For both the mother and father, we measured closeness using the following scheme: (1) if reported, for the biological mother/father who was either resident or non-resident at time Wave I of Wave I interviews, (2) if no report for the biological mother/father is present, we code the reported closeness to the non-biologically related mother/father when provided, and(3) if no data are reported for the biological and non-biological mother/father, we code a value of 'not at all' if the biological mother/father is unknown or reported known, but absent. A smaller sample using cases where respondents had non-missing self-reports for closeness for the biological mother/father only yielded substantively similar results.

Prior Add health research has combined parental closeness with a series of activities the parent and respondent were both involved in, but parental involvement measures restricted to activities within a 30 day period (Harris and Ryan 2003). However, parental closeness has been found to be a significant predictor of delinquency, while parental involvement remains non-significant (Roettger and Swisher 2011). Due to parental incarceration often separating incarcerated parents from children by significant physical distances, even while the vast majority of parents maintain contact with children, parental closeness serves as a measure of closeness when physical contact/involvement may limited (Braman 2004; Mumola 2000).

MAOA 2R Genotype. We measure the MAOA gene using data from the sibling sample collected at Wave III and the full population sample at Wave IV. The MAOA 2R genotype is a rare polymorphism of the Monoamine Oxidase A gene (MAOA) located at the region on the 1.25 kb region on the X chromosome. The 2-repeat (2R) variant of MAOA is located at 30-bp promoter region for the Variable Number of Tandem Repeats (VNTR) in MAOA, with other variations including the 3R, 3.5R,4R, and 5R variants of the gene (Eisenberger et al 2007; Sabol et et al 1998, Guo et al 2008b). The MAOA 2R genotype has been identified in population and biochemical analyses as a particularly promising candidate genotype for causing delinquency and violence; however research remains difficult due to both the genes rarity and disproportional concentration in minority populations (Sabol et al 1998; Guo et al 2008b; Kim et al 2006). As a result, while the MAOA gene has been extensively studied for its potential links to delinquency and violence (Kim et al 2006), analysis of the 2R genotype has been limited. As far as we know, the MAOA 2R genotype has only been directly studied using the sibling sample of Add Health in studies by Guang Guo and colleagues (Guo et al 2008a; Guo et al 2008b). Recently, with the genotyping of the full Add Health sample, it has become possible to analyze the MAOA 2R gene while addressing the issues of rarity. We thus examine the 2R genotype as a predictor of delinquency, vis-à-vis the 3R, 3.5R, 4R and 5R MAOA genotypes, among a sample of U.S. males.

In addition to these variables, we also control for neighborhood and familial variables. For family measures, we include a measure of family structure for a respondent residing with both biological parents, which is a protective factor relative to single parent, a home with one biological and one step-parent, and other family structures. To control for household income and the over-sampling of middle class blacks present in Add Health, we include a measure for parental education. An alternative measure used by Ford, Bearman and Moody (1999) for family socioeconomic status at Wave I was also incorporated into analysis, and produced virtually identical results; we do not include this measure due to it removing about one hundred cases from analysis. For neighborhood measures, we incorporate a variable for the proportion of African Americans residing in the census tract, a measure used to approximate relative deprivation in spatial analysis (Guo et al 2008). We also incorporate the county's overall violent crime rate as an attempt to control for the

level of violence in the broader community (unfortunately, reliable measures of violence at the local neighborhood level are not available in the data).

Statistical Model

To capture the interaction effects for the MAOA 2R and (1) mother/father incarceration and (2) mother/father closeness, we adopt a three-level multilevel modeling strategy which models the main and interactive effects of 2R and these parental measures on longitudinal delinquency. We estimate a random intercepts in which observations are nested within individuals and individuals are nested within families. The parameter estimates and corresponding variance components for these models are presented in Tables 2 and 3. These models are similar to those used by Guo et al (2008) in their analysis.

Results

[Table 2 about here]

[Table 3 about here]

Tables 2 and 3 report the results of multilevel regression estimates describing the association between the 2R allele, parental risk factors, and delinquency among black males in the United States. Paternal risks are presented in Table 2 and maternal risks in Table 3. In both tables, we discuss the main and moderating effects of paternal/maternal incarceration with the MAOA 2R gene in Models 1-4 of both tables, while we examine the moderating role of mother/father closeness in Models 5-6. We report p-values from the analysis to better gauge statistical significance.

Father/Mother Incarceration

Model 1 in both tables shows a strong and statistically significant association between delinquency and both father ($p<0.001$) and mother incarceration ($p<0.001$), suggesting that; having either a mother or father incarcerated prior to Wave I is associated with an increased risk in delinquency. Similarly, in Model 2, the MAOA 2R genotype has a strong and statistically significant main effect; those with the 2R allele are significantly more likely to engage delinquent behaviors ($b=.114$, $p<.036$). in Tables 2 & 3 (Note: models are identical). In Model 3 of both tables, we find that the addition of both father and mother incarceration does

not mediate the main effect of 2R. For table 2, we find that father incarceration ($p<0.001$) and the 2R genotype $p<0.032$) retain similar magnitudes and are simultaneously statistically significant For table 3, we find that both mother incarceration ($p<0.001$) and the 2R allele ($p<0.039$) retain similar magnitudes and are simultaneously statistically significant Thus, both maternal and paternal incarceration do not substantively alter the main effect 2R, with 2R and mother/father incarceration both being significant predictors of delinquency. These results provide strong evidence against the passive form of gene-environment correlation for the 2R allele, parental incarceration, and delinquent behaviors.

In model 4 of both tables, we include an interaction term between mother/father incarceration and the 2R genotype. For table 2, we find that father incarceration ($p<0.001$) and the 2R genotype ($p<0.058$) have significant or marginally significant main effects, while the interaction term remains non-significant ($p<.774$). For table 3, we find that mother incarceration ($p<0.002$) and the 2R genotype ($p<0.079$) have significant or marginally significant main effects, while the interaction term remains non-significant ($p<0.186$). Thus, we do not find any evidence for moderating effects suggesting that maternal or paternal incarceration interact with the 2R genotype.

As a whole, mother incarceration, father incarceration, and the 2R genotype all have direct and statistically significant associations for predicting delinquency. However, we have found that both MIx2R and FIx2R interactions are not significant. This suggests that mother/father incarceration and the 2R genotype exerts largely independent effects for the genetic and environmental components related to parental incarceration (both the incarceration directly, along with associated variables such as parental criminality, family instability, poverty, etc.). These findings do not support the social trigger gene-environment interaction model and also suggest that a gene-environment correlation is observed for mother/father incarceration, the MAOA 2R genotype, and delinquency

Father/Mother Closeness

In Models 5 & 6 of Tables 2-3, we also examine how maternal and paternal closeness may potentially moderate the 2R genotype, when controlling for parental incarceration. In doing so, we are able to examine

the extent to which mother/father closeness may alternatively explain the association of 2R, when controlling for the genetic and environmental associations which may be attributed to a biological mother or father undergoing incarceration.

Model 5 in Tables 2 and 3 provides a baseline relative to Model 3. In Table 2, we find a statistically significant, inverse relationship between father closeness and delinquency ($b=-0.33$, $p<0.001$) such that delinquency declines with increasing closeness to the father. At the same time, the 2R genotype remains significant ($p<0.022$), while father incarceration ($p<0.001$) remains a highly statistically significant factors for increased delinquency. In Table 3, we observe a highly significant, inverse relationship between mother closeness and delinquency ($b=-0.32$, $p<0.001$). While the 2R genotype remains a statistically significant relationship ($p<0.035$) as in Models 1-4, maternal incarceration is found to remain highly significant ($p<0.002$).

Model 6 in Tables 2-3 show the interaction between mother-father closeness and the main 2R MAOA genotype. In table 2, we find that the main effect of the 2R genotype remains statistically significant ($p<0.035$), while the main effect of father closeness is negative and highly significant ($p<0.001$). The interaction between father closeness and 2R is significant ($b=-0.086$, $p<0.013$) and the direction of the interaction suggests that father closeness reduces the risks associated with the 2R allele; these findings support the social control model described above.

We also find tentative evidence for a different gene-environment interaction for maternal closeness. While this value does not reach traditional statistical significance ($p<.070$), the direction of the association ($b=.098$) indicates that maternal closeness may exacerbate the risks associated with the 2R allele, and in the opposite direction of the effect for maternal closeness among those who do not have the 2R genotype ($b=-0.033$, $p<0.001$). The main effect of the 2R genotype becomes marginally significant and largely unchanged ($b=0.103$, $p<0.06$). Maternal incarceration remains a highly significant predictor of increased delinquency ($p<0.002$). We discuss this association in the conclusion. The general patterns suggest that the effect of 2R is moderated by closeness of the respondent to the biological father, but not less so by the biological mother.

Research suggests that absence and lack of closeness to a biological or non-biologically related father reduces delinquency and risk of incarceration (Harper and McLanahan 2002; Demuth and Brown 2004; Booth, Scott, and King 2010). The effects of both mother and father incarceration do not substantially change when measures of mother and father closeness are introduced in models 5-6. Our findings, generally fit those reported by Johnson (1987), where father closeness was a more significant predictor of delinquency and maternal closeness did not significantly vary within the sample.

Discussion

Research in the area of gene-environment interplay has produced exciting but sometimes contradictory results (Caspi et al. 2002; Freese and Shostak 2010). While the research in this area has generally focused on the moderating role of environmental factors as social triggers or social controls (Shanahan and Hofer 2005), there remains very little consensus about the generalizability of one particular gene-environment interaction model. Here, we show that social resources and protective factors may be more salient to the moderation of genetic associations for delinquent behavior than social risks or stressors, per se. This finding is important because it is in line with other studies in this area (Guo et al. 2008a; Boardman et al. 2012b) which provides evidence for GxE interactions within a social control framework, and it highlights the critical role of father closeness with respect to this specific form of problem behavior. Thus, with respect to delinquent behaviors and GxE research, our study is in-line with prior work suggesting that the environment may sometimes be best characterized as a protective factor, instead of only increasing risk.

Overall, our analyses find father closeness moderates the effects of 2R, and not maternal closeness (though marginally significant), father incarceration, and mother incarceration. We infer from our analysis that the association between 2R and delinquency is not mediated by either genetic or environmental factors related to a mother or father being incarcerated. Rather, the effect of 2R is only significantly moderated by father closeness ($p<0.013$), though the moderation by mother closeness is close to statistically significant

($p<0.07$). Importantly, the association between parental closeness, risky genotype, and delinquency among boys is opposite in sign when considering father vs. mother closeness.

There are three ways to interpret the fact that maternal closeness seems to trigger genetic tendencies for delinquent behavior. First, it may simply be a form of rGE called "evocative" gene-environment correlation. That is, mothers with children who are engaged in the most egregious forms of delinquent behavior may decide to spend a great deal of time with their children to reduce their involvement in risky behavior. Thus, the genetically risky children evoke an environment that may appear as a gene-environment interaction. Second, the social push and social distinction GxE models (Shanahan and Boardman 2009) focus on typical or nurturing environments as the contexts in which genetic risks become the most salient. That is, the riskiest enviornments (e.g., those in which there is virtually no maternal closeness), there is so much environmental "noise" that small genetic associations are washed out; it is only in relatively stable and predictable contexts are we able to see these small genetic associations.

How do we explain the moderation effects among our panel? As we discuss in more detail below, data limitations make it important to avoid making causal inference from the analysis. However, it is possible to deduce some basic conclusions from the results. The lack of mediation and moderation of the MAOA 2R genotype with both maternal and paternal incarceration are notable, given the inheritance of the 2R genotype from biological mothers to sons. If the environmental effects of parental incarceration played significant role in the expression of the 2R genotype, we would have expected father incarceration and, to a lesser extent, mother incarceration to play a role in the variation of 2R. If genetic effects of parental incarceration played a significant role in the expression of 2R, we would have expected an interaction between 2R and mother incarceration, but not father incarceration. The fact that the 2R genotype and father/mother incarcerations exert strong and significant effects, with very little change when examining for mediating and moderating effects of 2R, suggests that father/mother incarceration and the 2R genotype are largely independent effects, with environmental and genetic factors associated with parental incarceration and

son's delinquency playing no substantial role in either mediating or moderating the 2R genotype's association with delinquency.

Instead, father closeness is the only variable found to moderate the 2R genotype, even when controlling for father incarceration. This research fits with the general findings that closeness to a father, either biological or non-biological parent, reduces delinquency, while absence of a father leads to increased risk of delinquency and incarceration (Booth et al; Demuth and Brown 2004; Harper and McLanahan 2002). Thus, the closeness of a father to a son (or lack, thereof) moderates the expression of delinquency associated with the 2R genotype, while parental incarceration exerts an influence on delinquency independent of the 2R genotype. The results also fit the GxE model of social control, where environmental variables moderate the expression of the 2R genotype on delinquent behavior.

While beyond the scope of the Add Health data, we believe that the racial disparities for incarceration and two-parent biological households must be taken into account due to their potential role in generating gene-environment correlations. Regarding incarceration, one-third of all African American males and 70% of African American male high school dropouts born between 1975-1979 spend one-year or more in state or federal, along with 5% of all white males and 15% of white male high school dropouts. Thus, incarceration has become a uniquely common lifecourse event in the U.S. (Bonczar 2003; Pettit and Western 2004; Western and Wildeman 2009), along with the families and communities which they are imbedded in (Clear 2007; Western 2006). While the relative rates of incarceration between blacks and whites has remained constant for over a century, the exponential growth of incarcerated populations in the U.S. since the 1970s has also led to differential criminal justice involvement across generations (Western 2006). Thus, for our panel of respondents born in the late 1970s and early 1980s, they face a probably of criminal justice involvement much higher than their parents (Western and Wildeman, 2009). This may lead to a pattern where the 2R genotype associated with delinquency, but the genotype remains uncorrelated with mother/father incarceration.

Following trends in the rise of single-parent families over the last half-century (Livingston and Parker 2011), roughly one-third of our sample report not residing with both biological parents at Wave I, with the vast majority of those not living with both biological parents residing with their biological mother. The lack of a resident biological or non-biological father is associated with an increase in delinquency (Booth et al 2010), with father involvement and closeness also generally declining over time as years progress beyond the date when the father becomes non-resident (Cheadle, Amato, and King 2010; Livingston and Parker 2011). As such, declining father closeness and involvement may, aggregately, play a much larger role in moderating the expression of delinquency among respondents with the 2R genotype.

We note that here we have used demographic trends to construct a reasonable interpretation of the patterns of the data for the moderating effects of father closeness, and the lack of moderating effects for mother/father incarceration. However, as with most gene-environment correlations (rGE), the larger social environment in humans remains difficult to measure, due in no small part to the unobserved and larger shifting social phenomenon. In showing the heritability of smoking behaviors increased over decades as smoking became increasingly marginalized, Boardman et al (2012a) illustrate how changes in the larger social environment may be captured through use of additional data sources and longitudinal designs. Future research, which makes use of 2R genotypes from current or past studies, in combination with similar measures, may help to better determine how and why mother-father incarceration do not currently interact with 2R, while father closeness moderates 2R. While limitations of causal inference (genetic or otherwise) from existing datasets have been noted (Farrington et al 2007; Thornberry 2009), additions of genetic measures to multi-generational samples following delinquency and incarceration data of parents and children would allow such comparisons.

A broad array of familial, social, and neighborhood factors are thought to influence intergenerational delinquency, including low socioeconomic status, abusive parenting, lack of pro-social parenting skills, family instability, friendships, and poor school and neighborhood environments (Boutwell and Beaver 2010; Farrignton, Coid, & Murray 2009, Thornberry et al 2003; Roettger and Swisher 2011) Collectively, this

'criminogenic environment', as it is sometimes known, is associated with early-onset offending (Moffitt 1993; Tibbetts & Piquero 1999) and implicated in the link between parental incarceration and child antisocial behavior/delinquency (Farrington 2011; Wakefield and Wildeman 2011). While our observations with the 2R genotype are not consistent with those of DeLisi et al (2009), this does not invalidate their findings. Rather, with hundreds of genes and environmental variables at play for a complex phenotype, we additionally report that the criminogenic environment, as measured by parental incarceration, is also unrelated to the 2R genotypes association with delinquency.

As such, the interplay of thousands of genetic and environmental variables is an important qualifier. As more sophisticated analyses using Genome-Wide Association Studies (GWAS) suggests, fitting these pieces together while accounting for both type I and type II errors creates a Herculean task in measurement which severely limit advances with traditional statistical modeling (Turkheimer 2012). Future research on how criminogenic environments and genes interact to play a role in intergenerational delinquency has the promise of more fully unraveling this puzzle, while assuming the daunting burden of dealing with error generated from analysis of literally millions of the genetic markers and the environmental variables (for example, with $p<0.05$ and 1 million genetic variables, we would expect an average of 50,000 false-positives; a Bonferroni correction would imply an adjusted p-value of $5x10^{-8}$).

It also remains important note that, while mother and father incarceration may not interact with the 2R genotype, the genetic component of 2R may not be observed through mothers. Brunner et al (1993) observed an extremely rare MAOA mutation which was transmitted to males over multiple generations in a family, but delinquency was not observed among mothers. We would note that, while Guo et al (2008b) observed an association between possessing one or more MAOA 2R alleles and delinquency, but this done mean that a genetic pattern may manifest when examining 3 or more generations of a family. A few datasets such as the Cambridge Study in Delinquent Development may be able to sort out such a pattern, but both genetic and behavioral measures are needed to reveal such a genetic pattern. Given data limitations, unraveling such a pattern for the MAOA gene is currently beyond the scope of current research.

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Table 1. Descriptive statistics for all variables used in the analysis.

Variable	Mean (SD)
Serious Delinquency Wave I	0.71 (0.79)
Serious Delinquency Wave II	0.52 (0 .71)
Serious Delinquency Wave III	0.44 (0.67)
Serious Delinquency Wave IV	0.24 (0.52)
Father Incarcerated Prior to Wave I	0.12 (0.33)
Mather Incarcerated Prior to Wave I	0.02 (0.14)
Father Closeness at Wave I	3.83 (1.41)
Mother Closeness	4.46 (0.97)
MAOA 2R Genotype (2R)	0.01 (0.108)
Age at Wave I	15.68 (1.73)
Age at Wave II	16.20 (1.64)
Age at Wave III	21.91 (1.84)
Age at Wave IV	28.49 (1.84)
Race	
White [Reference]	0.54 (0.50)
Black	0.20 (0.40)
Hispanic	0.16 (0.37)
Asian	0.07 (0.25)
Native	0.02 (0.13)

Other Race	0.01
	(0.09)
Resided With Both Biological Parents at Wave I	0.56
	(0.50)
Parent Completed BA	0.25
	(0.43)
Percentage of Census Tract African American	0.15
	(0.25)
Violent Crime Rate	1.98
	(0.16)
Number of Respondents	5596
Number of Observations	21432

Table 2. Main and interactive effects of MAOA 2R genotype on delinquency among males as a function of paternal behaviors and closeness

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Father Incarcerated (FI)	0.150***		0.150***	0.150***	0.140***	0.141***
	(0.019)		(0.019)	(0.019)	(0.019)	(0.019)
MAOA 2R Genotype (2R)		0.114*	0.116*	0.110+	0.124*	0.114*
		(0.054)	(0.054)	(0.058)	(0.054)	(0.054)
2R X FI				0.045		
				(0.156)		
Father closeness (FC)					-0.033***	-0.032***
					(0.005)	(0.005)
2R x FC						-0.087*
						(0.035)
Age	0.421***	0.425***	0.426***	0.426***	0.408***	0.409***
	(0.046)	(0.046)	(0.046)	(0.046)	(0.046)	(0.046)
Age^2	-0.021***	-0.021***	-0.021***	-0.021***	-0.020***	-0.020***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Age^3	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Race						
White [Reference]						
Black	0.006	0.006	0.001	0.001	-0.005	-0.006
	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)
Hispanic	0.034+	0.034+	0.033+	0.033+	0.031+	0.031+
	(0.019)	(0.019)	(0.019)	(0.019)	(0.018)	(0.018)
Asian	0.012	0.008	0.013	0.013	0.005	0.005
	(0.025)	(0.025)	(0.025)	(0.025)	(0.025)	(0.025)
Native	0.076+	0.083+	0.076+	0.076+	0.069	0.069
	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)
Other Race	0.025	0.024	0.023	0.024	0.016	0.013
	(0.064)	(0.064)	(0.064)	(0.064)	(0.063)	(0.063)
Both Bio Parents (W1)	-0.063***	-0.089***	-0.065***	-0.065***	-0.018	-0.019
	(0.013)	(0.013)	(0.013)	(0.013)	(0.015)	(0.015)
Parent Completed BA	0.007	-0.001	0.007	0.006	0.008	0.007
	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)
Tract % African American	-0.059+	-0.067+	-0.059+	-0.060+	-0.063+	-0.063+
	(0.034)	(0.034)	(0.034)	(0.034)	(0.034)	(0.034)
County Violent Crime Rate (ln)	0.034	0.023	0.034	0.034	0.034	0.034
	(0.043)	(0.043)	(0.043)	(0.043)	(0.043)	(0.043)
Intercept	-1.986***	-1.986***	-2.071***	-2.074***	-1.976***	-1.977***
	(0.328)	(0.328)	(0.328)	(0.328)	(0.328)	(0.327)
Variance components						
Individual	0.015	0.040	0.026	0.030	0.041	0.054
Family	0.334	0.332	0.332	0.332	0.329	0.325
Residual	0.588	0.588	0.588	0.588	0.587	0.588
Log-Likelihood	-20726.47	-20756.6	-20724.2	-20724.1	-20724.2	-20698.6
Number of Respondents	5594	5594	5594	5594	5594	5594
Number of Observations	21432	21432	21432	21432	21432	21432

Table 3. Main and interactive effects of MAOA 2R genotype on delinquency among males as a function of maternal behaviors and closeness.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Mother Incarcerated (MI)	0.153***		0.152***	0.143**	0.134**	0.133**
	(0.043)		(0.043)	(0.043)	(0.043)	(0.043)
MAOA 2R Genotype (2R)		0.114*	0.112*	0.098+	0.114*	0.103+
		(0.054)	(0.054)	(0.056)	(0.054)	(0.055)
2R X MI				0.365		
				(0.276)		
Mother closeness (MC)					-0.032***	-0.033***
					(0.007)	(0.007)
2R x MC						0.098+
						(0.054)
Age	0.422***	0.425***	0.422***	0.422***	0.410***	0.410***
	(0.046)	(0.046)	(0.046)	(0.046)	(0.046)	(0.046)
Age^2	-0.021***	-0.021***	-0.021***	-0.021***	-0.020***	-0.020***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Age^3	0.000***	0.000***	0.000***	0.000***	0.000***	0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Race						
White [Reference]						
Black	0.008	0.006	0.004	0.004	0.010	0.011
	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)
Hispanic	0.035+	0.034+	0.034+	0.034+	0.038*	0.038*
	(0.019)	(0.019)	(0.019)	(0.019)	(0.019)	(0.019)
Asian	0.008	0.008	0.008	0.008	0.007	0.007
	(0.025)	(0.025)	(0.025)	(0.025)	(0.025)	(0.025)
Native	0.080+	0.083+	0.080+	0.081+	0.076+	0.076+
	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)	(0.044)
Other Race	0.024	0.027	0.027	0.027	0.032	0.031
	(0.064)	(0.064)	(0.064)	(0.064)	(0.064)	(0.064)
Both Bio Parents (W1)	-0.084***	-0.089***	-0.084***	-0.084***	-0.072***	-0.072***
	(0.013)	(0.013)	(0.013)	(0.013)	(0.013)	(0.013)
Parent Completed BA	-0.001	-0.001	0.000	0.000	-0.002	-0.003
	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)
Tract % African American	-0.067+	-0.067+	-0.068+	-0.068*	-0.067+	-0.068*
	(0.034)	(0.034)	(0.034)	(0.034)	(0.034)	(0.034)
County Violent Crime Rate (ln)	0.023	0.023	0.026	0.025	0.026	0.026
	(0.043)	(0.043)	(0.043)	(0.043)	(0.043)	(0.043)
Intercept	-1.986***	-1.986***	-1.925***	-2.003***	-1.925***	-1.924***
	(0.328)	(0.328)	(0.328)	(0.328)	(0.328)	(0.328)
Variance components						
Individual	0.015	0.040	0.026	0.030	0.041	0.054
Family	0.334	0.332	0.332	0.332	0.329	0.325
Residual	0.588	0.588	0.588	0.588	0.587	0.588
Log-Likelihood	-20752.6	-20756.6	-20750.4	-20749.6	-20738.3	-20736.7
Number of Respondents	5594	5594	5594	5594	5594	5594
Number of Observations	21432	21432	21432	21432	21432	21432