

Genes, Parenting, Self-Control, and Criminal Behavior

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Abstract

Self-control has been found to predict a wide variety of criminal behaviors. In addition, studies have consistently shown that parenting is an important influence on both self-control and offending. However, few studies have examined the role that biological factors may play in moderating the relationship between parenting, self-control, and offending. Using a sample of adolescent males drawn from the National Longitudinal Study of Adolescent Health ($N = 3,610$), we explore whether variants of the monoamine oxidase A gene (MAOA) and the dopamine transporter (DAT1) gene interact with parenting to affect self-control and offending. Results reveal that parenting interacts with these genes to influence self-control and offending, and that the parenting-by-gene interaction effect on offending is mediated by self-control. The effects of parenting on self-control and offending are most pronounced for those who carry plasticity alleles for both MAOA and DAT1. Thus, MAOA and DAT1 may be implicated in offending because they increase the negative effects of parenting on self-control. Implications for theory are discussed.

Keywords

crime, gene–environment interactions, self-control theory, parenting

In the past 20 years, Gottfredson and Hirschi's (1990) General Theory of Crime has become one of the most debated and tested theories in the field of criminology. Substantial evidence has emerged for the central claim that low self-control is strongly correlated with criminal and deviant behavior (Cohn & Farrington, 1999; Pratt &

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Cullen, 2000). Self-control is argued to emerge in childhood as a result of effective parenting and to remain relatively stable over the life course. Importantly, however, numerous studies find that parenting style is not the only factor influencing the development of self-control (Burt, Simons, & Simons, 2006; Teasdale & Silver, 2009; Wright, Beaver, Delisi, & Vaughn, 2008). This raises questions regarding what other factors may play a role in determining levels of self-control and in turn the extent of involvement in criminal (and analogous) behaviors.

We integrate self-control theory with a biosocial framework in which psychological and behavioral outcomes are the result of an interaction between the social environment and genetics. The integration of self-control theory and gene–environment interactions ($G \times E$) is a natural extension of recent findings in a variety of fields that show that certain genes, in combination with negative environmental triggers, are related to neuropsychological deficits such as attention deficit hyperactivity disorder (ADHD; Schilling, Walsh, & Yun, 2011) and low self-control (Belsky & Beaver, 2011) on one hand, and aggression and criminal offending (Caspi et al., 2002; Guo, Roettger, and Shih, 2007) on the other. Genes are related to neuropsychological and behavioral outcomes because they influence brain processes related to attention and learning (Schilling et al., 2011; Simons et al., 2011b).

Specifically, this study explores the way in which variants (alleles) of the monoamine oxidase A gene (MAOA) and the dopamine transporter (DAT1) gene interact with parenting to decrease levels of self-control and increase offending in a sample of adolescent males. Recent studies and reviews have reported that these genes are related to neuropsychological deficits and offending in this population (Caspi et al., 2002; Guo, Roettger, & Shih, 2007; Kim-Cohen et al., 2006; Schilling, Walsh, & Yun, 2011). The analysis also examines whether $G \times E$ effects behave in a fashion proposed by the cumulative plasticity hypothesis, which predicts that the more “plasticity” alleles an individual carries, the more susceptible they will be to environmental influences (Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007; Belsky et al., 2009; Belsky & Pluess, 2009).

The current study tests two key implications of this biosocial/self-control model. First, we test whether parenting has especially pronounced effects on (low) self-control among carriers of the risk alleles associated with MAOA and DAT1, and whether this $G \times E$ effect in turn influences levels of self-reported offending. Second, in line with predictions of self-control theory, we assess whether self-control mediates the $G \times E$ effect on involvement in criminal behavior. These predictions are tested with a sample of males drawn from the National Longitudinal Study of Adolescent Health (Add Health). Before presenting the analysis, we explicate our theoretical model more fully in the following sections.

Parenting, Self-Control, and Criminal Offending

Self-control theory posits a very parsimonious explanation for why certain individuals commit crimes fairly frequently and others almost never commit crime over the life course: Some individuals are low in self-control, and those low in self-control will be

inclined to commit crimes when presented with the opportunity. The definition of self-control within Gottfredson and Hirschi's theory is controversial because they never define it separately from the propensity to commit crime, but they do lay out a number of traits associated with low self-control. The person low in self-control lacks diligence, is impulsive, a thrill-seeker, unable to defer gratification, physical (as opposed to mental), and self-centered (Gottfredson & Hirschi, 1990).

Self-control theory posits that the primary source of self-control is parenting. Effective child-rearing results in children high in self-control, while ineffective child-rearing results in children with low self-control. In self-control theory effective and adequate parenting involves monitoring children's behavior, recognizing antisocial behavior, and correcting this behavior. Parents who engage in these parenting practices will produce children who are capable of delaying gratification, are sensitive to the needs of others, are willing to accept restraint, and are unlikely to use force or violence to attain gratification from others (Gottfredson & Hirschi, 1990). Some scholars, however, have been critical of self-control theory's low bar for parenting, noting that monitoring and discipline are most effective when paired with warmth and nurturance (Burt et al., 2006).

The overall evidence supporting the importance of self-control in crime causation that has accrued in the criminological literature in the past 20 years has been impressive. Much empirical evidence supports the central concepts of the theory, namely, that parenting is a key source of self-control and that self-control is a strong predictor of both crime and analogous behaviors (Akers & Sellers, 2009; Grasmick et al., 1993; Perrone, Sullivan, Pratt, & Margaryan, 2004). In a widely cited article, Pratt and Cullen (2000) conduct a meta-analysis of 21 empirical studies of self-control theory and find that self-control has consistent and strong effects on crime and analogous behaviors. A more recent meta-analysis by de Ridder et al. (2012) confirms that self-control is related to a host of antisocial behaviors as well as prosocial outcomes (i.e., academic achievement, emotional regulation). Other recent empirical tests add to self-control theory's base of empirical support (Boutwell & Beaver, 2010; Felson & Staff, 2006; Hay, 2001; Hay & Forrest, 2006; Nofziger, 2008).

Some tests of self-control theory, however, find that Gottfredson and Hirschi's theorizing about the relationship between parenting and self-control falls short, and may need elaboration. Burt et al. (2006) find that low self-control only partially attenuates the effect that poor parenting has on delinquency in a sample of African Americans. Simons, Simons, Chen, Brody, and Lin (2007) find that parenting is not the only factor related to low self-control, as family socioeconomic status (SES) also significantly predicts levels of self-control. In another study, Wright et al. (2008) find negligible effects of parenting on self-control. A consideration of the role of genetics in self-control theory may be an important elaboration if genes indeed moderate the effects of parenting on self-control and offending, a possibility we explore below.

G × E Effects and Cumulative Genetic Plasticity

Much of the research looking at the genetic basis of neuropsychological functioning and antisocial behavior has focused on variations in genes involved in the regulation

of neurotransmitters, such as serotonin and dopamine. Two such genes that have received much attention in this literature are the MAOA and DAT1. The MAOA gene is located on the X chromosome. It encodes the MAOA enzyme, which metabolizes neurotransmitters, including norepinephrine, serotonin, and dopamine and therefore plays a key role in regulating behavior (Belsky & Pluess, 2009). The MAOA gene has been infamously referred to as the “warrior gene” due to its observed relationship with aggressive and violent behavior (Beaver, DeLisi, Vaughn, and Barnes, 2010). The relationship between MAOA and antisocial behavior has only held in research on males. The MAOA gene is found on the X chromosome and males have only a single copy; females by contrast have two copies, so even if one copy of the gene is “defective” the other copy may compensate (Beaver et al., 2010; Simons et al., 2011).¹

Most studies looking at the link between MAOA and antisocial behavior find that environmental stressors have the most pronounced effects among individuals carrying the low-activity version of MAOA (the 2R and 3R alleles). In an influential study looking specifically at $G \times E$ effects involving MAOA, Caspi et al. (2002) find that young males in a sample of New Zealanders with the low-activity version of MAOA were most effected by childhood maltreatment in regard to their later antisocial behavior and aggression. Males in the sample with the high-activity version of MAOA who had also been the victims of childhood maltreatment displayed substantially less antisocial behavior later in life. It is important to note, however, that other researchers have failed to replicate the findings of Caspi and colleagues concerning MAOA, childhood maltreatment, and antisocial behavior utilizing various other samples (Haberstick et al., 2005; Young et al., 2006). In another study looking specifically at childhood maltreatment and MAOA, Kim-Cohen et al. (2006) find that 7-year-old boys with the low-activity variant of MAOA who had been abused were rated by their mothers and teachers as having more attention deficits than their abused peers with the high-activity version of MAOA. A number of other studies also reveal a significant interaction between MAOA and environmental adversity. In a large longitudinal study of adolescent twin boys, Foley et al. (2004) find that boys with the low-activity variant of MAOA are more likely than their high-activity carrying peers to be diagnosed with conduct disorder when exposed to high levels of childhood adversity. Nilsson et al. (2006) report similar results in a cross-sectional study, finding that maltreatment and living arrangement experiences were most related to criminal behavior among carriers of the low-activity variant of MAOA. Other studies have produced similar findings (Ducci et al., 2008; Widom & Brzustowicz, 2006).

More recent studies have also produced evidence $G \times E$ effects on antisocial behavior involving MAOA. Fergusson, Boden, John, Miller, and Kennedy (2011, 2012) find in two separate studies that MAOA moderated the effects of childhood maltreatment and school failure on property and violent offending and number of criminal convictions in later adolescence. Beaver et al. (2010) find in a study of White males in the Add Health data that the effect of verbal ability on self-control and delinquency is moderated by MAOA genotype. In a study looking at desistance, Beaver, Wright, DeLisi, and Vaughn (2008) find that several genes, including MAOA and DAT1, interact with marital status to predict patterns of desistance among males in the Add Health sample.

In addition, numerous studies in the $G \times E$ literature focus on the DAT1. DAT1 largely determines the magnitude and duration of synaptic dopamine signaling in the brain. It does this by transporting dopamine from the synaptic cleft back into the pre-synaptic knob for repackaging and reuse after it is finished exciting downstream neurons (Schilling et al., 2011). A particular variation of DAT1 (the 10R allele) is more efficient in this reuptake process than other variations, which means there is less dopamine in the synaptic cleft available for activation (Miller-Butterworth et al., 2008). This is problematic because dopamine activates pleasure centers in the brain, so it being too rapidly cleared from the synaptic cleft leads the individual to seek out pleasures that raise dopamine levels, whether legal or illegal.

Several studies look at the relationship between DAT1, the social environment, and sensation seeking and antisocial behaviors. Guo, Tong, and Cai (2008) find that the 10R allele of DAT1 has direct effects on the number of sexual partners that White males report having in the Add Health sample, and that the proportion of students in one's school who were having sex by age 16 exacerbates this relationship. Stevens et al. (2009) find that DAT1 moderates the effect of institutional deprivation on ADHD symptoms in a sample of children in Romanian orphanages, with those carrying 10R alleles showing more ADHD symptoms at 6, 11, and 15 years of age. Beaver and Belsky (2012) find that among other genes, DAT1 is associated with the intergenerational transmission of parenting practices. Specifically, carriers of the 10R allele and other plasticity (i.e., risk) alleles experience the highest levels of parenting stress when they themselves were exposed to negative maternal parenting (Beaver & Belsky, 2012). Vaughn, DeLisi, Beaver, and Wright (2009) find that DAT1 (along with 5-HTT) interacts with delinquent peer networks to predict chronic, serious criminal behavior in the Add Health sample. Other studies report similar results concerning DAT1 and antisocial outcomes (Beaver, Wright, & Walsh, 2008; Guo, Roettger, & Shih, 2007; Guo, Tong, et al., 2007).

In sum, studies show that MAOA and DAT1 moderate (i.e., amplify) the effects of environmental adversity on the development of self-control and antisocial behavior. But why do genes cause some individuals to engage in more risky and criminal behaviors when their environment is stressful? Belsky and Pluess (2009) observe that genes such as MAOA and DAT1 are related to the dopaminergic and serotonergic systems, with the dopaminergic system implicated in reward sensitivity and the serotonergic system implicated in sensitivity to punishment and displeasure (Simons et al., 2011b). Thus, some individuals, given their genetic makeup, are more responsive to environmental influence because of their different thresholds for experiencing pleasure and displeasure, and are therefore more readily shaped by rewards and punishments than are others. This differential susceptibility hypothesis posits that some individuals are more "malleable" or "plastic" based on their genetic makeup. The dominant diathesis-stress hypothesis, in contrast, has focused more narrowly on identifying genes that render some individuals more "vulnerable" or at risk of negative outcomes when exposed to environmental adversity.

The key difference between the differential susceptibility and diathesis-stress hypotheses thus concerns what happens when people are exposed to favorable

environmental conditions. While both models agree that certain individuals are worse off in unfavorable environments due to genetics, the two models differ in their predictions about what happens to these same individuals in favorable environments. While the diathesis-stress hypothesis predicts that individuals who are “vulnerable” genetically will have similar outcomes to their non-vulnerable peers in favorable environments, the differential susceptibility model argues that these more “plastic” individuals should actually fair better than their non-plastic peers. So while the differential susceptibility hypothesis predicts that the environment can affect individuals in a for-better-and-worse fashion, the diathesis-stress model focuses only on differences in individuals when they face unfavorable environments.

Belsky and Pluess (2009) further suggest that the more plasticity alleles an individual carries, the more susceptible to their environment they will be. This is known as the cumulative plasticity hypothesis. For example, parenting effects on self-control as well as offending should be most pronounced among carries of plasticity alleles for both MAOA and DAT1, compared with carries of a plasticity allele for either MAOA or DAT1 alone or those who do not carry the plasticity alleles at all. Several recent studies provide support for this hypothesis (Beaver & Belsky, 2012; Belsky & Beaver, 2011; Simons et al., 2011; Simons et al., 2012), which we will assess in the analysis presented below.

The Current Study

The current study extends past $G \times E$ studies of antisocial behavior in two important ways. First, we elaborate self-control theory via integration with a $G \times E$ approach, whereby parenting has more pronounced effects on self-control and offending among carriers of the risk alleles associated with MAOA (the 2R and 3R alleles) and DAT1 (the 10R allele). Specifically, we expect that males who carry the 2R or 3R allele of MAOA and are homozygous for the 10R allele of DAT1 will evidence the lowest levels of self-control and in turn higher levels of criminal offending when they experience a poor relationship with their primary caregiver, compared with males who carry neither of these plasticity alleles. In turn, drawing on recent research showing that low self-control partly mediates parenting effects on behavior, we expect that in our elaborated model low self-control will mediate a significant portion of the effect of the interaction between parenting and genotype on self-reported offending. Thus, parenting and genotype will interact (our $G \times E$) to shape levels of self-control and offending behavior, with self-control mediating the $G \times E$ effect on offending behavior.

The second significant expansion on past $G \times E$ studies is in testing Belsky and Pluess' (2009) proposition regarding cumulative genetic plasticity. The cumulative plasticity hypothesis predicts that carriers of plasticity alleles for both MAOA and DAT1 should show greater susceptibility to their environment than carriers of one or no plasticity alleles. With some recent exceptions (see Beaver & Belsky, 2012; Belsky & Beaver, 2011; Simons et al., 2011; Simons et al., 2012), few studies in the $G \times E$ literature focus on the cumulative influence of MAOA and DAT1 in amplifying the effects of environmental adversity on neuropsychological and behavioral outcomes.

We draw on these expectations from self-control theory and the cumulative plasticity hypothesis to derive hypotheses about the relationship between parenting, genetics, self-control, and offending. Self-control theory predicts that parenting (i.e., poor parent-child relationship [PPCR]) significantly influences levels of self-control (Hypothesis 1), while the cumulative plasticity hypothesis predicts that the effects of negative parenting on self-control will be most evident among males who carry plasticity alleles for both MAOA and DAT1 (Hypothesis 2). Hypotheses 3 to 4 make identical predictions with self-reported offending as the dependent variable. Drawing on self-control theory, Hypothesis 5 predicts that low self-control will significantly mediate the parenting by genotype interaction effect on criminal offending.

Data and Method

Sample

The analysis draws on data from Waves I, II, and IV of the Add Health. Add Health is a nationally representative sample of adolescents who were first recruited during the 1994-1995 school year while they were in Grades 7 to 12 (Harris et al., 2003; Udry, 1998). Add Health obtained a nationally representative sample of adolescents by utilizing a multistage stratified sampling process to select 80 high schools and 52 middle and junior high schools for inclusion in the study. More than 90,000 students completed in-school self-report surveys, and of this group a subsample was randomly chosen for the Wave I in-home component of Add Health. In total, 20,745 adolescents and 17,700 of their primary caregivers participated in the Wave I in-home component (Harris et al., 2003). Wave II data collection occurred approximately 1 to 2 years after Wave I data collection, Wave III data were collected during 2001-2002 when respondents were between 18 and 26 years old, and Wave IV data were collected during 2007-2008 when respondents were between 24 and 32 years old.

During Wave IV in-home interviews Add Health took saliva swabs from all interviewees for DNA analysis. In conjunction with the Institute for Behavioral Genetics (IBG) in Boulder, Colorado, Add Health genotyped Wave IV interviewees for a set of genetic markers of interest to biosocial researchers. The final sample for analysis includes 3,610 male respondents interviewed at Waves I, II, and IV who had complete data across waves. That Add Health is a large and nationally representative sample that includes information on genetics as well as social environments makes it well suited for the present analysis.

Measures

Criminal behavior. The dependent variable consists of nine items drawn from Wave II that ask respondents about various criminal activities they engaged in during the prior year. These items are a mixture of violent and property offending. Measures of violence included questions asking how often respondents used or threatened to use a weapon on someone to get something from them, pulled or actually used a knife or gun

on someone, used a weapon during a fight, or hurt someone so badly in a fight that they needed medical attention. Property offending measures included asking respondents how often they painted graffiti, stole cars, burglarized buildings, and stole items worth more than US\$50. A fuller description of these measures can be found in the appendix. This scale is closely related to scales that other researchers have developed for use in the Add Health data set (Guo, Roettger, and Cai, 2008; Hagan & Foster, 2003; Haynie, 2001, 2003). We summed these nine items into a global measure of involvement in criminal activity in the past year ($\alpha = .74$). We log this measure due to extensive skew, resulting in a scale with a relatively normal distribution (higher scores indicate more offending).

PPCR. The measure of parenting consists of five items drawn from Wave I. These items focus on the respondent's relationship with their mother, and include asking how close they feel to their mother, how much they think she cares about them, whether she is warm and loving most of the time, whether they are satisfied with how they and their mother communicate, and their overall satisfaction with their relationship with their mother. We summed these five items to create a measure of PPCR ($\alpha = .81$). We transformed this scale by standardizing it to facilitate interpretation of the $G \times E$ interaction terms. Higher scores on this scale indicate less maternal warmth and more maternal disengagement.

This scale of parenting is limited because it does not directly measure parental monitoring or disciplinary practices, which are key concepts in self-control theory that relate to an adolescent's level of self-control (Gottfredson & Hirschi, 1990). These types of items are unfortunately fairly limited in the Add Health data set. Yet parental warmth, attachment, monitoring, and disciplinary style are all highly correlated (Simons & Burt, 2011; Simons et al., 2007), and the parents of low self-control individuals usually evince a combination of a lack of warmth, monitoring, and consistent discipline (Burt et al., 2006). Prior research has shown that these items have predictive validity in regard to levels of self-control (Belsky & Beaver, 2011).

While an ideal measure of the parent-child relationship (i.e., the environment) would range from "bad" to "good," our measure more approximates a range of "bad" to "not bad." Note that this limits our ability to differentiate between the diathesis-stress and differential susceptibility models of genetic effects, and thus we take a conservative approach and refrain from making claims as to which model the results best support.

Low self-control. The appropriate method for measuring self-control has been the source of much debate in the criminological literature (Beaver, DeLisi, Mears, & Stewart, 2009; Longshore, 1998; Longshore & Turner, 1998). In the current study, we use a slightly altered version of a scale of low self-control developed by Beaver et al. (2009) for use in the Add Health survey. This low self-control scale contains 21 items, detailed in the appendix, from both parent and self-report responses from Wave I interviews. The items in this scale measure a respondent's temper, self-centeredness, attention span, and use of rational decision making. A composite measure of self-control was

created by summing these items ($\alpha = .71$). The scale is normally distributed with higher scores indicating lower levels of self-control. Prior research has shown that this scale has predictive validity (Beaver et al., 2009; Belsky & Beaver, 2011).

MAOA/DAT1 dummy variables. The literature on MAOA has revealed that two low-activity versions of this gene (2R and 3R) are associated with negative behavioral and mental health outcomes among males (Belsky & Pluess, 2009; Caspi et al., 2002; Kim-Cohen et al., 2006). Following past research, we code MAOA to reflect the non-presence (0) or presence (1) of either the 2R or 3R allele. Based on this coding, about 60% of respondents in this sample were not carriers of the 2R or 3R alleles, while 40% of respondents were carriers of either the 2R or 3R allele. DAT1 has a 40-base pair (bp) variable number of tandem repeats that can be repeated 3 to 11 times (Beaver et al., 2008). Past research has shown that males who are homozygous for the 10R allele of DAT1 (10R/10R) are significantly more susceptible to a number of behavioral and psychological problems (Beaver et al., 2008; Guo et al., 2007; Schilling et al., 2011). Accordingly, we code DAT1 to reflect the non-presence (0) or presence (1) of two 10R alleles. Based on this coding, 41% of respondents in this sample had an allelic combination other than 10R/10R, and 59% of respondents were 10R homozygotes.

We combined the MAOA and DAT1 measures into a set of dummy variables distinguishing the number and type of plasticity alleles an individual carries. We defined these dummy variables as follows: two plasticity alleles = carriers of the 2R or 3R alleles of MAOA and 10R-allele DAT1 homozygotes (25% of the sample); MAOA only = carriers of the 2R or 3R alleles of MAOA and not 10R-allele DAT1 homozygotes (16%); DAT1 only = 10R-allele DAT1 homozygotes and no 2R or 3R MAOA allele (34%). The reference category includes those who do not carry any of the plasticity alleles associated with MAOA and DAT1 (25%). The Hardy–Weinberg equilibrium test shows that the distribution of DAT1 among this sample does not differ significantly from that predicted on the basis of simple Mendelian inheritance.²

Controls. We also include in all analyses several general controls derived from Wave 1 that have been shown to be correlated with involvement in crime: age; dummy variables for Hispanic, non-Hispanic Black, Native American, Asian, and Other (with non-Hispanic White as the reference category); parent's education (1 = 4-year degree or more); and parent receiving public assistance (1 = yes). About 56% of the sample is non-Hispanic White, 15% Hispanic, 18% non-Hispanic Black, with the remainder comprising Native American (3%), Asian (8%), and members of other racial/ethnic groups (1.1%). About 27% of parents are college graduates, while 7% report receiving public assistance. We also control for affiliations with delinquent peers, a well-known correlate of offending. This concept is represented by three items that measure a respondent's association with substance-using friends. Specifically, Add Health respondents were asked at Wave I how many of their three closest friends smoke at least one cigarette per day, drink alcohol at least once a month, and smoke marijuana at least once a month. We standardized these three items and summed them to create the measure of affiliation with substance-using peers ($\alpha = .74$).

Analytic Strategy

We test hypotheses using ordinary least squares (OLS) regression, given that the outcome variables are fairly normally distributed. The models test whether a PPCR significantly influences levels of self-control (Hypothesis 1), and whether this effect is amplified among carriers of the plasticity alleles of MAOA and DAT1 (Hypothesis 2). We further test these predictions using self-reported offending as the outcome (Hypotheses 3-4). Finally, we test whether low self-control in turn mediates the $G \times E$ effect on levels of criminal offending (Hypothesis 5). We utilize the appropriate weight, cluster, and strata variables in all analyses to account for the complex Add Health survey design. Tests using variance inflation factors (VIFs) showed that multicollinearity was not a problem in any of the equations.³ All of the various tables presented include all 3,610 respondents.

Results

Descriptive Statistics

Table 1 shows descriptive statistics and mean comparisons for the four genetic subgroups (two plasticity alleles, one MAOA plasticity allele, one DAT1 plasticity allele, and no plasticity alleles). The average respondent in the sample was about 16 years old at the time of Wave II data collection. As expected, criminal offending levels at Wave II (W2) are fairly low. Importantly, there are no mean differences between these groups with respect to criminal behavior, PPCR, or low self-control. However, there are some racial/ethnic differences of note. Whites are underrepresented, whereas Blacks and Asians are overrepresented, in the two plasticity allele groups. There is clear empirical evidence that DAT1 genotype varies with ancestry and race/ethnicity (Kang, Palmatier, & Kidd, 1999), while there is circumstantial evidence that MAOA genotype may vary with race/ethnicity (Balciuniene et al., 2001; Gilad, Rosenberg, Przeworski, Lancet, & Skorecki, 2002; Sarich & Miele, 2004).

Table 2 presents the correlation matrix for the study variables, which serves as a check for gene–environment correlations (rGE). Gene–environment correlation refers to a non-random distribution of environments among different genotypes (Simons et al., 2011), which potentially confounds $G \times E$ effects (Guo, Roettger, & Cai, 2008; Guo, Tong, & Cai, 2008). Table 2 shows that there is not a significant correlation between PPCR and the genetic subtypes, indicating an absence of rGE effects in these data. In addition, an independent-samples *t* test confirmed no mean-difference in PPCR between the most and least plastic individuals in the sample. Furthermore, genotype does not significantly correlate with criminal behavior at Wave II. As expected, PPCR is significantly related to levels of self-control at Wave I and criminal behavior at Wave II. In addition and in line with expectations, self-control is significantly and positively correlated with criminal behavior. Of note, individuals with 0 plasticity alleles report slightly lower levels of self-control compared with the other genetic subtypes.

Table 1. Descriptive Statistics and Mean Comparisons by Genotype ($N = 3,610$).

Variables	Range	MAOA and DAT1 ($n = 897$)	MAOA (2R or 3R) ($n = 576$)	DAT1 (10R/10R) ($n = 1,235$)	None ($n = 902$)
		<i>M</i> (<i>SE</i>)	<i>M</i> (<i>SE</i>)	<i>M</i> (<i>SE</i>)	<i>M</i> (<i>SE</i>)
Dependent variable					
Criminal behavior W2	0.69-3.53	2.26 (.01)	2.26 (.01)	2.25 (.00)	2.26 (.01)
Independent variable					
PPCR	-1.38-5.45	-0.01 (.03)	0.05 (.04)	0.02 (.02)	-0.05 (.03)
Mediating variable					
Low self-control	25-84	46.44 (.24)	46.93 (.30)	46.51 (.20)	47.16 (.23)
Controls					
White	0/1	0.41 (.02)	0.55 (.02)	0.60 (.01)	0.66 (.02)**
Hispanic	0/1	0.15 (.01)	0.14 (.01)	0.15 (.01)	0.15 (.01)
Black	0/1	0.25 (.01)	0.22 (.02)	0.14 (.01)	0.12 (.01)**
Native American	0/1	0.03 (.01)	0.02 (.01)	0.03 (.00)	0.02 (.00)
Asian	0/1	0.14 (.01)	0.06 (.01)	0.07 (.01)	0.03 (.01)**
Other	0/1	0.01 (.00)	0.01 (.00)	0.01 (.00)	0.01 (.00)
Age W2	11-21	16.04 (.05)	16.06 (.07)	16.19 (.04)	16.12 (.05)
Parent's education	0/1	0.29 (.01)	0.25 (.02)	0.28 (.01)	0.27 (.01)
Parent receiving public assistance	0/1	0.06 (.01)	0.08 (.01)	0.08 (.01)	0.06 (.01)
Affiliation with delinquent peers	-0.97-2.45	-0.12 (.03)	-0.04 (.04)	-0.08 (.03)	-0.17 (.03)

Note. Because these statistics are weighted and adjusted for survey design, standard errors are produced rather than standard deviations. Mean one-way ANOVAs denote significant genotype comparisons. MAOA = monoamine oxidase A gene; DAT1 = dopamine transporter; W2 = Wave II; PPCR = poor parent-child relationship.

* $p < .05$. ** $p < .01$.

Multivariate Analysis

Table 3 presents OLS regression models using low self-control as the outcome of interest. Model 1 examines the effects of PPCR and genotype on levels of self-control, while controlling for various factors. This model shows that more detached parenting results in lower self-control, as predicted by self-control theory and in support of Hypothesis 1. Interestingly, compared with carriers of 0 plasticity alleles, males who carry plasticity alleles for both MAOA and DAT1 (-0.91), or just DAT1 alone (-1.05), evidence higher levels of self-control. Among the controls, older respondents report higher levels of self-control, and individuals with more delinquent affiliations report lower levels of self-control.

Model 2 in Table 3 provides a test of the cumulative plasticity hypothesis. The cumulative plasticity hypothesis suggests that those who carry plasticity alleles for both MAOA and DAT1 should evince a stronger response to their social environment than those who carry plasticity alleles for neither or only one of the two. To test this possibility, we created interactions between the genetic dummy variables and PPCR (0 plasticity alleles is the referent). As shown in Model 2, carriers of plasticity alleles for both MAOA and DAT1 (-0.81 ; $p < .05$), or just the 10R/10R combination of DAT1 (-1.03 ; $p < .05$), evidence higher levels of self-control compared with those who carry

Table 2. Correlation Matrix for the Study Variables (*N* = 3,610).

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.
1. Criminal behavior W2	X																
2. PPCR	.07**	X															
3. MAOA and DAT1	.02	-.01	X														
4. MAOA (2R or 3R)	-.01	.03	-.25**	X													
5. DAT1 (10R/10R)	-.02	.02	-.42**	-.31**	X												
6. None	.00	-.03	-.33**	-.25**	-.42**	X											
7. Low self-control	.17**	.36**	-.02	.01	-.02	.04*	X										
8. White	-.05**	.05**	-.17**	-.01	.05**	.11**	.07**	X									
9. Hispanic	.04*	-.03	-.01	-.01	.01	.01	-.02	-.47**	X								
10. Black	.02	-.08**	.11**	.05**	-.07**	-.08**	-.07**	-.52**	-.20**	X							
11. Native American	.04*	.00	.01	-.01	.01	-.02	.03	-.18**	-.07**	-.08**	X						
12. Asian	-.02	.06**	.15**	-.03	-.02	-.10**	-.02	-.33**	-.12**	-.13**	-.05**	X					
13. Other	.02	-.01	.01	-.01	-.01	.01	-.01	-.12**	-.05**	-.05**	-.02	-.03	X				
14. Age W2	.01	.19**	-.03	-.02	.03*	.00	.03	-.05**	.07**	-.04**	.00	.05**	.01	X			
15. Parent's education	-.03	.02	.03	-.03	.01	-.01	-.03	.02	-.16**	.03*	-.01	.14**	.01	-.03	X		
16. Parent receiving public assistance	.05**	.01	-.02	.01	.02	-.02	.02	-.13**	.10**	.11**	.02	-.06**	-.03	-.02	-.14**	X	
17. Affiliation with delinquent peers	.21**	.19**	-.01	.03	.02	-.04*	.27**	.05**	.02	-.06**	.02	-.05**	.02	.29**	-.06**	.04*	X
M	.84	-.012	.025	.016	.034	.025	46.72	.056	.015	.018	.003	.008	.001	.16,11	.027	.007	-.011
SE	.12	.01	.01	.01	.01	.01	.12	.01	.01	.01	.00	.00	.00	.03	.01	.00	.02

Note. W2 = Wave II; PPCR = poor parent-child relationship; MAOA = monoamine oxidase A gene; DAT1 = dopamine transporter.
p* < .05. *p* < .01.

Table 3. Low Self-Control Regressed on PPCR, Genotype, and Controls ($N = 3,610$).

Variables	Model 1	Model 2
	Coefficient (SE)	Coefficient (SE)
Environment and genetic variables		
PPCR	2.97 (.23)**	2.59 (.33)**
MAOA and DAT1	-0.91 (.39)*	-0.81 (.41)*
MAOA (2R or 3R)	-0.78 (.53)	-0.73 (.53)
DAT1 (10R/10R)	-1.05 (.36)**	-1.03 (.36)**
Dummy variable interactions		
PPCR $\times$ MAOA and DAT1		1.67 (.80)*
PPCR $\times$ MAOA (2R or 3R)		-0.01 (.46)
PPCR $\times$ DAT1 (10R/10R)		-0.08 (.45)
Controls		
Hispanic	-0.67 (.50)	-0.67 (.49)
Black	-0.68 (.36)	-0.71 (.36)
Native American	1.86 (.98)	1.74 (.98)
Asian	-0.45 (.67)	-0.47 (.66)
Other	-0.90 (.73)	-0.96 (.68)
Age W1	-0.53 (.11)**	-0.52 (.11)**
Parent's education	-0.19 (.31)	-0.19 (.31)
Parent receiving public assistance	0.60 (.56)	0.68 (.56)
Affiliation with delinquent peers	2.06 (.17)**	2.07 (.17)**
Constant	56.10 (1.86)**	55.93 (1.83)**
R^2	.21	.21

Note. Zero plasticity allele is the reference category for all genetic variables and $G \times E$ terms. Non-Hispanic White is the reference category for all race/ethnic groups. This table includes unstandardized coefficients (linearized standard errors) from OLS models. PPCR = poor parent-child relationship; MAOA = monoamine oxidase A gene; DAT1 = dopamine transporter; W1 = Wave 1; $G \times E$ = gene-environment interactions; OLS = ordinary least squares.

* $p < .05$. ** $p < .01$.

plasticity alleles (when parenting is at the mean). The coefficient for PPCR is the effect for males who carry 0 plasticity alleles, which remains positive and highly significant (2.59; $p < .01$). Turning to the interaction terms, and in line with Hypothesis 2, having plasticity alleles for both MAOA and DAT1 interacts with PPCR to significantly predict low self-control in the expected direction: The effect of PPCR on low self-control is significantly greater for the MAOA and DAT1 group than among the 0 plasticity alleles group. Plasticity alleles for only MAOA or DAT1 separately do not have a significant interaction with PPCR. This means that among these genetic subgroups the effect of PPCR on low self-control is not significantly different from the effect for individuals with 0 plasticity alleles.

We probed the interaction effect found in Model 2 of Table 3 by assessing the effect of PPCR on low self-control by the number and type of plasticity alleles an individual

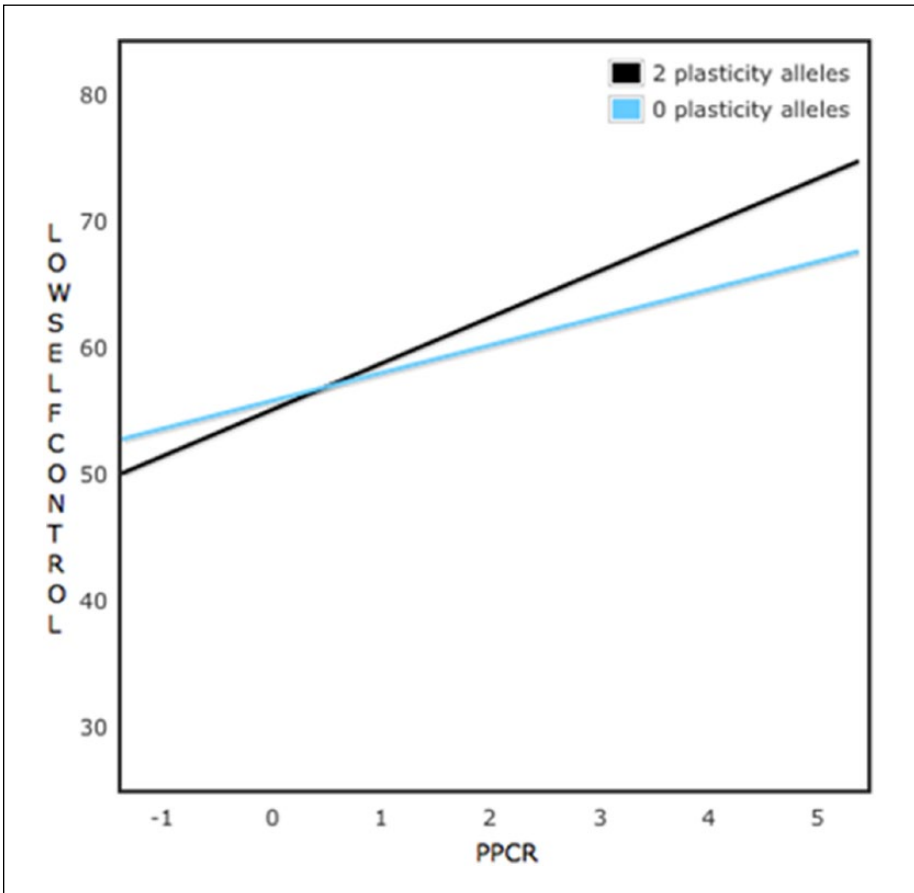


Figure 1. Interaction between number of plasticity alleles (0 vs. 2) and PPCR in the prediction of self-control for males.

Note. PPCR = poor parent-child relationship.

carries. While there is a highly significant effect of PPCR on low self-control regardless of the number and type of plasticity alleles one carries (supportive of self-control theory), the magnitude of this effect is significantly greater for carriers of the plasticity alleles for MAOA and DAT1 (4.26) relative to males who do not carry these risk alleles (2.59). Thus, the main contrast is between those who carry both of these plasticity alleles and those who carry none, and the difference between the two coefficients is statistically significant (see Table 3, Model 2). Consistent with the cumulative plasticity hypothesis, these findings suggest that individuals with plasticity alleles for both MAOA and DAT1 respond more strongly to their environment, specifically parenting, than individuals who carry none or only one of these plasticity alleles. Figure 1 presents the graphical plot of the interaction between PPCR and the number of plasticity

Table 4. Criminal Behavior W2 Regressed on PPCR, Genotype, Low Self-Control, and Controls.

Variables	Model 1	Model 2	Model 3
	Coefficient (SE)	Coefficient (SE)	Coefficient (SE)
Environment and genetic variables			
PPCR	0.01 (.00)*	0.00 (.01)	-0.01 (.01)
MAOA and DAT1	0.00 (.01)	0.00 (.01)	0.00 (.01)
MAOA (2R or 3R)	-0.02 (.01)	-0.02 (.01)	-0.01 (.01)
DAT1 (10R/10R)	-0.01 (.01)	-0.01 (.01)	-0.01 (.01)
Dummy variable interactions			
PPCR × MAOA and DAT1		0.04 (.02)*	0.03 (.02)
PPCR × MAOA (2R or 3R)		0.01 (.01)	0.01 (.01)
PPCR × DAT1 (10R/10R)		0.01 (.01)	0.01 (.01)
Mediating variable			
Low self-control			0.01 (.00)**
Controls			
Hispanic	0.01 (.01)	0.01 (.01)	0.02 (.01)
Black	0.02 (.01)	0.02 (.01)	0.02 (.01)*
Native American	0.05 (.02)*	0.04 (.02)*	0.04 (.02)
Asian	-0.01 (.02)	-0.01 (.02)	-0.01 (.01)
Other	0.02 (.02)	0.02 (.02)	0.02 (.02)
Age W2	-0.01 (.00)**	-0.01 (.00)**	-0.01 (.00)**
Parent's education	-0.01 (.01)	-0.01 (.01)	-0.01 (.01)
Parent receiving public assistance	0.00 (.01)	0.01 (.01)	0.00 (.01)
Affiliation with delinquent peers	0.04 (.01)**	0.04 (.01)**	0.04 (.01)**
Constant	2.40 (.04)**	2.39 (.04)**	2.25 (.05)**
R ²	.07	.07	.08

Note. Zero plasticity allele is the reference category for all genetic variables and G × E terms. Non-Hispanic White is the reference category for all race/ethnic groups. This table includes unstandardized coefficients (linearized standard errors) from OLS models. PPCR = poor parent-child relationship; MAOA = monoamine oxidase A gene; DAT1 = dopamine transporter; W2 = Wave II; G × E = gene-environment interactions; OLS = ordinary least squares.

* $p < .05$. ** $p < .01$.

alleles a respondent carries in the prediction of levels of self-control. As the only significant contrast found in Model 2 of Table 3 is between those carrying 0 and 2 plasticity alleles, we only graph this contrast for the sake of parsimony. We can see that while self-control worsens as the parent-child relationship worsens for individuals with 0 and 2 plasticity alleles, the slope is steeper for individuals with 2 plasticity alleles.

Table 4 presents a similar modeling strategy with criminal behavior at Wave II (W2) substituted as the outcome of interest. The baseline model (Model 1) shows that the genes of interest in this study do not have direct effects on criminal behavior. As expected and in support of Hypothesis 3, PPCR is significantly (but with a modest

effect size of .0127) and positively predictive of criminal behavior, with less warmth and more disengagement resulting in more offending. Among the controls, Native Americans report significantly more offending than do non-Hispanic Whites, and older respondents report less offending.

Model 2 in Table 4 again shows that the genetic dummy variables do not evidence main effects on criminal behavior. The coefficient for PPCR is its effect on criminal behavior at Wave II for males with 0 plasticity alleles, which is not statistically significant. As predicted by Hypothesis 4, however, having plasticity alleles for both MAOA and DAT1 interacts with PPCR to significantly predict criminal behavior. The effect of PPCR on criminal behavior is significantly stronger among males with 2 plasticity alleles than among those with 0 plasticity alleles. We probed the interaction effect between PPCR and genotype on criminal behavior that is found in Model 2 of Table 4. Most important, the only group that displays a statistically significant (but modest) effect of PPCR on criminal behavior is those who carry plasticity alleles for both MAOA and DAT1 (.0356; $p = .036$). Again, these results support the contention in the cumulative plasticity perspective that individuals with more plasticity alleles are more susceptible to environmental influence.

As a final test of expectations from self-control theory, low self-control is introduced in Model 3 as a potential mediator of the interaction between PPCR and genotype. As can be seen in Model 3, the effect of the interaction between 2 plasticity alleles and PPCR on criminal behavior is mediated by low self-control. Low self-control (.01; $p = .000$) thus mediates the PPCR-by-genotype ($G \times E$) effect on criminal behavior at Wave II, supporting Hypothesis 5 and self-control theory. Figure 2 presents the graphical plot of the interaction between PPCR and the number of plasticity alleles a respondent carries in the prediction of levels of criminal behavior at Wave 2. As the only significant contrast found in Model 2 of Table 4 is between those carrying 0 and 2 plasticity alleles, we only graph this contrast for the sake of parsimony. We can see that as the parent-child relationship worsens for individuals with 2 plasticity alleles, they evince slightly more criminal behavior.

Discussion

This article elaborates self-control theory (Gottfredson & Hirschi, 1990) via integration with a biosocial framework. Specifically, we test whether variants of the MAOA and DAT1 genotypes moderate the relationship between parenting, self-control, and criminal behavior. In addition, we test predictions concerning cumulative plasticity that focus on the importance of the number of plasticity alleles a person carries. Utilizing a sample of males drawn from the Add Health study (Udry, 2003), results reveal that males who carry the 2R or 3R alleles of MAOA and are homozygous for the 10R allele of DAT1 are more likely to have lower levels of self-control and in turn engage in more criminal behaviors when they experience a PPCR than their counterparts who do not carry either of these plasticity alleles. Probing the interaction reveals that the effect of a PPCR on criminal behavior is only statistically significant among the genetic subgroup of males who carry plasticity alleles for both MAOA and DAT1.

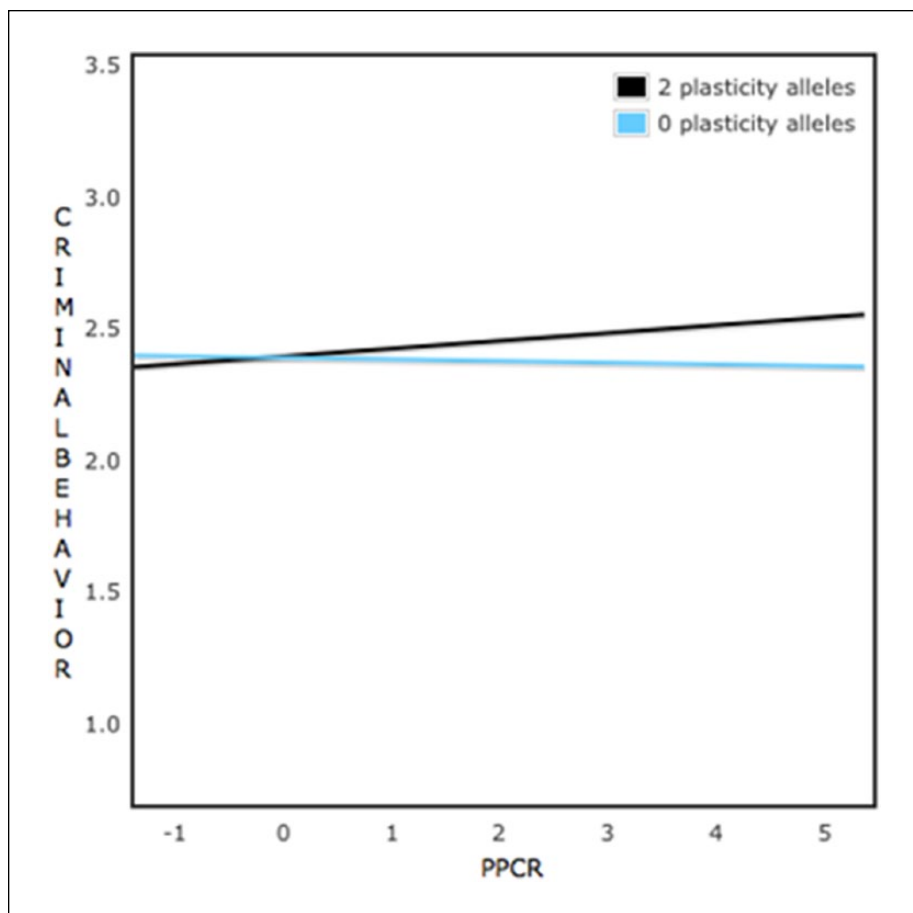


Figure 2. Interaction between number of plasticity alleles (0 vs. 2) and PPCR in the prediction of criminal behavior at Wave II for males.

Note. PPCR = poor parent-child relationship.

In support of self-control theory, this $G \times E$ effect on criminal behavior is in turn mediated by low self-control.

These results bring together previous findings that separately consider the relationship between genotype and attention deficits and antisocial behaviors. They point to the possibility that MAOA and DAT1 matter for criminal offending precisely because they are important, in combination with environmental triggers, for shaping neuropsychological attributes like levels of self-control. Given the important place accorded self-control within the criminological literature (Akers & Sellers, 2009; Pratt & Cullen, 2000), and the results of both this study and others in the $G \times E$ literature (Belsky & Beaver, 2011), future research would do well to further probe the genetic basis of

neuropsychological deficits that approximate low self-control. In addition, research should continue to identify the environmental factors, like parenting, that shape levels of self-control and test whether these effects are moderated by genotype.

While the current study makes important contributions to the $G \times E$ and criminological literatures, some limitations should be noted. First, the measure of parenting utilized in this study is not an optimal measure for testing propositions based on self-control theory. Self-control theory focuses on monitoring and discipline with respect to how self-control is shaped, while the measure of parenting in the current study focuses on the quality of the parent-child relationship. While the family literature has shown that loving parenting and watchful parenting tend to go hand in hand (Simons & Burt, 2011; Simons et al., 2007), a more comprehensive measure of parenting that includes items tapping parental involvement and monitoring would be best. In addition, our measure lacks the full range of variation necessary for a full test of the differential susceptibility versus diathesis-stress hypotheses, and thus we do not speculate as to which model the results most support.

A second measurement related issue is that the measures tapped to represent self-control in the Add Health data are not commonly used measures, and may miss some important elements of Gottfredson and Hirschi's (1990) concept of self-control, such as preferences for risk-taking. The above two issues would be best solved by testing the model put forth in this article while utilizing a different data set that includes genetic information and items that more fully tap the concepts of parenting and self-control. An additional limitation is that while this study is based on a nationally representative data set, only males are included in the models and thus the results are not generalizable to all adolescents. Future studies should assess whether $G \times E$ interaction effects are evident among females. This study is notable, however, because the sample size ($N = 3,610$) is much larger than has typically been available in $G \times E$ research.

In conclusion, this study provides evidence that a PPCR interacts with MAOA and DAT1 genotype to increase risks for both developing low self-control and engaging in criminal behavior. These findings support the utility of self-control theory as a theoretical model to explain previous findings concerning genetics, neuropsychological deficits approximating low self-control, and antisocial behaviors. This study is additionally important theoretically in showing the utility of combining traditional criminological theories with a biosocial modeling approach. This kind of theory building and modeling strategy is important because it helps further develop ideas centered on the concept that the environment and biology are always interacting to shape how we experience and react to our world. Moving forward, criminology as a discipline should focus on and consider central this kind of theory building and modeling.

Appendix

Items for Scaled Variables

Criminal behavior

In the past 12 months, how often did you . . .

1. Paint graffiti or signs on someone else's property or in a public place?
2. Drive a car without its owner's permission?
3. Steal something worth more than US\$50?
4. Go into a house or building to steal something?
5. Use or threaten to use a weapon to get something from someone?
6. Use a weapon in a fight?
7. Hurt someone badly enough to need bandages or care from a doctor or nurse?
8. You pulled a knife or gun on someone.
9. You shot or stabbed someone.

Poor parent-child relationship

1. How close do you feel to your mother?
2. How much do you think she cares about you?
3. Most of the time, your mother is warm and loving toward you.
4. You are satisfied with the way your mother and you communicate with each other.
5. Overall, you are satisfied with your relationship with your mother.

Low self-control

1. All things considered, how is your child's life going?
2. You get along well with your child.
3. You can trust your child.
4. Does your child have a bad temper?
5. You never argue with anyone.
6. When you get what you want, it is usually because you worked hard for it.
7. You never criticize other people.
8. You usually go out of your way to avoid having to deal with problems in your life.
9. Difficult problems make you very upset.
10. When making decisions, you usually go with your "gut feeling" without thinking too much about the consequences of each alternative.
11. When you have a problem to solve, one of the first things you do is get as many facts about the problem as possible.
12. When attempting to find a solution to a problem, you usually try to think of as many different ways to approach the problem as possible.
13. When making decisions, you generally use a systematic method for judging and comparing alternatives.
14. After carrying out a solution to a problem, you usually try to analyze what went right and what went wrong.
15. You like yourself just the way you are.
16. You feel like you are doing everything just about right.
17. You feel socially accepted.
18. Do you have trouble getting along with your teachers?
19. Do you have trouble paying attention in school?

20. Do you have trouble keeping your mind focused?
21. Do you have trouble getting your homework done?

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Notes

1. Limited research suggests that $G \times E$ findings concerning monoamine oxidase A gene (MAOA) may not be applicable to females (Sjöberg et al., 2007).
2. Hardy–Weinberg equilibrium assumes that individuals are diploid, and this assumption is violated in the case of MAOA among human males, who are not diploid on the X chromosome. For dopamine transporter (DAT1), the Hardy–Weinberg equilibrium test results are $\chi^2 = 1.159, p = .282$.
3. For all of the predictor variables, in all models presented, variance inflation factor (VIF) < 10 and $1/\text{VIF} > 0.1$.

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