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The Intersection of Genes and Neuropsychological Deficits in the Prediction of Adolescent Delinquency and Low Self-Control

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Gottfredson and Hirschi's *A General Theory of Crime,* Moffitt's developmental taxonomy theory, and Caspi et al.'s Gene × Environment study are three of the most influential pieces of contemporary criminological scholarship. Even so, there has been little attempt to integrate and empirically assess these three perspectives simultaneously. This article addresses this gap in the literature by analyzing phenotypic and genotypic data from the National Longitudinal Study of Adolescent Health (Add Health). The results revealed that all three perspectives have considerable empirical support, where neuropsychological deficits interact with the MAOA genotype to predict adolescent delinquency and levels of self-control for White males. The theoretical implications of the findings are noted.

Keywords: *Add Health; delinquency; MAOA; low self-control; neuropsychological deficits*

Three of the most important pieces of contemporary criminological scholarship are Gottfredson and Hirschi's (1990) book *A General Theory of Crime,* Moffitt's (1993) developmental taxonomy theory, and Caspi et al.'s (2002) study published in *Science,* which revealed a Gene × Environment interaction on antisocial behaviors. The significance of these publications should not be casually glossed over; collectively, they have been cited more than 5,000 times and have guided much empirical and theoretical research both within criminology and across many other disciplines. Moreover, the authors of these works are among the most frequently cited and well-respected criminologists worldwide (Cohn & Farrington, 2007).[1] Against this

backdrop, these three publications represent the most influential works by some of the leading "criminological giants."

Each of these three works applies a slightly different theoretical framework to the study of crime and invokes different factors thought to explain the etiology of antisocial behaviors. For example, Gottfredson and Hirschi highlighted the connection between low self-control and antisocial behaviors, Moffitt underscored the role of neuropsychological deficits in the development of life-course persistent offending, and Caspi et al. pointed to the interactive effects that genes and the environment have on serious, violent offending. Although these perspectives offer somewhat divergent explanations to criminal and delinquent involvement, it is possible to extract key variables from each of these perspectives, empirically test the merits of each, and begin to map out the ways in which these perspectives may be interrelated.

Gottfredson and Hirschi's General Theory of Crime

Gottfredson and Hirschi's (1990) general theory of crime posits that all crime, delinquency, and analogous behaviors are the result of low self-control and a criminal opportunity. Most empirical research testing the merits of this theory has ignored the effect of a criminal opportunity (but see Burton, Cullen, Evans, Alarid, & Dunaway, 1998; LaGrange & Silverman, 1999) and instead has focused on how individual variation in self-control is connected to delinquent behaviors. According to Gottfredson and Hirschi, persons low in self-control are impulsive, are self-centered, are risk seekers, prefer simple tasks, prefer physical activities over mental ones, and have a temper. Together these six elements are thought to be indicators of a single underlying trait termed low self-control.

The brunt of research investigating Gottfredson and Hirschi's theory, however, has focused on the relationship between low self-control and crime. Collectively the results of these studies have provided substantial empirical support in favor of the general theory. For example, low self-control has been found to be predictive of antisocial behaviors in samples of males and females (Vazsonyi, Pickering, Junger, & Hessing, 2001), in samples of adolescents (LaGrange & Silverman, 1999) and adults (Evans, Cullen, Burton, Dunaway, & Benson, 1997; Grasmick, Tittle, Bursik, & Arneklev, 1993), in samples of adolescent and adult offenders (Cauffman, Steinberg,

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& Piquero, 2005; DeLisi, 2001; Jones, Cauffman, & Piquero, 2007), in samples drawn from America (Burton et al., 1998; Evans et al., 1997; Grasmick et al., 1993) and other nations (Vazsonyi et al., 2001), and in samples of Whites (Burton et al., 1998) and minorities (Burt, Simons, & Simons, 2006). Empirical support for the general theory was confirmed recently in a meta-analysis by Pratt and Cullen (2000).

There is clear and convincing evidence of a relatively modest and robust relationship between low self-control and various types of antisocial outcomes. Perhaps as a result, researchers have recently turned their attention toward examining the antecedent causes of self-control. According to Gottfredson and Hirschi, the origins of self-control were to be found in social factors, not biological ones, and parents were the primary agent responsible for fostering self-control (or the lack thereof) in their children. Specifically, Gottfredson and Hirschi outlined three intertwined parental socialization tactics that caused the emergence of self-control: supervision, recognition of deviant acts, and the consistent punishment of childhood transgressions. The logic underlying this explanation is that parents who engaged in these three socialization techniques are, on average, likely to inculcate high levels of self-control in their children, whereas parents who do not employ these tactics are likely to raise children with low levels of self-control. For Gottfredson and Hirschi, parents have only a small window of opportunity to promote the development of self-control; by around the ages of 8 to 10 years, levels of self-control will become fixed and will remain relatively stable throughout the remainder of the life course.

An emerging line of research has tested Gottfredson and Hirschi's parental socialization thesis and found some support in favor of it. Consistent with the theory, levels of self-control have been found to be influenced by a number of different parenting techniques, including parental supervision, parental attachment, parental efficacy, and other intrafamilial factors (Burt et al., 2006; Gibbs, Giever, & Higgins, 2003; Gibbs, Giever, & Martin, 1998; Perrone, Sullivan, Pratt, & Margaryan, 2004; Polakowski, 1994; Unnever, Cullen, & Pratt, 2003). At the same time, there is some evidence to suggest that there are other social institutions—beyond those found in the family—that affect individual levels of self-control. Researchers, for instance, have identified school- and neighborhood-level characteristics as salient contributors to the development of self-control (Beaver, Wright, & Maume, 2008; Pratt, Turner, & Piquero, 2004; Turner, Piquero, & Pratt, 2005). Collectively the results of these studies indicate that a number of different social factors are important in the genesis of self-control; however, the effects of these variables account for only a small percentage of the explained variation, much less than what Gottfredson and Hirschi hypothesized. Thus there are likely other factors that are also important contributors to the emergence of self-control.

Moffitt's Developmental Taxonomy

In 1993, Moffitt set forth her theory of life-course-persistent (LCP) and adolescence-limited (AL) offenders, which has come to be known as the developmental taxonomy.

In explicating her theory, Moffitt began by arguing that there are two distinct groups of offenders (i.e., LCP offenders and AL offenders), each with their own unique etiology. LCP offenders are characterized as displaying antisocial proclivities early in life, well before the age of criminal responsibility. For example, as children, LCP offenders are typically physically aggressive, extremely difficult to manage, and unruly. As adolescents, LCP offenders engage in an array of delinquent activities, such as drinking alcohol, using and abusing drugs, and skipping school. What separates LCP offenders from other adolescent delinquents is their involvement in acts of serious violence, such as assault, armed robbery, and rape. As adults, LCP offenders continue engaging in serious antisocial acts and usually accumulate a lengthy criminal record. LCP offenders are aptly named because they begin displaying antisocial behaviors in childhood and persist with such behaviors throughout the remainder of their life. Approximately 5% of all adolescents (almost all of whom are male) are LCP offenders, but they account for the vast majority of all serious violent offenses (Moffitt, 1993).

Moffitt also described a second type of youthful delinquents—called AL offenders—who are very different from LCP offenders. Moffitt recognized that youthful delinquency is age normative, with the majority of adolescents at least dabbling in minor forms of illegal behaviors, such as petty theft, truancy, and underage drinking. However, AL offenders neither show signs of antisocial behaviors early in life nor continue offending into adulthood; their delinquent behaviors are restricted to adolescence.

Moffitt was very clear that different factors work to produce LCP offenders and AL offenders. According to Moffitt, the origin of LCP offending is the interaction between two different factors: neuropsychological deficits and an adverse home environment. Moffitt's identification of neuropsychological problems as a causal agent in the etiology of LCP offenders was an important contribution to the criminological literature. As Moffitt explained, children with neuropsychological problems are frequently hard to manage, manifest difficult temperaments, and engage in serious troublesome behavior from a very early age (Moffitt, 1990). The constellation of problems caused by neuropsychological deficits thus places these children at high risk for becoming an LCP offender. Moffitt also identified the child's home environment as a second factor implicated in the development of LCP offending. A transactional process unfolds where neuropsychologically impaired children elicit responses from their rearing environment. Most of the time parents are equipped with the necessary skills to respond to and deal with difficult children in an appropriate manner. When this occurs, the environment is able to blunt the problems associated with neuropsychological problems. However, in some situations a neuropsychologically impaired child is raised by abusive or otherwise criminogenic parents. The reactions by these types of parents exacerbate the antisocial propensities of their child, and the end result is the formation of a LCP offender. LCP offenders, in short, are created by complex interactions between neuropsychological deficiencies and an adverse home environment. Of importance, research has revealed support in favor of Moffitt's explanation to LCP offenders (for excellent overviews of the research, see Moffitt, 2003, 2006).

AL offenders, on the other hand, are not produced by neuropsychological deficits or any inborn propensity but instead are largely the product of social mimicry. To understand the role of social mimicry in the genesis of AL offenders it is important to draw attention to the fact that in modern societies, adolescents, although biologically mature, are not afforded all of the same privileges that are extended to adults. For example, adolescents are not legally allowed to purchase and consume alcohol, they are not allowed to vote, and they are required to attend school. The end result is that there is a disjuncture between their biological maturity and their social immaturity—something Moffitt referred to as the maturity gap. Adolescents recognize, however, that LCP offenders are not "trapped" in the maturity gap because they engage in adultlike behaviors, such as drinking, smoking, and skipping school (Piquero & Brezina, 2001). To erase the dissonance of the maturity gap, AL offenders thus begin to mimic the more mundane forms of delinquency displayed by LCP offenders. Once AL offenders become adults, they are no longer trapped in the maturity gap and their delinquent behaviors evaporate.

Caspi et al.'s Gene × Environment Study

Numerous studies have been conducted using samples of twin dyads and samples of adoptive children to estimate the genetic and environmental effects on antisocial behaviors. This line of behavioral genetic research has revealed that antisocial behaviors are the result of environmental influences, genetic influences, and, perhaps most important, the interaction between genes and the environment. Although twin- and adoption-research designs are useful, they can only estimate the proportion of variance in a given trait or behavior that is due to the environment and the proportion of variance that is due to genetic factors. These types of behavioral genetic methodologies cannot reveal which specific environments are important and which specific genes are important. Other research designs, ones that include measures of the environment and measures of genes, are needed to address questions related to the precise environments and genes implicated in criminal and delinquent behaviors.

Caspi et al. (2002) were the first researchers to examine the interactive effects between a measured environment and a measured gene on a behavioral phenotype. Specifically, Caspi and his colleagues hypothesized that a history of childhood maltreatment would interact with the monoamine oxidase A (MAOA) genotype to predict involvement in criminal and violent behaviors. The MAOA gene codes for the production of the enzyme MAOA, which breaks down neurotransmitters in the synapse (Ellis, 1991). The MAOA genotype comes in two broad classes: a high-activity version and a low-activity version. Based on the existing genetic literature, Caspi et al. hypothesized that the low-activity version of MAOA would confer an increased susceptibility to antisocial behaviors, but only among those respondents who had been maltreated as children. The high-activity version of MAOA, on the other hand, was thought to buffer the criminogenic effects of childhood maltreatment.

Caspi et al., in short, expected the MAOA gene to interact with childhood maltreatment in the prediction of antisocial behavior. To test this hypothesis, they analyzed a sample of White males from the Dunedin Multidisciplinary Health and Development Study.

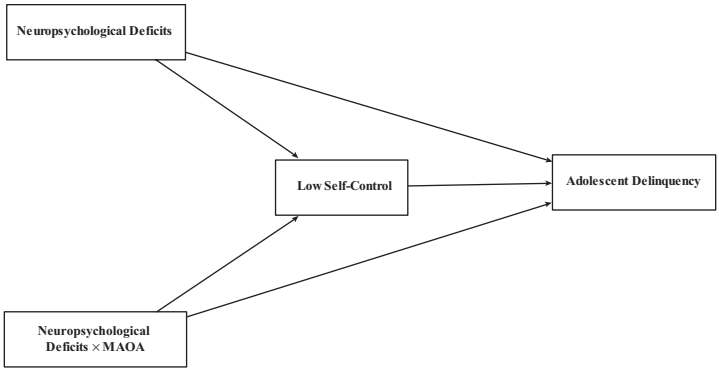
Their analysis revealed three broad findings. First, respondents who were maltreated as a child were significantly more likely to be diagnosed with conduct disorder, to be convicted of a violent offense, to score high on an antisocial personality symptoms scale, to score high on a disposition to violence scale, and to score high on a composite measure of antisocial behavior. Second, the main effect of the MAOA gene was not a statistically significant predictor of any of the five antisocial outcome measures. Third, and perhaps most important, childhood maltreatment interacted with MAOA to predict a statistically significant amount of variation in four of the five dependent variables. In substantive terms this interaction meant that the *low-activity MAOA genotype had an effect on antisocial behavior only among those respondents who had been maltreated as a child.* The effect sizes for this interaction term were quite impressive; although only 12% of the sample had been maltreated as a child and had the low-functioning version of the MAOA allele, they accounted for 44% of all violent convictions. Subsequent replication studies have both substantiated (Foley et al., 2004; Frazzetto et al., 2007; Kim-Cohen et al., 2006; Widom & Brzustowicz, 2006) and contradicted (Haberstick et al., 2005; Huizinga et al., 2006; Young et al., 2006) the MAOA × Maltreatment interaction, but a recent meta-analysis found that this Gene × Environment interaction was statistically significant across studies (Kim-Cohen et al., 2006).

Theoretical Overlap

At first glance it might seem as if these three pieces of research are distinct; however, it is possible to draw theoretical and empirical linkages among them. We begin by pointing out that there is a growing recognition that neuropsychological deficits have a direct effect on antisocial behaviors (Moffitt, 1990) and that levels of self-control are, in part, due to neuropsychological functioning (Beaver, Wright, & DeLisi, 2007; Wikström & Treiber, 2007). Figure 1 displays the potential interrelationships among low self-control, MAOA, neuropsychological deficits, and delinquency.

To further elucidate the interconnections among these three perspectives it is important to point out that an emerging body of research—known as genetic imaging—has begun to map the ways in which certain genetic variants correspond to differences in brain structure and brain functioning. This is particularly important to Caspi et al.'s (2002) study because genetic imaging studies have revealed that individuals possessing the low-activity version of MAOA—the same variant linked with antisocial behaviors—have reduced limbic volume, have a hypoactive hippocampus, and have reduced activity in certain regions of the prefrontal cortex (Meyer-Lindenberg et al., 2006; Meyer-Lindenberg & Weinberger, 2006). It is likely that the effect of

Figure 1
Schematic Conceptualization of the Interrelationships Among Neuropsychological Deficits, Monoamine Oxidase A, Low Self-Control, and Adolescent Delinquency



Note: Monoamine oxidase A (MAOA) is not posited to have a direct effect on low self-control or adolescent delinquency.

MAOA on antisocial behaviors reported by Caspi et al. (2002) was at least in part due to differences in brain circuitry between the low- and high-activity genotypes. Meyer-Lindenberg and colleagues (2006) echoed this view when they concluded that

> the genetic data presented here . . . argue against an association of MAOA genotype with instrumental aggression and for a genetic risk for impulsive violence; and indicate that, whereas both instrumental and impulsive aggression may be present to varying degrees in most violent offenders, the risk imparted by the specific genetic variation studied here contributes to the impulsive dimension of this complex behavior. (p. 6272)

Note that Meyer-Lindenberg et al. highlighted the importance of MAOA in impulsive behaviors—that is, behaviors that flow from the lack of self-control (Wright & Beaver, 2005). If this is the case, then levels of self-control should be partially explained by the MAOA gene.

Last, Moffitt's (1993) developmental taxonomy and Caspi et al.'s (2002) Gene × Environment interaction study point to the importance of interactions in the etiology of antisocial behaviors. Recall that Moffitt's theory argues that an adverse home environment interacts with neuropsychological deficits, whereas Caspi et al.'s (2002) study found that MAOA interacted with childhood maltreatment. It is also instructive to point out that genes usually do not have a direct effect on complex behavioral phenotypes but usually interact with the environment (Moffitt, 2005;

Rutter, 2006), with other genes (Beaver, Wright, DeLisi, Walsh, et al., 2007), or with individual differences. As a result, we do not show any direct effects of MAOA in Figure 1.

In our study, we analyze data from the National Longitudinal Study of Adolescent Health (Add Health) to examine whether the theoretical linkages presented in Figure 1 are empirically supported. Specifically, and in line with the current theoretical (Moffitt, 1993; Wikström & Treiber, 2007) and empirical (Beaver, Wright, DeLisi, Walsh, et al., 2007) research, we hypothesize that neuropsychological deficits will contribute to individual differences in levels of self-control and delinquent involvement. Moreover, given that research has in general failed to detect significant main effects of MAOA on antisocial outcomes (Caspi et al., 2002), we posit that MAOA will not have any direct effects either on low self-control or on adolescent delinquency. We do, however, hypothesize that MAOA will interact with neuropsychological deficits to predict a significant amount of variation in self-control and in delinquency.[2]

Method

Data

Data for this study come from the Add Health. The Add Health is a longitudinal and nationally representative sample of American youths in 7th through 12th grade (Udry, 2003). The sample was selected by employing multistage stratified random sampling techniques. Using this sampling design, 132 middle schools and high schools were identified and chosen for inclusion in the sample. On a specified school day in 1994-1995, all students in attendance were then administered a self-report survey that asked questions about their behaviors, their friends, and their relationships with family members. More than 90,000 students participated in the Wave 1 in-school component of the Add Health study. A subsample of respondents was then chosen to be reinterviewed in their home, along with their primary caregiver (usually their mother). During these interviews, information was gathered about the youth's delinquent involvement, their personality traits, and their social relationships. In total, 20,745 adolescents and 17,700 of their primary caregivers participated in the Wave 1 in-home interview (Harris et al., 2003).

Approximately 1 to 2 years later, the second round of in-home interviews was conducted with 14,738 adolescents. Because relatively little time had lapsed since the Wave 1 interviews, and because most of the respondents were still adolescents, most of the questions asked at Wave 1 were retained on the Wave 2 survey instruments. Respondents, for example, were asked about their temperament, history of risky and criminal behaviors, and other topics germane to adolescents. In 2001-2002, when most of the respondents were between the ages of 18 and 26 years old, the third wave of data was collected. Given that most of the respondents were now young adults,

the questions asked at Wave 1 and Wave 2 were no longer age appropriate. As a result, the questionnaires were redesigned to include items that were pertinent to young adulthood. Participants, for instance, were asked about their lifetime contact with the criminal justice system, their marital status, and their childbearing history. More than 15,000 respondents participated in the Wave 3 component of the Add Health study (Harris et al., 2003).

One of the unique aspects of the Add Health sample is that at Wave 3 a subsample of respondents was asked to submit buccal cells for genotyping. Only those participants who had a sibling or a co-twin who was also taking part in the Add Health study were eligible to be included in the DNA subsample. Altogether, 2,574 respondents were genotyped, making the Add Health study one of the largest samples ever to include genotypic information. For reasons to be discussed shortly, the final analytical sample was restricted to White male participants.

Measures

MAOA

Most genes comprise two different copies: one copy is inherited maternally and one copy is inherited paternally. The exception to this rule is for genes that are located on the X chromosome. Males have one X chromosome and one Y chromosome, whereas females have two X chromosomes and no Y chromosomes. As a result, males have only one copy of any X linked gene, whereas females have two copies of all X-linked genes. Given the wide gender gap in offending behaviors, genes located on the sex chromosomes (i.e., the X chromosome and the Y chromosome) have been identified as some of the most promising candidate genes in the etiology of antisocial phenotypes (Ellis, 2005).

One X-linked gene that has been found to be related to crime and delinquency is the MAOA gene. The MAOA gene varies from person to person because of a section of DNA in the promoter region that can be repeated a variable number of times. Some people, for example, may inherit an MAOA gene with three repeats, whereas other people may inherit an MAOA gene with four repeats. These alternative copies of a gene (e.g., the different repeat lengths for MAOA) are referred to as alleles and, in the case of MAOA, the number of repeats (e.g., three repeats, four repeats, etc.) is what distinguishes one allele from another.

In general, the MAOA gene is divided into two groups: a low MAOA activity group and a high MAOA activity group. The low MAOA activity alleles have been found to increase the risk of developing a range of different maladaptive outcomes, including criminal and delinquent behaviors (Kim-Cohen et al., 2006). Following prior researchers analyzing the Add Health data (Haberstick et al., 2005), the low MAOA activity group comprised the 2- and 3-repeat alleles, and the high MAOA activity group comprised the 3.5-, 4-, and 5-repeat alleles. The MAOA variable is

thus a dichotomous variable, where respondents possessing low MAOA activity alleles were assigned a value of 1 and respondents possessing high MAOA activity alleles were assigned a value of 0.

Neuropsychological Deficits

To examine the effect of neuropsychological functioning on antisocial phenotypes, we included a measure of neuropsychological deficits. Prior researchers have employed an array of different scales to measure neuropsychological abnormalities (Cauffman et al., 2005), but tests that index verbal abilities are used quite frequently and have been endorsed by some of the leading life-course criminology scholars (Lynam, Moffitt, & Stouthamer-Loeber, 1993; Moffitt, 1990; Moffitt, Lynam, & Silva, 1994; Piquero, 2001). One verbal assessment battery that has been widely used, and that was available in the Add Health study, was the Peabody Picture Vocabulary Test (PPVT). The PPVT is a standardized test that has been shown to be a valid and a reliable way of measuring verbal skills and receptive vocabulary (D'Amato, Gray, & Dean, 1988; Dunn & Dunn, 1981) and has been shown to correlate with more direct measures of neuropsychological functioning (Quattrocchi & Golden, 1983). During Wave 1 interviews, respondents were administered the PPVT, and as a result, we used scores on the PPVT to evaluate neuropsychological functioning. Originally, scores on the PPVT were coded such that higher values indicated better verbal abilities. However, we reverse-coded the PPVT variable so higher scores reflected more neuropsychological deficits.

Low Self-Control Scales

Low self-control is one of the strongest and most consistent predictors of antisocial behaviors (Pratt & Cullen, 2000). Even so, there is dissension over the most reliable and valid way to measure individual variation in self-control (DeLisi, Hochstetler, & Murphy, 2003; Longshore, Stein, & Turner, 1998; Longshore, Turner, & Stein, 1996; Marcus, 2003, 2004; Piquero & Rosay, 1998). The most frequently employed measurement strategy is Grasmick et al.'s (1993) low self-control scale. Although there are a number of items in the Add Health sample that index levels of self-control, the variables composing the Grasmick et al. scale were not collected. Fortunately, the relationship between low self-control and antisocial behavior is not contingent on the use of the Grasmick et al. scale or any other individual measure (Pratt & Cullen, 2000).

Prior researchers analyzing the Add Health data have identified five items that measure low self-control (Beaver, 2008; Boutwell & Beaver, 2008; Perrone et al., 2004; Wright, Beaver, DeLisi, & Vaughn, 2008). Specifically, during Wave 1 interviews, respondents were asked whether they had trouble getting along with their teachers, trouble paying attention, trouble keeping their mind focused, and trouble finishing their homework. In the words of Perrone and her associates, "these questions tap into the simple tasks, physical activities, and impulsivity components of self-control"

(p. 302). In addition, a fifth question asked respondents to indicate whether they felt that they did everything just right. This last item indexes the self-centeredness component of low self-control (Perrone et al., 2004).

Responses to these items were then summed together to form the Wave 1 low self-control scale ($\alpha = .67$). The same questions were asked again during Wave 2 interviews, allowing us to make a duplicate Wave 2 low self-control scale ($\alpha = .65$). Both scales were coded such that higher values represented lower levels of self-control. The self-control scales were subjected to additional statistical tests to ensure they had appropriate psychometric properties. Principal components factor analysis revealed that all of the items loaded on one factor. Confirmatory factor analysis also indicated that the five items were significant indictors of one latent construct.[3] Reliability analysis showed that removing any of the items would reduce the overall value of Cronbach's alpha. Collectively, the results of these statistics suggest that the self-control scales are modestly reliable and valid.

Delinquency Scales

During Wave 1 and Wave 2 interviews, respondents were asked a series of questions pertaining to their involvement in a broad range of different delinquent behaviors. Respondents, for example, were asked how frequently in the past 12 months they had sold drugs, damaged property, stolen something from a store, and lied to their parents, among others. The response set to these questions was as follows: 0 (*never*), 1 (*one or two times*), 2 (*three or four times*), 3 (*five or more times*). In line with prior research (Beaver & Wright, 2005), we summed responses to these questions to create a 15-item Wave 1 delinquency scale ($\alpha = .84$) and a 14-item Wave 2 delinquency scale ($\alpha = .79$). Higher scores indicated more delinquent involvement.

Involvement in mundane forms of delinquency is relatively normal conduct among adolescents (Moffitt, 1993). Youths who engage in acts of serious physical violence, however, are much rarer and are more likely to persist with their antisocial behaviors into adulthood. They are, in other words, at risk for becoming career criminals or LCP offenders (DeLisi, 2005). To explore the factors that are associated with the development of these more serious types of delinquency behaviors, we identified a subset of questions (from the full delinquency scales) that measured serious physical violence. The Wave 1 violent delinquency scale was created from four different items that indexed physical fighting and weapon use ($\alpha = .72$). The Wave 2 violent delinquency scale was created from three different items that tapped physical fighting, weapon use, and involvement in gangs ($\alpha = .53$). These scales were coded such that higher scores reflected greater involvement in violent acts.

Control Variable

To control for the age-graded nature of delinquent involvement (Laub & Sampson, 2003) and of the development of self-control, we included age as a continuous variable

Table 1
Descriptive Statistics for Selected Add Health Variables and Scales for White Males

Measure	*M*	Skewness	*SD*	Range
Monoamine oxidase A	0.36	0.578	0.48	0-1
Neuropsychological deficits	33.26	0.272	12.84	3-75
Low self-control (Wave 1)	6.63	0.691	3.15	1-20
Low self-control (Wave 2)	6.37	0.650	3.08	1-17
Delinquency scale (Wave 1)	4.65	2.077	5.38	0-42
Delinquency scale (Wave 2)	2.79	2.847	3.92	0-37
Violent delinquency scale (Wave 1)	1.21	2.311	1.85	0-12
Violent delinquency scale (Wave 2)	0.29	3.658	0.71	0-7
Age (Wave 1)	16.08	–0.079	1.66	12-20

measured in years. To model the potentially nonlinear effect of age on the outcome measures, age squared was also included as a control variable in the analyses.

Plan of Analysis

The analysis for this article proceeded in a number of linked steps. First, we estimated a series of multivariate regression models employing the delinquency scales as dependent variables. As shown in Table 1, the delinquency scales were highly skewed, thereby violating one of the main assumptions of ordinary least squares (OLS) regression. Therefore, to provide more accurate estimates of overdispersed data, negative binomial regression was employed with the delinquency scales. Second, we calculated multivariate regression equations for the Wave 1 and Wave 2 low self-control scales. Table 1 reveals that these scales approximated a normal distribution, and as a result, we used OLS regression for the low self-control scales. To check the robustness of the models, we recalculated all of the OLS equations using negative binomial regression. The results were identical, and thus we present only the findings generated from OLS regression. Because some of the observations were not independent of each other (i.e., siblings from the same household), Huber/White standard errors were calculated for all of the models. Huber/White standard errors allow for unbiased tests of significance for the coefficients when observations lack independence.

All of the multivariate models included the MAOA variable and the measure of neuropsychological deficits as independent predictor variables in the analyses. In addition, we also created a multiplicative interaction term between MAOA and neuropsychological deficits to examine whether the effects of neuropsychological deficits are moderated by MAOA. To reduce problems with multicollinearity, the

MAOA variable and the neuropsychological deficits measure were mean centered prior to creating the interaction term (Jaccard, Turrisi, & Wan, 1990). Tolerance values and variance inflation factors were also estimated and revealed that multicollinearity was not problematic in any of the analyses.

Finally, we chose to restrict our sample to White males for two main reasons. First, the MAOA gene is thought to have its most powerful effects on males (because they have only one MAOA allele, not two like females). As a result, most geneticists investigating the association between MAOA and antisocial outcomes have used samples composed solely of males (Caspi et al., 2002), including researchers analyzing the Add Health data (Haberstick et al., 2005). We followed the lead of these researchers and removed females from the final analytical sample. Second, and in line with prior Add Health researchers (Haberstick et al., 2005), we removed all non-White respondents from the sample. Pooling together all racial categories is unadvised when examining the effects that measured genes have on a phenotype because of population stratification effects. We lacked sufficient statistical power to examine the statistical models for African American males. With this selection criteria in place, and after removing one monozygotic twin from each monozygotic twin pair (Haberstick et al., 2005), we were left with a final sample size of 767 White males.

Results

The analysis began by employing the Wave 1 delinquency scale as the dependent variable in a negative binomial regression equation. As shown in the far left column of Table 2, MAOA, neuropsychological deficits, age, and age squared were entered into the equation as predictor variables. Only age and age squared had a statistically significant effect on the dependent variable. In the next model, the MAOA × Neuropsychological Deficits multiplicative interaction term was introduced. The results of this model revealed that the interaction between MAOA and neuropsychological deficits exerted a statistically significant and positive effect on delinquent involvement at Wave 1. In the third equation, the Wave 1 low self-control scale was introduced into the model. It was the strongest predictor of delinquency. The inclusion of the low self-control scale attenuated the effect of the MAOA × Neuropsychological Deficits term.

The results of the analysis thus far suggest that MAOA and neuropsychological deficits interact to predict delinquent involvement at Wave 1. We next turn our attention to the models predicting the Wave 2 delinquency scale. These equations are presented in the right-hand columns of Table 2. Similar to the results for the Wave 1 delinquency scale, neither the MAOA variable nor the neuropsychological deficits measure had an effect on the Wave 2 delinquency scale. Once again, however, the MAOA × Neuropsychological Deficits interaction term had a statistically significant and positive effect on delinquent involvement even after controlling for the Wave 2 low self-control scale. The Wave 2 low self-control scale was the strongest predictor

Table 2
Negative Binomial Regression Equations Predicting Adolescent Delinquency for White Males

	Delinquency at Wave 1						Delinquency at Wave 2					
Measure	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*
MAOA	.03	.09	.09	.10	.08	.11	–.05	.10	.04	.11	.12	.11
Neuropsychological deficits	.01	.00	.01*	.00	.00	.00	–.00	.00	–.01	.00	–.00	.00
MAOA × Neuropsychological Deficits			.02*	.01	.01†	.01			.02*	.01	.02*	.01
Contemporaneous low self-control					.12*	.01					.14*	.02
Age	1.01*	.47	.95*	.47	.76	.47	1.76*	.67	1.64*	.68	1.62*	.64
Age^2	–.03*	.01	–.03*	.01	–.02	.02	–.06*	.02	–.05*	.02	–.05*	.02

Note: Huber/White standard errors presented. MAOA = monoamine oxidase A.
*p = .05, two-tailed. †p = .10, two-tailed.

of the Wave 2 delinquency scale. Taken together, the models estimated in Table 2 provide initial evidence that MAOA and neuropsychological deficits interact to predict variation in both the Wave 1 and Wave 2 delinquency scales.

We next extended the analyses by using the Wave 1 and Wave 2 violent delinquency scales as dependent variables in a series of negative binomial regression equations. The left-hand column of Table 3 shows that MAOA did not have a significant main effect on the Wave 1 violent delinquency scale, but the neuropsychological deficits variable was positively related to the outcome measure. Specifically, respondents with more neuropsychological deficits also reported being more involved in violent behaviors. Next, the MAOA × Neuropsychological Deficits interaction term was entered into the equation. Consistent with the models estimated in Table 2, the MAOA × Neuropsychological Deficits interaction term had a statistically significant and positive effect on the Wave 1 violent delinquency scale. Once the effects of the Wave 1 low self-control scale were held constant, the MAOA × Neuropsychological Deficits interaction was no longer a statistically significant predictor of violent delinquency. Not surprisingly, low self-control maintained a positive association with the dependent variable.

The right-hand columns of Table 3 display the results of the equations using the Wave 2 violent delinquency scale as the dependent variable. Across all of the models, the neuropsychological deficits measure was positively associated with violent delinquency. Of particular importance was that the MAOA × Neuropsychological Deficits had a statistically significant effect on the Wave 2 violent delinquency scale. This interaction effect, however, was no longer statistically significant once the effects of the Wave 2 low self-control scale were held constant. As with all of the

Table 3
Negative Binomial Regression Equations Predicting Adolescent Violent Delinquency for White Males

	Violent Delinquency at Wave 1						Violent Delinquency at Wave 2					
Measure	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*
MAOA	.11	.12	.17	.12	.13	.13	.03	.19	.06	.19	.17	.20
Neuropsychological deficits	.01*	.00	.02*	.00	.01*	.00	.03*	.01	.03*	.01	.03*	.01
MAOA × Neuropsychological Deficits			.02*	.01	.01	.01			.03**	.01	.02	.01
Contemporaneous low self-control					.11*	.02					.11*	.03
Age	.29	.65	.21	.65	–.05	.66	3.01*	1.12	2.94*	1.12	2.89*	1.25
Age^2	–.01	.02	–.01	.02	–.00	.02	–.10*	.04	–.09*	.04	–.09*	.04

Note: Huber/White standard errors presented. MAOA = monoamine oxidase A.
*p = .05, two-tailed. **p = .061.

other models, the low self-control scale had a substantive effect on the violent delinquency scale.

The models calculated in Tables 2 and 3 revealed two main findings. First, MAOA interacted with neuropsychological deficits in the prediction of the two delinquency scales and in the prediction of the two violent delinquency scales. Second, the low self-control scale also had statistically significant and positive effects on all four of the delinquency scales. Still, these analyses did not explore the ways in which MAOA and neuropsychological deficits may contribute to levels of self-control. We thus turn to the models that examine these interrelationships.

To explore the effects that MAOA and neuropsychological deficits had on levels of self-control, we calculated OLS regression equations using the Wave 1 and the Wave 2 low self-control scales as dependent variables. The left-hand columns of Table 4 reveal the results of the equations predicting the Wave 1 low self-control scale. In line with the other models, the MAOA × Neuropsychological Deficits interaction term had a statistically significant and positive effect on the Wave 1 low self-control scale. An almost identical set of findings was reported for the Wave 2 low self-control scale: MAOA and neuropsychological deficits interacted to predict variation in the dependent variable.[4]

Discussion

This study tested the merits of three of the most influential pieces of criminological scholarship: Gottfredson and Hirschi's (1990) *A General Theory of Crime,*

Table 4
OLS Regression Equations Predicting Adolescent Low Self-Control for White Males

	Low Self-Control at Wave 1				Low Self-Control at Wave 2			
Measure	*B*	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*
MAOA	.28	.24	.45†	.26	.05	.25	.24	.27
Neuropsychological deficits	.02†	.01	.02*	.01	.01	.01	.02	.01
MAOA × Neuropsychological Deficits			.04*	.02			.04*	.02
Age	2.37*	1.14	2.27*	1.14	3.59*	1.27	3.47*	1.27
Age^2	–0.07*	0.04	–0.07†	0.04	–0.11*	0.04	–0.11*	0.04

Note: Huber/White standard errors presented. OLS = ordinary least squares; MAOA = monoamine oxidase A.
*p = .05, two-tailed. †p = .10, two-tailed.

Moffitt's (1993) developmental taxonomy, and Caspi et al.'s (2002) Gene × Environment interaction study. Analysis of the Add Health data revealed three main findings. First, and consistent with the extant literature, MAOA did not have a significant direct effect on any of the delinquency scales or on the two low self-control scales. In contrast, the measure of neuropsychological deficits was a statistically significant predictor of violent delinquency, even after controlling for the effects of low self-control.

Second, neuropsychological deficits interacted with MAOA to predict delinquent involvement cross-sectionally and longitudinally. The Neuropsychological Deficits × MAOA interaction had effects on a global measure of delinquency as well as a more restricted delinquency scale that only included measures of serious violence. However, this interaction effect was attenuated and, in some models, reduced to statistical insignificance once measures of low self-control were introduced into the equations.

Third, the Neuropsychological Deficits × MAOA term was a statistically significant predictor of the Wave 1 low self-control scale and the Wave 2 low self-control scale. The evidence thus revealed that low self-control partially mediated the link between the Neuropsychological Deficits × MAOA interaction and delinquent involvement. This is an especially intriguing finding as it suggests that genetic factors may exert part of their influence on criminogenic traits, and these traits, in turn, affect the propensity to engage in antisocial behaviors. Future research should begin to examine whether other criminogenic traits mediate the effect between genetic markers and antisocial behaviors. As for now, our findings draw attention to the close interconnections among low self-control, delinquent involvement, neuropsychological dysfunctions, and MAOA.

At first glance it might seem as though Gottfredson and Hirschi's (1990) theory, Moffitt's (1993) theory, and Caspi et al.'s (2002) study have very little in common. A close reading of their works, however, opens up some possibilities for empirical integration. From our perspective, the common thread linking all of them appears to be found in the brain. For instance, recent criminological research has provided some evidence that self-control is housed in the brain (Beaver, Wright, & DeLisi, 2007; Wikström & Treiber, 2007; Wright & Beaver, 2005). The role of neuropsychological functioning is a critical factor in Moffitt's theory, and recent genetic imaging studies reveal that variants of the MAOA genotype are associated with differences in brain structure (Meyer-Lindenberg et al., 2006). Of course, the findings reported here need to be replicated, but they do indicate that a much more explicit focus needs to be placed on how neural functions and processes may relate to criminal propensities. In this way, we echo the viewpoint of Moffitt (2005), who called for more research into the relationship between specific genes, correlations between vulnerability genes and risky environments, and interactions between them.

There are three limitations of our study that need to be addressed. First, only a subsample of respondents was genotyped, which necessarily raises questions about the generalizability of the findings. However, it is important to underscore the fact that researchers have analyzed the allelic frequencies of the MAOA gene in the Add Health data and found them to be similar to those reported in other samples, including the sample analyzed by Caspi and his colleagues (Haberstick et al., 2005).

Second, we did not include a direct measure of LCP offending and instead employed a violent delinquency scale. We opted not to create a measure of LCP offending because the Add Health study is a community-based sample and community-based samples typically do not include an adequate number of LCP offenders (DeLisi, 2005), which reduces statistical power. The measures of violent delinquency, however, contained enough variation to perform statistical analyses with sufficient power. Nonetheless, whether the findings reported here would generalize to LCP offenders remains an open-empirical question that is deserving of research attention.

Third, similar to prior research (e.g., Caspi et al., 2002; Haberstick et al., 2005), the analyses were restricted to White males. To stay consistent with extant research, and because we lacked statistical power, we were unable to examine the effects of MAOA on minorities. Likewise, we did not examine MAOA in females because MAOA is X-linked, meaning that females have two copies of this gene, whereas males have only one. As a direct result, the effect of MAOA on antisocial behaviors is thought to be confined to males. Future researchers should address the possibility that MAOA has effects that may extend to females.

In closing, current findings are supportive of an interdisciplinary scientific approach to the study of crime and delinquency, which views antisocial behavior as a multifactorial phenotype that is the result of genetic factors, neural substrates, and environmental factors working independently and synergistically. Unfortunately there is a relative dearth of criminological studies that have attempted to model empirically the ways in which genes and the environment interlock to produce

delinquent behaviors. Instead contemporary social science research continues to view behavior as either being due to nature or nurture and fails to recognize the complex interplay between the two. An abundance of research has shown that the nature/nurture distinction is a false dichotomy and the most fruitful approach to the study of crime would be to employ an interdisciplinary perspective that specifies the close linkages among antisocial behaviors, environmental factors, and genetics.

Notes

1. We realize that Moffitt and Caspi were not trained as criminologists per se, but they have contributed greatly to the criminological literature. As a result, we refer to them as criminologists in this study because we are focusing on their contributions to the criminological literature. We thank an anonymous reviewer for bringing this important point to our attention.

2. Although Caspi et al.'s (2002) study, Moffitt's (1993) theory, and Gottfredson and Hirschi's (1990) theory all emphasize the role that parents play in the development of antisocial phenotypes, we opted not to model parental effects for three main reasons. First, our study represents an initial effort to identify some, but not all, of the theoretical linkages among these three perspectives. By no means do we claim that the integrated model that we present here is the only way that these perspectives can be linked together; rather, we chose to provide a cautious first step toward showing some of the theoretical overlap that does exist across these perspectives. Future researchers should build on and extend our work by explicating the different ways in which parental socialization techniques can be integrated into a unified perspective. Second, the effects that parents have, at least in an integrated model, would likely be specified as moderating effects. Two-way interactions are inherently difficult to detect and higher order interactions are even more difficult to detect. Third, prior research analyzing the Add Health data has revealed that after genetic factors are held constant, parental socialization variables have little effect on delinquent involvement and levels of self-control (Wright, Beaver, DeLisi, & Vaughn, 2008). In the current analysis we were unable to control for heritability and thus our models would be misspecified (Wright & Beaver, 2005). Taken together, these three reasons provided the impetus for why parenting effects were not modeled directly in our study.

3. For the principal components analysis, the eigenvalue for the Wave 1 low self-control scale was 2.176 and this single factor accounted for 43.52% of the variance. No other factor had an eigenvalue greater than 1, indicating the presence of a single factor. For the Wave 2 low self-control scale, the eigenvalue was 2.082 and no other factor had an eigenvalue of greater than 1.0. This single factor explained 41.64% of the variance. In confirmatory factor analysis, all of the observable indicators had statistically significant loadings for the latent factor. We should also mention that correlation between low self-control and the delinquency scales hovers around $r = .2$ to $r = .3$. These correlations are in line with the average effect size reported by Pratt and Cullen (2000) in their meta-analysis of the low self-control/delinquency nexus. Thus, the low self-control scales also have predictive validity.

4. All of the models were recalculated controlling for whether the respondent received public assistance. The substantive results remained unchanged; however, given that including this measure reduced the sample size significantly, we opted to present the models that did not control for public assistance. Other measures tapping socioeconomic status were also explored, and again the pattern of findings was unaltered.

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