Development and Psychopathology **29** (2017), 1235–1252

doi:10.1017/S0954579416001279

The interaction between monoamine oxidase A and punitive discipline in the development of antisocial behavior: Mediation by maladaptive social information processing

CHARDÉE A. GALÁN,[a] DANIEL EWON CHOE,[b] ERIKA E. FORBES,[a] AND DANIEL S. SHAW[a]
[a]*University of Pittsburgh; and* [b]*University of California, Davis*

Abstract

Previous studies demonstrate that boys' monoamine oxidase A (*MAOA*) genotype interacts with adverse rearing environments in early childhood, including punitive discipline, to predict later antisocial behavior. Yet the mechanisms by which *MAOA* and punitive parenting interact during childhood to amplify risk for antisocial behavior are not well understood. In the present study, hostile attributional bias and aggressive response generation during middle childhood, salient aspects of maladaptive social information processing, were tested as possible mediators of this relation in a sample of 187 low-income men followed prospectively from infancy into early adulthood. Given racial–ethnic variation in *MAOA* allele frequencies, analyses were conducted separately by race. In both African American and Caucasian men, those with the low-activity *MAOA* allele who experienced more punitive discipline at age 1.5 generated more aggressive responses to perceived threat at age 10 relative to men with the high-activity variant. In the African American subsample only, formal mediation analyses indicated a marginally significant indirect effect of maternal punitiveness on adult arrest records via aggressive response generation in middle childhood. The findings suggest that maladaptive social information processing may be an important mechanism underlying the association between *MAOA* × Parenting interactions and antisocial behavior in early adulthood. The present study extends previous work in the field by demonstrating that *MAOA* and harsh parenting assessed in early childhood interact to not only predict antisocial behavior in early adulthood, but also predict social information processing, a well-established social–cognitive correlate of antisocial behavior.

Antisocial behavior (AB) consists of a diverse constellation of aggressive, destructive, and norm-violating behaviors and is frequently persistent, resistant to treatment, and highly stable from early childhood into adolescence and adulthood (Loeber, 1982; Shaw, Gilliom, Ingoldsby, & Nagin, 2003). In addition to the serious consequences that such behavior has on society in terms of property loss and victims of violence, youth who commit antisocial acts and those who endorse violent attitudes are often significantly impaired in psychological, occupational, and social domains (Bongers, Koot, van der Ende, & Verhulst, 2004; Odgers et al., 2008). Individuals displaying high levels of AB are more likely to use illegal substances, experience depressive symptoms, and fail to achieve occupational stability (Fergusson, Horwood, & Ridder, 2005; Rose, Dick, Viken, Pulkkinen, & Kaprio, 2004). Moreover, research has shown that the most persistent 5%–6% of offenders are responsible for more than half of crimes committed (Aguilar, Sroufe, Egeland, & Carlson, 2000; Loeber, 1982). The tremendous negative impact of AB to the individual and society has provided the impetus for identifying biological and environmental risk factors, particularly among young men who outnumber females in frequency and seriousness of AB (American Psychiatric Association, 2013; Kessler et al., 1994).

Spanning family, peer, school, and neighborhood domains, the environmental risk factors for AB are numerous and to some degree dependent on children's developmental status. For instance, during early childhood when children are more psychologically and physically dependent on parents, harsh and inconsistent parenting (Edens, Skopp, & Cahill, 2008; Gershoff, 2002; Odgers et al., 2008) as well as factors that compromise parenting (e.g., parental psychopathology, low satisfaction with social support; Shaw, Bell, & Gilliom, 2000; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990) have been linked to children's emerging AB. As children move into the school-age period and adolescence, school quality (Thomas, Bierman, Thompson, Powers, & Conduct Problems Prevention Research Group, 2008), peer relationships (Trentacosta & Shaw, 2009), and neighborhood factors (Leventhal & Brooks-Gunn, 2000) play an increasingly critical role in the onset and maintenance of AB. Although the bulk of longitudinal research on AB has been concerned with school-age children, adolescents, and adults, studies demonstrating the greater malleability of child behavior and family relationships

This research was supported by National Institute on Drug Abuse Grant K05 DA025630 and NIMH Grant R01 MH050907 (to D.S.S.) and National Institute on Drug Abuse Grant R01 DA02622 (to D.S.S. and E.E.F.), all from the National Institute on Drug Abuse. This work was also supported by a National Science Foundation Graduate Research Fellowship (to C.A.G.). We thank the families for participation in the study and the research staff for their help with data collection and management.

Address correspondence and reprint requests to: Chardée A. Galán, Department of Psychology, University of Pittsburgh, 210 South Bouquet Street, 4425 Sennott Square, Pittsburgh, PA 15260-0001; E-mail: cag127@pitt.edu.

during early versus later childhood (Reid, Webster-Stratton, & Baydar, 2004) have motivated attempts to trace the environmental precursors of AB beginning in infancy and toddlerhood (Egeland, Kalkoske, Gottesman, & Erikson, 1990; Shaw, Hyde, & Brennan, 2012). As noted above, particularly relevant to the emergence of AB in early childhood are caregiving practices that model, reinforce, or elicit child oppositional and aggressive behavior, including overcontrolled, rejecting, unresponsive, and uninvolved caregiving, all of which have received extensive support as risk factors for the emergence of conduct problems during early childhood and the subsequent development of more serious AB in adolescence and adulthood (Burnette, Oshri, Lax, Richards, & Ragbeer, 2012; Campbell, Shaw, & Gilliom, 2000; Shaw et al., 1998, 2012). Exposure to punitive parenting in toddlerhood, including the use of spanking, yelling, and coercion, is one of the strongest and most replicable caregiving predictors of AB (e.g., Odgers et al., 2008; Shaw, Criss, Schonberg, & Beck, 2004). Punitive parenting may provide a model of aggressive and hostile behavior, leading to increasing levels of AB across childhood, adolescence, and adulthood (Shaw et al., 2000; Shaw & Gross, 2008). For example, using the current sample of low-income boys, observational measures of harsh parenting during early childhood discriminated patterns of AB and predicted juvenile court involvement between 15 and 18 years old (Shaw et al., 2012).

Evidence from quantitative and molecular approaches indicates that in addition to environmental influences, 50%–60% of the variance in AB can be attributed to genetic factors (Ferguson, 2010; Gunter, Vaughn, & Philibert, 2010; Rhee & Waldman, 2002). However, it should be noted that estimates from behavioral genetics studies do not disentangle pure genetic effects from the effects of gene–environment interactions and correlations. Nonetheless, meta-analyses of twin and adoption studies suggesting moderate to high heritability of AB (Rhee & Waldman, 2002; Waldman & Rhee, 2006) have kindled the search for specific candidate genes that may account for variation in this phenotype (e.g., Burt & Mikolajewski, 2008). Several lines of converging evidence suggest that genes underlying various aspects of the dopaminergic and serotonergic systems play a role in the etiology and pathophysiology of AB, and one of the most highly studied candidate genes is the monoamine oxidase A (*MAOA*) gene. This gene is located on the X chromosome (Xp11.23–11.4) and encodes for the *MAOA* enzyme, which selectively degrades serotonin, dopamine, and norepinephrine following reuptake from the synaptic cleft (Levy, 1989). A common functional polymorphism in the *MAOA* gene's transcriptional control region is the 30-base pair *MAOA* upstream variable number of tandem repeats (*MAOA-u* VNTR), which alters the transcriptional efficiency of the *MAOA* gene, resulting in high- or low-activity *MAOA* (Sabol, Hu, & Hamer, 1998). Relative to 3.5- and 4-repeat variants (H-*MAOA* genotype), the presence of 2-, 3-, or 5-repeat alleles is associated with lower *MAOA* expression and activity (L-*MAOA* genotype; Sabol et al., 1998). Several studies have linked the L-*MAOA* genotype with a range of antisocial behaviors, including aggression and violent behavior, particularly for men (Reti et al., 2011; Widom & Brzustowicz, 2006), gang affiliation, and use of a weapon (Beaver, DeLisi, Vaughn, & Barnes, 2010). While such findings indicate a potential main effect of *MAOA* genotype on AB, the picture is considerably complicated by a large number of failed replications, suggesting that genetic effects may be obscured when environmental context is not also considered (Guo, Roettger, & Shih, 2007; Reif et al. 2007).

Despite exposure to harsh and punitive parenting in toddlerhood, and even in the presence of a genotype that might otherwise be expected to confer vulnerability for AB, many youth are well adjusted later in life (Moffitt, 2005). In accordance with the concepts of multifinality and equifinality, multiple pathways to resilient and maladaptive functioning are possible, and it is increasingly recognized that models of gene–environment interplay may explain the development of AB better than either factor alone. The two most examined forms of gene–environment interplay include gene–environment correlation (*r*GE), which occurs when individuals select or create environmental experiences that are guided by heritable dispositions, and Gene × Environment interaction (G × E), which is characterized by genetic differences in sensitivity to particular environmental effects (Kendler & Eaves, 1986). In a widely cited report of a putative G × E interaction, Caspi et al. (2002) demonstrated that while there were no main effects of *MAOA* on AB, genetic variation moderated effects of childhood maltreatment on subsequent development of AB in adolescence and adulthood. Maltreated boys with the low-activity *MAOA* genetic variant were more likely than those with the high-activity *MAOA* allele to be disposed toward violent behavior, have violent crime convictions, and have diagnoses of conduct disorder as adolescents and antisocial personality disorder as adults. This interaction was corroborated in a majority of initial replication efforts, and meta-analyses support the robustness of this effect (Byrd & Manuck, 2013; Kim-Cohen et al., 2006; Taylor & Kim-Cohen, 2007). However, despite underscoring the complex nature of antisocial behavioral development that encompasses both genetic and environmental components, existing G × E literature on AB is limited by a predominant focus on Caucasian samples (Caspi et al., 2002, Frazzetto et al., 2007; Kim-Cohen et al., 2006), with little research dedicated to the exploration of G × E interactions in racial–ethnic minorities (Beaver, Nedelec, Wilde, Lippoff, & Jackson, 2011; Yungyu et al., 2004). The lack of research on African American (AA) individuals, in particular, is a salient limitation based on the disproportionately high levels of AB and exposure to harsh parenting in this population (Dodge & Pettit, 2003; Gershoff, 2002). In addition, many studies in the *MAOA* literature focus on exposure to extreme environmental circumstances, such as child abuse, but many family environments are characterized by more normative expressions of anger and use of punitive discipline, which often precede child abuse. As a notable exception, Choe, Shaw, Hyde, and Forbes (2014), using the present sample of low-income, ethnically

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

diverse men (44% AA), demonstrated that harsh parenting in early childhood predicted antisocial outcomes in adolescence and adulthood only for men with the L-*MAOA* genotype. Although in need of replication, these findings suggest that parenting practices within the normative range, not merely extreme environmental adversity such as abuse, are critically relevant to the development of AB for both Caucasian and AA men who are genetically vulnerable.

Although Caspi et al.'s (2002) seminal study has generated a literature that includes over 80 replication or extension attempts, the mechanisms by which early childhood adversity and *MAOA* interact to amplify risk for AB have received little empirical attention. Dodge (2009) argues that "the question of mechanisms in gene–environment interaction effects is one of the most important questions to be answered in psychology in the next two decades" (p. 1). Dodge was among the first to postulate potential neural, molecular genetic, and social–cognitive mechanisms of G × E interaction, emphasizing social information processing (SIP) patterns as a potential process through which gene–environment effects may operate on AB. Arguing that the genetically influenced neurochemical actions of the *MAOA* enzyme have cognitive–emotional correlates, Dodge (2009) hypothesizes that the low-activity *MAOA* allele may be associated with a pattern of autonomic arousal and defensive information processing that is characterized by hypervigilance to hostile cues, hostile attributional biases, and selection of self-defensive, aggressive goals. These hypotheses have yet to be empirically tested; hence, examination of SIP as a potential mediator of G × E interactions in relation to AB is warranted.

Several theorists posit that children's perceptions and interpretations of their social worlds in part mediate relations between their environments and social adjustment (Crittenden & Ainsworth, 1989; Cummings & Davies, 1994; Dodge, Pettit, Bates, & Valente, 1995). Specifically, SIP theory (Crick & Dodge, 1994; Dodge & Pettit, 2003) is broadly concerned with the cognitive processes that are deployed to generate a behavioral response during a social interaction, including selective attention to social cues, attributions, and inferences about those cues, the generation of goals, and accessing behavioral scripts from memory. Two critical aspects of maladaptive SIP include tendencies to attribute hostile intent to others and to generate aggressive responses when faced with ambiguous social situations with negative outcomes. Often referred to as *hostile attributional bias* (HAB) and *aggressive response generation* (ARG), respectively, these maladaptive patterns of SIP have been reported among community and clinical populations, including rejected and aggressive elementary school boys (Guerra & Slaby, 1989; Lochman, 1987) and violent incarcerated offenders (Slaby & Guerra, 1988). Although HAB can be identified in children as young as 4 years of age, research suggests that these early attributional biases only persist in a percentage of children and do not solidify into a stable processing pattern until the end of middle childhood (Dodge et al., 1995). In addition, a review by Orobio de Castro, Veerman, Koops, Bosch, and Monshouwer (2002) examining effects of HAB on aggressive behavior reported that stronger effect sizes were identified for 8- to 12-year-olds than for younger children. Thus, the present study examines SIP patterns in middle childhood because of children's capacity to assess attributional biases during this developmental period and the high predictive validity of SIP patterns in relation to later AB.

Individual differences in SIP response patterns are thought to mediate the effects of social threat on aggressive behavior (Crick & Dodge, 1994) and to arise from genetic polymorphisms and early adverse experiences (Dodge, 2009; Eisenberger, Way, Taylor, Welch, & Lieberman, 2007). Specifically, exposure to harsh parenting and frequent expressions of anger may provide a model of aggressive and hostile behavior, facilitating the internalization of hostile schemas and frequent access to aggressive responses in both threatening and benign situations (Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak, Cicchetti, Klorman, & Brumaghim, 1997). These schemas may then be used to guide interpretation and response to future social conflict, facilitating the development and growth of AB (Dodge et al., 1995). Nonetheless, while environmental influences on SIP have been empirically demonstrated, genetic effects have garnered limited empirical attention and interactions between environmental and genetic influences remain to be examined.

Despite compelling theory that maladaptive SIP in part accounts for relations between adverse family contexts and the emergence of AB, the initial empirical evidence has been inconsistent. While studies focused on child maltreatment provide support for the mediational role of child SIP (Dodge, Price, Bachorowski, & Newman, 1990; Schwartz & Proctor, 2000; Weiss, Dodge, Bates, & Pettit, 1992), studies focused on parental psychopathology and family instability have not corroborated such findings (Downey & Walker, 1989; Schultz, Izard, & Ackerman, 2000). Moreover, to the best of our knowledge, no study has examined whether maladaptive SIP mediates genotype-dependent environmental influences on risk for AB. Thus, the present study extends the findings of Choe et al. (2014), who using the present sample, demonstrated that punitive parenting in early childhood interacted with *MAOA* genotype to predict adolescent and adult antisocial outcomes. Using the same longitudinal sample of low-income, ethnically diverse boys followed prospectively from ages 1.5 to 22, the present study probes whether individual differences in SIP patterns in middle childhood constitute a potential mediating mechanism connecting interactions between *MAOA* and punitive parenting in toddlerhood with AB in late adolescence, when violent attitudes may signify problems with adolescent-onset AB, and in early adulthood, which is prognostic of lifelong criminal behavior, the latter based on endorsement of AB and violent attitudes. Specifically, we hypothesize that the interactive effects of *MAOA* and early punitive parenting on later AB and correlates of AB will be mediated by SIP during middle childhood. On a more exploratory level, the present study also tests whether *MAOA* and SIP interact to predict early adulthood outcomes,

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

but we formed no a priori hypotheses, as the present study represents the first of its kind to examine this genotype–phenotype interaction (G × P).

Method

Participants

Participants were drawn from the Pitt Mother and Child Project, a prospective longitudinal study of child vulnerability and resilience in low-income, high-risk youth (Shaw et al., 2003). Beginning in 1991, 310 infant boys and their primary caregivers were recruited from Women, Infants, and Children Program Nutritional Supplement Clinics in Allegheny County, Pennsylvania, when the boys were between 6 and 17 months old. Participation was limited to boys because of the project's original focus on the developmental precursors of AB, which occurs at higher rates in men (American Psychiatric Association, 2013; Kessler et al., 1994). At the time of recruitment, the boys were between 6 and 17 months, and 53% of them were Caucasian, 36% were AA, 5% were biracial, and 6% were of other races (e.g., Asian American or Hispanic). At the study's outset, the mean per capita income was $241 per month ($2,892 per year), and the mean Hollingshead socioeconomic status (SES) score was 24.5, indicating a working-class sample (Hollingshead, 1975). Mothers ranged in age from 17 to 43 years ($M = 28.20$ years), and in reporting their relationship status, 63% were married or cohabitating, 28% had always been single, 8% were divorced or separated, and 1% were other (e.g., widowed). Fifty-nine percent of the mothers had 12 years of education or less. Thus, a large proportion of the boys in this study were considered to be at elevated risk for antisocial outcomes because of their low SES and sex.

Retention rates have been consistently high throughout the two decades of data collection. Of the original 310 families, some data are available for 306 families (98.7%) at the age 1.5 assessment, 275 families (89%) at the age 10 assessment, 251 families (81%) at the age 17 assessment, and 256 families (83%) at the age 20 and 22 assessments. The total sample size is 187 young men with both race–ethnicity and *MAOA* genotype (just 3.5 and 4.5 repeats) data.

Procedure

For this study, 2- to 3-hr assessments were conducted in families' homes and/or laboratory settings with mothers and their participating child at ages 1.5, 2, 3.5, 5, 5.5, 6, 8, 10, 11, 12, 15, 17, 20, 21, 22, and 23 years old. The assessments providing data for the present study occurred at ages 1.5, 10, 17, 20, and 22 years. Mothers were videotaped interacting with their child in age-appropriate tasks at 1.5 years, and when sufficiently old, boys completed questionnaires regarding their behavior and attitudes. All participants provided consent and were compensated for their time after each assessment. All procedures received Institutional Review Board approval at the University of Pittsburgh.

DNA extraction and genotyping

Saliva samples were collected from participants using Oragene™ DNA self-collection kits. DNA was isolated from the samples according to the manufacturer's instructions (DNA Genotek, 2006), and *MAOA-u* VNTR genotyping was performed using polymerase chain reaction amplification and gel electrophoresis. Consistent with previous translations, this yielded alleles with two-, three-, four-, and five-repeat lengths, which were then grouped according to their level of transcriptional activity (Sabol et al., 1998). Alleles with four-repeat length were categorized as the low-activity form of *MAOA* (i.e., L-*MAOA*), while those with two-, three-, and five-repeat lengths constituted the high-activity form of *MAOA* (H-*MAOA*). However, men with the two- or five-repeat variants were excluded because the activity levels of these alleles remain unclear (Kim-Cohen et al., 2006). Thus, analyses compared men hemizygous for the three-repeat allele (i.e., L-*MAOA*) to those hemizygous for the four-repeat allele (i.e., H-*MAOA*). The results remained the same after adding participants with the extreme lengths two and five to the L-*MAOA* group. Hardy–Weinberg equilibrium could not be calculated due to hemizygosity in males.

Analyses were conducted separately by race because of evidence suggesting racial–ethnic variation in *MAOA* allele frequencies (e.g., Sabol et al., 1998). We relied on caregiver report rather than on genetic ancestry-informative markers to determine child race-ethnicity because researchers using ancestry-informative markers found that genetically distinct clusters corresponded well with self-reported race, namely, Caucasian and AA (Enoch, Shen, Xu, Hodgkinson, & Goldman, 2006; Hodgkinson et al., 2008). Because of a limited sample size, separate analyses for racial groups precluded computing three-way statistical interactions among race, *MAOA* genotype, and maternal punitiveness.

Measures

Punitive discipline. At 1.5 years old, boys engaged in two structured laboratory tasks with their mothers, which each lasted for 5 min and were designed to elicit varying levels of stress and harsh parenting behaviors. Following a 15-min free-play task in which a set of attractive toys were introduced to the child while mothers completed questionnaires with the lead examiner, a 5-min clean-up task was introduced during which the mother was instructed to direct her child to place all of the free-play toys in a laundry basket. Mothers were informed that they could offer help to their child as necessary aside from actually placing the toys in the basket. The second videotaped interaction involved the mother and child completing three teaching tasks, which consisted of three puzzles that were purposefully geared to slightly older children to elicit individual differences in parenting behavior.

Using the Early Parenting Coding System (Winslow & Shaw, 1995), trained observers later coded the videos for parenting behaviors that have been shown by previous researchers to be related to children's adjustment (e.g., parental

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

intrusiveness). The Early Parenting Coding System consists of nine categories of parenting strategies coded molecularly and six global ratings. For the purposes of the present study, the global ratings of maternal punitiveness from the clean-up and puzzle teaching tasks will be utilized, which coders generated after viewing the entire video clip. Global ratings of punitiveness measured the degree to which the mother was too strict, demanding, or harsh considering the child's behavior during the task. Global ratings were made on a 4-point scale, with higher scores indicating higher levels of strictness in parenting methods. Trained coders attained excellent reliability ($\kappa = 0.94$).

Maladaptive SIP. When boys were 10 years old, a vignette procedure was used to assess HAB and ARG (Dodge & Somberg, 1987). Participants were orally presented with eight social vignettes and accompanying pictures and asked to imagine that they were the "target child" in the story. In each vignette, the behavior of another boy leads to a negative outcome for the target child (e.g., being bumped or exclusion from an activity). The other child's motives are intentionally ambiguous, and after viewing each vignette, participants were asked to attribute intent to the "other boy" (did the other boy hurt the target child on purpose?) and to indicate how they would respond in the situation (e.g., tell a teacher or yell at the boy). Participants' attributions of intent were scored as hostile and assigned a score of 1 if they stated that the "other boy" intentionally caused the negative outcome. All other attributions were coded as nonhostile and assigned a score of zero. The final HAB score used for the present analyses reflected the number of vignettes to which the participants responded with a hostile attribution. Internal consistency was found to be satisfactory in the current sample ($\alpha = 0.65$). Hypothetical responses involving acts or threats of physical or verbal retaliation were coded as 1s, while responses that were verbally engaging or ambiguous in their adaptive value (e.g., doing nothing or making commands) were coded as zeros. Responses were summed to create a scale of ARG ($\alpha = 0.76$). Although HAB and ARG are often aggregated into a single index of "maladaptive SIP" ($r = .29$ in present study), the emerging literature suggests different neural substrates for each (Choe, Shaw, & Forbes, 2015). Thus, HAB and ARG were examined as mediating variables in separate sets of models.

AB and age-related correlates. Three aspects of AB were examined: violent attitudes, official arrests, and self-reported engagement in AB. In an effort to better reflect the multifaceted nature of AB, we retained these measures as separate observed variables rather than combining them into a single latent variable (Loeber & Stouthamer-Loeber, 1998). At age 17, men's attitudes toward reactive violence (i.e., violence in response to actual or perceived threat) and culture of violence (i.e., general view of violence as an acceptable and valued activity) were assessed using the Attitudes Towards Violence Scale (Funk, Elliott, Urman, Flores, & Mock, 1999). For each of the 15 items, men rated how much they agreed with the statement on a 5-point scale from 1 (*strongly disagree*) to 5 (*strongly agree*). Sample items included "It's okay to do whatever it takes to protect myself" and "People who use violence get respect." Items were summed to create a total violent attitudes score ($\alpha = 0.83$).

Official court records of arrests were obtained from local county offices to assess involvement with the legal system in early adulthood. The number of arrests in Pennsylvania was summed, and if court records could not be obtained for a participant, these data were considered missing.

At ages 20 and 22, men rated the frequency of AB during the past year using the 62-item Self-Report of Delinquency Questionnaire (Elliott, Huizinga, & Ageton, 1985). Using a 3-point scale (1 = *never*, 2 = *once/twice*, 3 = *more often*), men rated the extent to which they engaged in aggressive and delinquent behavior (e.g., stealing or assault), alcohol and drug use, and related offenses. Items were summed at each age to create an index of participants' delinquency. As self-reports of AB at ages 20 ($\alpha = 0.92$) and 22 ($\alpha = 0.93$) were highly correlated with one another ($r = .63$), the two scores were averaged together into a composite scale of self-reported AB in early adulthood.

Data analysis plan

We examined attrition, missing data, descriptive statistics, and correlations in SPSS. We then examined *MAOA* allele frequencies and differences in study variables by *MAOA* genotype and race. We used Mplus 7.2 to test for statistical interactions in multiple-group mediation models in which we regressed men's violent attitudes, official arrests, or AB on SIP problems in middle childhood (either HAB or ARG) and maternal punitiveness in toddlerhood, and regressed SIP problems on maternal punitiveness. As shown in our analytic model in Figure 1, each multiple-group mediation model tests three path coefficients representing the interaction of *MAOA* and a predictor or mediator variable: Path A tests *MAOA* genotype differences in the effect of maternal punitiveness on SIP problems in middle childhood (G × E interaction); Path B tests *MAOA* genotype differences in the effect of SIP problems in middle childhood on violent attitudes in late adolescence, official arrests, or AB in early adulthood (G × P interaction); Path C tests *MAOA* genotype differences in the effect of maternal punitiveness on violent attitudes, official arrests, or AB (G × E interaction). To reiterate, multiple-group mediation models were estimated separately for AA men and Caucasian men.

Multiple-group models with structural equation modeling software offers advantages over ordinary least squares regression for testing statistical interactions by simultaneously estimating all associations among variables and allowing use of the missing data estimator, maximum likelihood with robust standard errors, which is robust to nonnormally distributed data (see Descriptive Statistics section). Multiple-group models test for statistical interactions by comparing path coeffi-

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

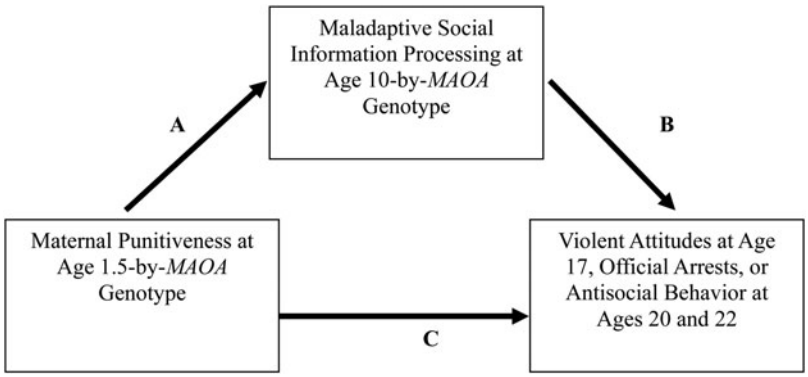

Figure 1. Analytic multiple-group mediation model regresses violent attitudes in late adolescence and official arrests or antisocial behavior in early adulthood on maternal punitiveness in toddlerhood and maladaptive social information processing (SIP) in middle childhood (i.e., hostile attributional bias or aggressive response generation), and regresses maladaptive SIP on maternal punitiveness. Multiple-group models can estimate identical or separate path coefficients for genotype groups with the latter indicating statistical interactions when path coefficients significantly differ between groups and model fit improves with separate coefficients. Group differences in path coefficients reflect statistical interactions between monoamine oxidase A (*MAOA*) and predictor variables (i.e., maternal punitiveness; Paths A and C) or mediating variables (i.e., maladaptive SIP; Path B). All models were estimated separately for African American men and Caucasian men.

cients across two or more groups as opposed to testing main and interactive effects in ordinary least squares regression and probing significant interaction terms post hoc. We tested for mediation in our multiple-group models with bootstrapping, an iterative process of random sampling that estimates significance levels of indirect effects in a more conservative and robust manner than other formal tests of mediation (Hayes, 2009). This approach to testing for mediation does not require a significant direct effect of the independent variable on the dependent variable. According to Edwards and Lambert (2007), a *subgroup approach* to combining moderation and mediation (i.e., sample split into subgroups based on the moderator variable and mediation analyzed within each subgroup) is recommended in structural equation modeling but has two main drawbacks relevant to our study: lower statistical power because analyses are conducted within subgroups, and an inability to test differences in mediation across levels of a moderator variable. Despite these limitations, our moderator variable, *MAOA* genotype, yields subgroups for statistical comparison.

We followed the standard practice of fixing and freeing cross-group equality constraints on path coefficients to determine which estimates differed between L-*MAOA* and H-*MAOA* groups. We used chi-square difference ($\Delta\chi^2$) tests for statistical comparison of nested models with and without equality constraints. Significant $\Delta\chi^2$ values indicated improvements in overall model fit when estimating separate path coefficients for each *MAOA* group (Satorra & Bentler, 2001). The results include model chi-square (χ^2), comparative fit index, root mean square error of approximation, and standardized root mean square residual. According to Kline (2005), a standardized root mean square residual of <0.10 is favorable, a root mean square error of approximation of ≤ 0.05 indicates a close fit, a comparative fit index of >0.95 reflects a good fit, and nonsignificant, low χ^2 values are desired. Within the text and tables, we report effect sizes as standardized βs and the amount of variance in mediator and dependent variables explained by the models as R^2 values. The p values presented with βs throughout are from their unstandardized estimates (b) because unstandardized p values better account for standard errors (shown in the tables) and are less biased than their standardized versions.

Results

Attrition and missing data analyses

Of the original 310 men who were recruited as toddlers to the larger study from which these data were derived, 187 men were included in final analyses. The remaining 123 (39.7%) men were excluded because of attrition and missing data on study variables (e.g., usable DNA sample for which $n = 187$). Comparison of these groups on study variables indicated they only differed on men's ARG at age 10, t (229) = 2.85, $p = .005$, 95% confidence interval [0.21, 1.15]. Men who were not part of the present report (e.g., because of attrition, refusal to provide DNA, or an unusable DNA sample) scored higher on ARG ($M = 2.34$, $SD = 1.79$) than men with complete data for the current study ($M = 1.66$, $SD = 1.69$).

Descriptive statistics and bivariate analyses

Table 1 provides separate descriptive statistics and correlations of study variables for Caucasian and AA men. Skewness and kurtosis values for maternal punitiveness indicated nonnormally distributed data, which warranted our use of maximum likelihood with robust standard errors to estimate missing data in multiple-group models. AA men's *MAOA* genotype was unrelated to study variables, whereas Caucasian men's *MAOA* genotype was correlated with both HAB and ARG. Specifically, Caucasian men carrying low-activity *MAOA* alleles had higher levels of SIP problems in middle childhood than Caucasian men carrying high-activity *MAOA* alleles.

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

Table 1. *Descriptive statistics and correlations among study variables for Caucasian and African American men*

Variables	1	2	3	4	5	6	7	*M* (*SD*)
1. Age 1.5 maternal punitiveness	—	.26†	.47***	.25*	.12	.31**	−.17	1.15 (0.38)
2. Age 10 hostile attributional bias	−.02	—	.30*	−.24†	.17	−.04	.07	5.35 (1.81)
3. Age 10 aggressive response generation	.11	.24*	—	.03	.23†	.33*	−.05	2.05 (1.72)
4. Age 17 violent attitudes	.17†	.20†	.22*	—	−.06	.50***	.09	35.60 (10.84)
5. Official arrests	.14	.26*	.12	.29**	—	.16	.00	1.05 (1.90)
6. Age 20 and 22 antisocial behavior	.10	.24*	.05	.41**	.34***	—	−.21†	9.03 (8.23)
7. *MAOA* genotype[a]	.06	−.29**	−.22*	−.06	−.13	−.09	—	1.54 (0.50)
M	1.12	4.33	1.35	33.79	0.38	10.57	1.69	
SD	0.33	2.07	1.57	8.28	0.99	7.67	0.46	

Note: MAOA, Monoamine oxidase A. For values below the diagonal, Caucasian men $n = 82–104$; for values above the diagonal, African American men $n = 52–76$.
[a]Low-activity three-repeat carriers coded as 1; high-activity four-repeat carriers coded as 2.
†$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

MAOA allele frequencies by racial group

We found similar *MAOA* allele frequencies among Caucasian and AA men as previous reports (Choe et al., 2014; Reti et al., 2011; Sabol et al., 1998). Among AA men ($n = 83$), 49% carried four-repeat *MAOA* alleles (i.e., H-*MAOA*), approximately 42% carried three-repeat *MAOA* alleles (i.e., L-*MAOA*), 6% carried two-repeat alleles, and about 2% carried five-repeat alleles. Among Caucasian men ($n = 106$), 68% carried four-repeat *MAOA* alleles, 30% carried three-repeat *MAOA* alleles, 2% carried five-repeat *MAOA* alleles, and zero men carried two-repeat alleles. L-*MAOA* and H-*MAOA* men did not differ at age 1.5 on mothers' education level, occupational status, or family income, even when examined within each racial group.

Racial group differences

We found similar race differences for socioeconomic indicators as reported in Choe et al. (2014). Caucasian families ($M = \$1{,}227.06$, $SD = \$703.34$) reported higher monthly incomes than AA families ($M = \$841.29$, $SD = \$550.70$) when men were 1.5 years old, $t\ (186) = 4.09$, $p < .001$, 95% confidence interval [\$199.69, \$571.84]. As shown in Appendix A, when compared with Caucasian men, AA men reported more aggressive responses at age 10 ($p = .013$), reported more hostile attributions at age 10 ($p = .003$), and had more arrests in their official criminal records ($p = .002$). These results are consistent with our previous finding that AA boys were more likely to be arrested as juveniles than Caucasian boys (Choe et al., 2014). There were no race differences in maternal punitiveness or self-reported violent attitudes and AB.

MAOA genotype differences in study variables by racial group

As shown in Appendix B, there were no *MAOA* genotype differences in study variables for AA men. In contrast, there were several *MAOA* genotype differences among Caucasian men for SIP problems at age 10. L-*MAOA* Caucasian men made more hostile attributions than H-*MAOA* Caucasian men ($p = .006$). L-*MAOA* Caucasian men also reported more aggressive responses than H-*MAOA* Caucasian men ($p = .043$). These *MAOA* genotype differences among Caucasian men are consistent with correlations in Table 1 that indicate a genotype–phenotype correlation between Caucasian men's *MAOA* genotype and maladaptive SIP in middle childhood.

Overall evidence of gene–environment and genotype–phenotype interactions

Table 2 summarizes results of 12 multiple-group mediation models testing interactions between men's *MAOA* genotype and maternal punitiveness or SIP problems in relation to violent attitudes in late adolescence, official arrests, or AB in early adulthood (i.e., separate analyses for AA and Caucasian men testing either HAB or ARG as the mediating variable and one of three antisocial outcomes in separate models [$2 \times 2 \times 3 = 12$]). There was overlap in interactions tested across models, so redundant tests were not counted in the overall number of comparisons. In five unique tests of potential G × E interactions per racial group (i.e., HAB, ARG, and the three antisocial outcomes regressed on the interaction of maternal punitiveness and *MAOA* [$1 + 1 + 3 = 5$]), we found two significant G × E interactions for AA men and three significant and unique G × E interactions for Caucasian men, amounting to 40% and 60% success rates, respectively. In six unique exploratory tests of G × P interactions between *MAOA* genotype and SIP problems per racial group (i.e., the three antisocial outcomes regressed on interactions between *MAOA* and HAB or ARG [$3 \times 2 = 6$]), we found one G × P interaction each for AA men and Caucasian men, amounting to 16.7% success rates per group. To correct for multiple comparisons in model testing, we employed a post hoc false discovery rate controlling procedure that accounts for the expected proportion of errors among rejected hypotheses (Benjamini & Hochberg, 1995). We applied this correction to significance

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

Table 2. *Summary of multiple-group mediation models testing gene–environment (G × E) interactions and gene–phenotype (G × P) interactions between monoamine oxidase A and maternal punitiveness (MP), hostile attributional bias (HAB), or aggressive response generation (ARG) in relation to young men's violent attitudes, official arrests, and antisocial behavior (AB)*

	Outcome Variable				
Racial Group and Predictor Variable	Middle Childhood Maladaptive Social Information Processing		Late Adolescence Antisocial Outcome	Early Adulthood Antisocial Outcomes	
	Age 10 HAB	Age 10 ARG	Age 17 Violent Attitudes	Official Arrests	Age 20 and 22 AB
African American					
Age 1.5 MP	#1–3 Main effect	—	#1 Main effect	#2 Null	#3 Main effect
Age 10 HAB	—	—	#1 Main effect	#2 Null	#3 Null
Age 1.5 MP	—	#4–6 G × E	#4 G × E	#5 Null	#6 Null
Age 10 ARG	—	—	#4 G × P	#5 Main effect	#6 Null
Caucasian					
Age 1.5 MP	#7–9 Null	—	#7 G × E	#8 Null	#9 G × E
Age 10 HAB	—	—	#7 Null	#8 G × P	#9 Main effect
Age 1.5 MP	—	#10–12 G × E	#10 G × E	#11 Null	#12 G × E
Age 10 ARG	—	—	#10 Null	#11 Null	#12 Null

Note: Hash marks (#) with numbers indicate specific multiple-group mediation models demonstrating the effect noted in the cell. Shaded cells are null or significant effects replicated in each racial group of young men.

levels of statistical values for AA and Caucasian men's multiple-group comparisons (i.e., testing *MAOA* group differences) and model coefficients (testing whether estimates differ from zero). All evidence of statistical interactions remained significant after applying the false discovery rate correction.

Main effects and statistical interactions with HAB

Table 3 presents results of six multiple-group mediation models testing interactions between mothers' punitiveness in toddlerhood, *MAOA* genotype, and men's HAB in middle childhood in relation to violent attitudes in late adolescence, official arrests, and AB in early adulthood. We found no evidence of statistical interactions for AA men in these models; only main effects. AA mothers' punitive behavior toward their sons during toddlerhood, regardless of *MAOA* genotype, positively predicted HAB in middle childhood (see Table 3 #1–#3, first and fourth columns of statistical values), violent attitudes in adolescence (see #1, second and fifth columns of statistical values), and self-reported AB in early adulthood (see #3, second and fifth columns of statistical values). It was unexpected that AA men's HAB negatively predicted violent attitudes at age 17 (see Table 3 #1, third and sixth columns of statistical values) but was not associated with arrests or AB in early adulthood (see #2–#3, third and sixth columns of statistical values). In sum, regardless of *MAOA* genotype, maternal punitiveness in toddlerhood positively predicted AA men's HAB, violent attitudes, and AB, but their HAB only negatively predicted violent attitudes.

In addition, as can be seen in Table 3, Caucasian mothers' punitiveness toward their toddler-age sons did not explain HAB in middle childhood (see #7–#9, first and fourth columns of statistical values); however, HAB positively predicted AB in early adulthood, regardless of *MAOA* genotype (see #9, third and sixth columns of statistical values). A significant G × P interaction indicated that HAB positively predicted official arrest records, but only for Caucasian men carrying low-activity *MAOA* alleles (see #8, third and sixth columns of statistical values). As previously found in Choe et al. (2014), two G × E interactions indicated that among Caucasian men carrying low-activity *MAOA* alleles, high levels of maternal punitiveness in toddlerhood predicted greater violent attitudes in late adolescence (see #7, second and fifth columns of statistical values) and AB in early adulthood (see #9, second and fifth columns of statistical values). We only found G × E interactions for Caucasian men in models with HAB that replicated findings from Choe et al. (2014); however, we demonstrated a G × P interaction in which higher levels of HAB predicted greater arrests in early adulthood only in L-*MAOA* Caucasian men. Moreover, higher levels of HAB predicted greater AB for all Caucasian men.

Main effects and statistical interactions with ARG

Table 4 presents results of six multiple-group mediation models testing interactions between men's *MAOA* genotype, mothers' punitive caregiving, and men's ARG in relation to violent attitudes, official arrests, and AB. All tests of G × E

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

Table 3. *Multiple-group mediation models testing monoamine oxidase A (MAOA) genotype differences in associations between maternal punitiveness (MP) at age 1.5, hostile attributional bias (HAB) at age 10, violent attitudes at age 17, official arrests, and antisocial behavior at ages 20 and 22*

		Low-Activity *MAOA* Group's b (*SE*) β			High-Activity *MAOA* Group's b (*SE*) β					
Dependent Variable	Racial Group	MP → HAB	MP → DV	HAB → DV	MP → HAB	MP → DV	HAB → DV	$\Delta\chi^2$ (*df*)	Model χ^2 (*df*)	RMSEA SRMR
Violent attitudes	#1 African American	0.69 (0.25) .33** $R^2 = .11$	5.69 (2.18) .49** $R^2 = .32$	−2.69 (0.71) −.48***	0.69 (0.25) .19** $R^2 = .04$	5.69 (2.18) .25** $R^2 = .20$	−2.69 (0.71) −.42***	Null	0.72 (3)	0.000 0.034
	#7 Caucasian	0.00 (0.25) .00 $R^2 = .00$	6.94 (0.92) .51*** $R^2 = .28$	0.75 (0.49) .15	0.00 (0.25) .00 $R^2 = .00$	0.14 (1.04) .01 $R^2 = .04$	0.75 (0.49) .20	10.39 $p = .001$ (1)	0.67 (2)	0.000 0.028
Official arrests	#2 African American	0.59 (0.23) .29* $R^2 = .09$	0.02 (0.06) .08 $R^2 = .04$	0.02 (0.03) .15	0.59 (0.23) .16* $R^2 = .03$	0.02 (0.06) .04 $R^2 = .02$	0.02 (0.03) .12	Null	4.27 (3)	0.106 0.080 CFI = 0.57
	#8 Caucasian	0.02 (0.26) .01 $R^2 = .00$	0.05 (0.04) .13 $R^2 = .30$	0.07 (0.02) .53**	0.02 (0.26) .01 $R^2 = .00$	0.05 (0.04) .17 $R^2 = .04$	0.01 (0.01) .09	10.28 $p = .001$ (1)	1.81 (2)	0.000 0.043
Antisocial behavior	#3 African American	0.63 (0.25) .31* $R^2 = .10$	2.83 (1.22) .27* $R^2 = .07$	−0.64 (0.55) −.12	0.63 (0.25) .17* $R^2 = .03$	2.83 (1.22) .24* $R^2 = .08$	−0.64 (0.55) −.19	Null	1.54 (3)	0.000 0.048
	#9 Caucasian	0.04 (0.25) .02 $R^2 = .00$	3.52 (0.94) .34*** $R^2 = .19$	0.98 (0.34) .25**	0.04 (0.25) .01 $R^2 = .00$	0.12 (1.38) .01 $R^2 = .06$	0.98 (0.34) .25**	9.65 $p = .002$ (1)	2.72 (2)	0.083 0.044 CFI = 0.94

Note: Significant chi-square difference ($\Delta\chi^2$) tests indicate differences between low- and high-activity *MAOA* allele carriers; shaded cells are the unstandardized (b) and standardized (β) coefficients estimated separately for *MAOA* groups. Standard errors (*SE*) and *p* values are from b estimates, and R^2 values represent amount of variance in mediator or dependent variables explained by model. All comparative fit index (CFI) values equal 1.00 unless noted in the final column. Hash marks (#) with numbers indicate specific multiple-group mediation models summarized in Table 2. RMSEA, Root mean square error of approximation; SRMR, standardized root mean square residual.

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 4. *Multiple-group mediation models testing monoamine oxidase A (MAOA) genotype differences in associations between maternal punitiveness (MP) at age 1.5, aggressive response generation (ARG) at age 10, violent attitudes at age 17, official arrests, and antisocial behavior at ages 20 and 22*

		Low-Activity *MAOA* Group's b (*SE*) β			High-Activity *MAOA* Group's b (*SE*) β					
Dependent Variable	Racial Group	MP → ARG	MP → DV	ARG → DV	MP → ARG	MP → DV	ARG → DV	$\Delta\chi^2$ (*df*)	Model χ^2 (*df*)	RMSEA SRMR
Violent attitudes	#4 African American	1.31 (0.24) .69*** $R^2 = .48$	8.76 (3.00) .75** $R^2 = .30$	−3.40 (1.37) −.55*	−0.46 (0.46) −.14 $R^2 = .02$	1.34 (2.41) .06 $R^2 = .02$	−068 (1.64) −.10	38.17 $p < .001$ (3)	0.00 (0)	0.000 0.000
	#10 Caucasian	1.30 (0.17) .56*** $R^2 = .31$	6.10 (1.37) .44*** $R^2 = .25$	0.62 (0.68) .10	−0.25 (0.23) −.11 $R^2 = .01$	0.21 (1.06) .02 $R^2 = .01$	0.62 (0.68) .12	32.45 $p < .001$ (2)	0.12 (1)	0.000 0.012
Official arrests	#5 African American	1.25 (0.21) .68*** $R^2 = .46$	−0.08 (0.05) −.26† $R^2 = .10$	0.07 (0.02) .43**	−0.58 (0.37) −.17 $R^2 = .03$	0.19 (0.14) .30 $R^2 = .18$	0.07 (0.02) .36**	19.87 $p < .001$ (2)	0.01 (1)	0.000 0.002
	#11 Caucasian	1.30 (0.17) .56*** $R^2 = .31$	0.04 (0.03) .11 $R^2 = .03$	0.01 (0.02) .08	−0.24 (0.22) −.10 $R^2 = .01$	0.04 (0.03) .15 $R^2 = .03$	0.01 (0.02) .12	10.84 $p < .001$ (1)	1.54 (2)	0.000 0.030
Antisocial behavior	#6 African American	1.26 (0.22) .68*** $R^2 = .47$	2.00 (1.06) .19† $R^2 = .09$	0.78 (0.55) .14	−0.54 (0.44) −.16 $R^2 = .03$	2.00 (1.06) .17† $R^2 = .07$	0.78 (0.55) .22	44.10 $p < .001$ (1)	0.69 (2)	0.000 0.033
	#12 Caucasian	1.30 (0.17) .56*** $R^2 = .31$	3.88 (1.48) .36** $R^2 = .11$	−0.28 (0.78) −.06	−0.24 (0.22) −.10 $R^2 = .01$	0.00 (1.51) .00 $R^2 = .003$	−0.28 (0.78) −.05	21.13 $p < .001$ (2)	0.48 (1)	0.000 0.024

Note: Significant chi-square difference ($\Delta\chi^2$) tests indicate differences between low- and high-activity *MAOA* allele carriers; shaded cells are the unstandardized (b) and standardized (β) coefficients estimated separately for *MAOA* groups. Standard errors (*SE*) and *p* values are from b estimates, and R^2 values represent amount of variance in mediator or dependent variables explained by model. All comparative fit index (CFI) values equal 1.00. Hash marks (#) with numbers indicate specific multiple-group mediation models summarized in Table 2. RMSEA, Root mean square error of approximation; SRMR, standardized root mean square residual.
†$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

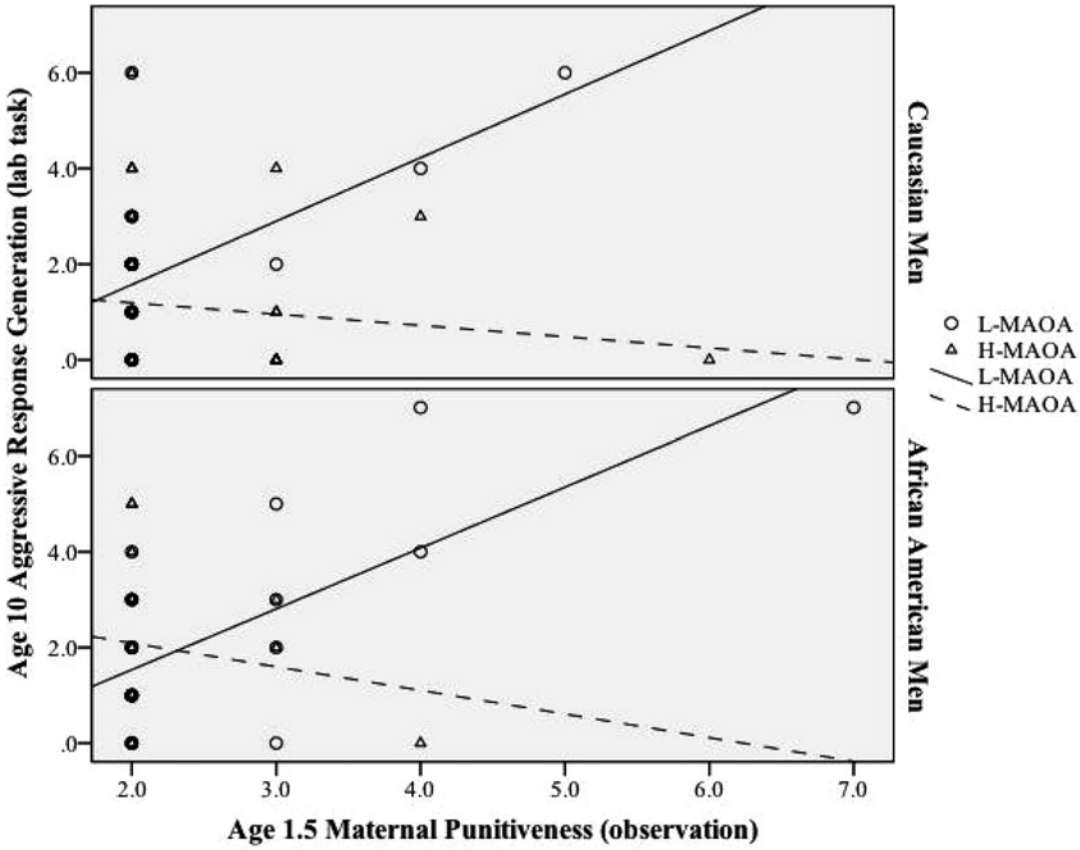

Figure 2. Scatterplots with best fitting regression lines show associations between laboratory paradigm-assessed aggressive response generation at age 10 and maternal punitiveness observed at age 1.5 by monoamine oxidase A (*MAOA*) genotype (circles and solid lines are low-activity *MAOA* (L-*MAOA*) carriers' data points and regression lines, respectively; triangles and dashed lines are high-activity *MAOA* (H-*MAOA*) carriers' data points and lines). There are separate plots for African American and Caucasian men. The top plot shows a positive linear association between maternal punitiveness and aggressive response generation for L-*MAOA* Caucasian men ($R^2 = .34$) but not for H-*MAOA* Caucasian men ($R^2 = .01$). The bottom plot similarly shows a positive association between maternal punitiveness and aggressive response generation for L-*MAOA* African American men ($R^2 = .52$) but not for H-*MAOA* African American men ($R^2 = .02$).

interactions between *MAOA* and maternal punitiveness in relation to ARG were significant (see Table 4 #4–#6 and #10–#12, first and fourth columns of statistical values). As expected and among both AA men and Caucasian men, maternal punitiveness in toddlerhood positively predicted ARG in middle childhood but only for men carrying low-activity *MAOA* alleles. Models explained between 31% and 48% of variance in L-*MAOA* men's ARG and about 2% of variance in H-*MAOA* men's ARG. Although multiple-group models provide a means of testing G × E interactions, they do not visually plot them like when probing interaction terms in ordinary least squares regression. Therefore, we created scatter plots with best fitting regression lines in SPSS that illustrate the reported G × E interactions between maternal punitiveness and ARG for each racial group (see Figure 2).

In models with ARG, we again found evidence of G × E interactions between *MAOA* and maternal punitiveness in relation to violent attitudes and AB identified in Choe et al. (2014) and reported in the HAB results for Caucasian men (see Table 4 #10 and #12, second and fifth columns of statistical values). We also found a significant G × E interaction between *MAOA* and maternal punitiveness in relation to violent attitudes for AA men (see #4, second and fifth columns of statistical values), which is surprising because this statistical interaction was not significant in Choe et al. (2014) or in our HAB model in which maternal punitiveness had a main effect on AA men's violent attitudes. Differences across models' results may be due to variation in third variables (i.e., HAB and early externalizing behavior). Nonetheless, maternal punitiveness positively predicted violent attitudes for L-*MAOA* men but not for H-*MAOA* men, regardless of race.

In terms of main effects, ARG positively predicted official arrests for AA men (see Table 4, #5, third and sixth columns of statistical values) but was unrelated to AA men's self-reported AB (see #6, third and sixth columns of statistical values) and Caucasian men's antisocial outcomes (see #10–#12, third and sixth columns of statistical values). Regardless of *MAOA* genotype, maternal punitiveness was unrelated to AA men's official arrests and AB (see #5–#6, second and fifth columns of statistical values), and Caucasian men's official arrests (see #11, second and fifth columns of statistical values). High levels of ARG predicted greater arrests among

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

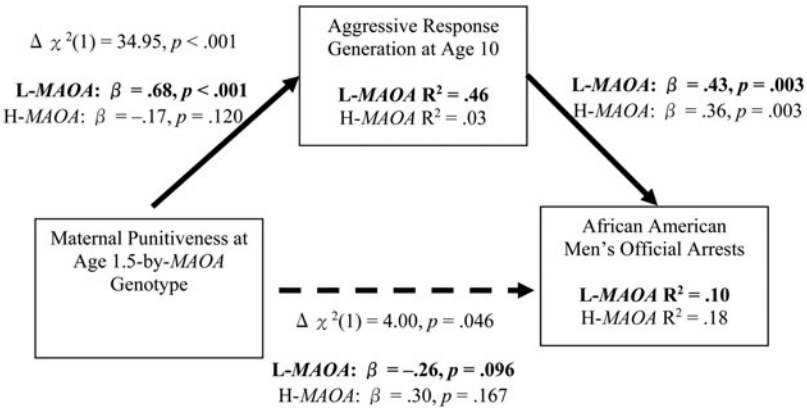

Figure 3. Model χ^2 (1) = 0.01, p = .946; comparative fit index = 1.00; root mean square error of approximation = 0.00, 95% CI [0.00, 0.07]; standardized root mean square residual = 0.002. Path coefficients for African American men with low-activity *MAOA* (L-*MAOA*, n = 35) are displayed in bold text above estimates for African American men with high-activity *MAOA* (H-*MAOA*, n = 41). A significant chi-square difference test ($\Delta\chi^2$) indicates *MAOA* genotype differences in model fit and statistical evidence of a gene–environment interaction. Standardized path coefficients and unstandardized p values are shown. Dashed lines indicate nonsignificant coefficients for both groups. A bootstrap test (5,000) of mediation indicated a marginally significant indirect effect of maternal punitiveness on L-*MAOA* African American men's official arrest records in early adulthood through their aggressive response generation in middle childhood (β = 0.29, p = .060). The path coefficient from aggressive response generation to official arrests did not differ by *MAOA* genotype, $\Delta\chi^2$ (1) = .012, p = .732, so one coefficient was estimated for both groups (standardized estimates slightly differ but are statistically equivalent in unstandardized form).

all AA men but was unrelated to any antisocial outcome for Caucasian men.

A significant G × P interaction indicated that ARG negatively predicted violent attitudes for AA men with low-activity *MAOA* alleles but not for AA men with high-activity *MAOA* alleles (see Table 4, #4, third and sixth columns of statistical values). Similar to our finding that HAB negatively predicted violent attitudes for all AA men, high levels of ARG predicted fewer violent attitudes in L-*MAOA* AA men.

Evidence of mediated moderation with ARG

Model #5 from Tables 2 and 4 was the only multiple-group mediation model to produce even a marginally significant indirect effect. As shown in Figure 3, maternal punitiveness at age 1.5 positively predicted ARG at age 10 for L-*MAOA* AA men but not for H-*MAOA* AA men. When these path coefficients were estimated separately for L-*MAOA* and H-*MAOA* men, overall model fit was significantly better than a more restrictive model that estimated identical values for *MAOA* groups. ARG predicted all AA men's official arrest records in early adulthood, regardless of genotype. Maternal punitiveness at age 1.5 did not predict arrests among AA men carrying L-*MAOA* or H-*MAOA* alleles, although their coefficients differed from each other. Both moderated effects were computed in a model that fit the data significantly better than a model in which one set of path coefficients was estimated for both *MAOA* groups, $\Delta\chi^2(2) = 19.87$, $p < .001$. A bootstrap model indicated a trend-level indirect effect of maternal punitiveness on L-*MAOA* AA men's arrests in early adulthood via ARG at age 10 (β = 0.29, p = .060). This indirect effect was not significant for H-*MAOA* men (β = –0.06, p = .308). These findings modestly support *mediated moderation* as defined by Edwards and Lambert (2007); the path from maternal punitiveness to ARG varied by *MAOA* genotype, whereas the path from ARG to arrests was unaffected by *MAOA*. The effect of AA mothers' early punitive behavior on sons' ARG in middle childhood was moderated by *MAOA* genotype, such that greater maternal punitiveness only predicted more of L-*MAOA* AA boys' ARG, which in turn predicted more arrests for all AA men.

The same model for Caucasian men replicated the interaction between maternal punitiveness and *MAOA* in relation to ARG, but it failed to predict Caucasian men's arrests in early adulthood. The effect of Caucasian mothers' punitive behavior during toddlerhood on sons' ARG in middle childhood was moderated by *MAOA* genotype, such that early maternal punitiveness only predicted more ARG among L-*MAOA* Caucasian men. Caucasian men's risk of being arrested in early adulthood was not explained by maternal punitiveness. Because evidence of genotype–phenotype correlation for Caucasian men may confound evidence of G × P interactions, we ran multiple-group models for Caucasian men that simultaneously tested path coefficients from HAB and ARG to antisocial outcomes (see online-only supplementary figures). We found that HAB predicted official arrests and ARG predicted violent attitudes only for L-*MAOA* Caucasian men, while ARG and HAB were only correlated with each other for H-*MAOA* Caucasian men. Interactions between ARG, HAB, and *MAOA* could not be tested due to insufficient statistical power.

Discussion

Previous studies have demonstrated that *MAOA* and adverse caregiving environments in early childhood interact to predict

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

AB (e.g., Byrd & Manuck, 2013; Caspi et al., 2002; Choe et al., 2014), but the underlying mechanisms explaining this relation are unknown. The goal of the present study was to examine whether individual differences in SIP patterns in middle childhood mediated interactions between punitive parenting in toddlerhood and *MAOA* in relation to AB in late adolescence and early adulthood among a sample of low-income, ethnically diverse boys. We found partial support for this hypothesis among AA men, with ARG but not HAB playing a mediating role. First, the interaction of *MAOA* and HAB during middle childhood predicted arrest records among Caucasian but not AA men. The findings did not support HAB as a mediator of the interaction between *MAOA* and maternal punitiveness in predicting adult AB. With regard to ARG, the interaction between *MAOA* and maternal punitiveness predicted greater aggressive responses in both AA and Caucasian men during middle childhood. Specifically, the association between maternal punitiveness and ARG was significantly stronger in men with the L-*MAOA* versus the H-*MAOA* variant. Second, ARG positively predicted arrests in early adulthood in AA men regardless of genotype but did not predict AB in Caucasian men. Third, there was a marginally significant indirect effect of maternal punitiveness on L-*MAOA* AA's arrests in early adulthood via their ARG in middle childhood, but similar results were not found in the Caucasian subsample.

Indirect effect of Maternal Punitiveness × MAOA interaction on arrests through SIP

Perhaps the most novel finding was that ARG may account for the interactive effects of punitive parenting and *MAOA* on arrest history among low-income AA men, although this indirect effect measured over a period of two decades was only a marginal trend. Although studies of G × E interactions in relation to AB have proliferated in psychological research, the processes through which these factors exert their impact have received much less empirical attention. The findings from the present study suggest that SIP may be a mechanism by which genetic and environmental factors confer risk for AB. Although evidence for an indirect effect was only modest, findings converge with neuroimaging evidence indicating that activity in the dorsal anterior cingulate cortex during a social-exclusion task mediates the *MAOA*–aggression link (Meyer-Lindenberg et al., 2006). Specifically, individuals with the low-expression *MAOA* allele were more affected by negative social situations than individuals with the high-expression *MAOA* allele, demonstrating higher levels of interpersonal hypersensitivity and heightened dorsal anterior cingulate cortex activity to social rejection. Meyer-Lindenberg et al. (2006) also demonstrated that individuals with the L-*MAOA* variant showed amygdala hyperreactivity to threatening and aversive emotional stimuli. Thus, although meditational findings in the present study were only marginally significant, taken together with the preceding neuroimaging evidence, findings suggest that *MAOA* may interact with adverse environmental contexts to disrupt the threat detection system, as indicated by maladaptive SIP and altered brain functioning, which then leads to serious and lasting antisocial tendencies. It is possible that the marginal mediation effect may have only been found in the AA subsample because of AA youths' greater variability in arrest records in the present sample, which could have been explained by their SIP patterns. In addition, based on the large number of years between assessments of parenting in early childhood and assessments of AB in early adulthood, as well as the relatively small sample size for identifying G × E interactions, the current findings likely underestimate the contribution of maladaptive SIP as a mediator. Of note, there was also evidence of selective attrition, with those lost to attrition scoring higher on ARG than the retained sample. It is possible that these men may have been lost to imprisonment or homicide, suggesting that the failure to include these men in the present analyses may have also underestimated maladaptive SIP as a mediator.

The interaction between MAOA and maternal punitiveness in predicting SIP

Extensive literature has demonstrated that severe parenting behaviors, such as physical abuse and other forms of maltreatment, predict maladaptive SIP. However, many family environments are characterized by frequent expressions and experiences of negative emotions, such as anger and distress, but not necessarily exposure or victimization to family violence. The present study extends the current work in the field by demonstrating that more normative parenting practices, not merely extreme social stressors, can play a strong role in tempering the role of genetic factors in the development of ARG. However, contrary to previous literature, maternal punitiveness was not directly linked to individual differences in ARG, but was found to interact with the *MAOA* polymorphism to amplify risk for maladaptive processing patterns. Although Dodge (2011) proposed that patterns of SIP are acquired through the interaction of early environmental adversity with "specific (albeit unidentified, as of yet) genes" (p. 22), to our knowledge the present study is the first of its kind to empirically identify evidence of such an interaction. These findings extend Caspi et al.'s (2002) original results, demonstrating that *MAOA* and harsh parenting not only interact to predict AB but also predict a well-established social–cognitive correlate of AB (Crick & Dodge, 1994).

Effect of SIP on AB in early adulthood

The results indicated that high levels of SIP problems at age 10 predicted a greater number of criminal arrests in early adulthood in AA men but not among Caucasian men. AA men who were more likely to generate aggressive responses to ambiguous social conflict were likely to be arrested by their early 20s. Choe et al. (2015) demonstrated similar findings, linking ARG to criminal history, although they did not examine this association separately by race. These findings are

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

consistent with the extensive body of literature indicating that SIP problems in childhood have long-standing consequences for adolescents' and adults' AB (e.g., Hyde, Shaw, & Moilanen, 2010).

Racial–ethnic differences in SIP

AA men displayed more maladaptive patterns of processing social information than Caucasian men, reporting more HAB and aggressive responses to hypothetical and ambiguous interpersonal conflicts. Racial–ethnic minority youth, particularly AAs, are disproportionately overrepresented in low-SES and disadvantaged neighborhoods, and these contexts may predispose some AA children to developing maladaptive patterns of social–cognitive processing (Duncan, Brooks-Gunn, & Klebanov, 1994). Economically impoverished neighborhoods characterized by high levels of community violence and crime expose youth to more hostile attitudes and more serious forms of AB. Consistent with SIP and social learning theories, frequent exposure to anger and hostility in the neighborhood may repeatedly evoke threat responses and facilitate children's hypervigilance and internalization of hostile schemas (Bandura, 1973; Crick & Dodge, 1994). In support of these interpretations, emerging neuroimaging findings indicate heightened amygdala response to angry facial expresses among children and adolescents from lower SES backgrounds (Muscatell et al., 2012). In addition, children from lower SES families were more likely to appraise ambiguous social situations as negative or hostile in intent (Chen, Langer, Raphaelson, & Matthews, 2004). These findings suggest that socioeconomic disadvantage may alter neural stress responses to threat that manifests as heightened sensitivity to threat cues.

Racial disparities in exposure to neighborhood dangerousness and to experiences of discrimination may also lead AA parents to socialize their children in particular ways that lead to different perceptions of the world (Dodge, Pettit, & Bates, 1994). Specifically, in efforts to promote safety and protection in dangerous and threatening neighborhoods, parents may encourage their children to make automatic attributions of threat to others, even encouraging aggression as a legitimate and functional means of self-protection (Coie & Dodge, 1998). Nonetheless, current findings link HAB in middle childhood to AB in early adulthood among Caucasian, but not AA men. Thus, despite racial–ethnic differences in the frequency of SIP problems, tendencies to attribute hostile intent to others appears to be a more robust risk factor of AB for Caucasians than for AA youth. As alluded to earlier, it is possible that AA boys are more likely to cultivate aggressive response patterns or HAB to adapt to their surroundings and ensure their survival based on their increased likelihood of residing in dangerous and hostile neighborhoods (see Racial group differences subsection in the Results section). For example, although children with a HAB tend to misinterpret some threat cues, they are better able to detect real signs of threat when they do occur, emphasizing that context partly determines whether the behavior is adaptive or maladaptive. Thus, while the detection of hostility may be functional in threatening environments, such as dangerous neighborhoods, readily accessing aggressive solutions from memory or by habit in response to perceived or actual threats still confers increased risk for AB.

Although HAB and ARG often occur in parallel (Crick & Dodge, 1994), the current results suggest that these dimensions of SIP are distinct from one another based on the more consistent pattern of findings for ARG. These findings align with previous research suggesting different neural substrates for HAB and ARG, with ARG but not HAB predicting increased amygdala reactivity to ambiguous social threat cues (Choe et al., 2015). Although HAB and ARG are often aggregated into a single index of "maladaptive SIP" (Orobio de Castro et al., 2002), the present findings underscore the importance of examining these dimensions separately, particularly in racially diverse samples.

One unexpected finding of the present study was that HAB was unrelated to maternal punitiveness in toddlerhood. This was surprising, as research indicates that children exposed to family abuse and violence selectively attend to hostile cues (Pollak & Tolley-Schell, 2003) and exhibit an interpretational bias toward perceiving others as angry (Pollak et al., 1997). Nonetheless, the present study's findings are consistent with those of Schultz and Shaw (2003), who demonstrated that HAB was not predicted by early family risk factors in the current sample. One possibility is that our methodology did not adequately assess atypical hostile attributions for the present study's sample. Use of in vivo, experimental manipulations of ambiguous peer scenarios may better capture children's implicit attributions as they occur during interpersonal exchanges in real time. Similar to other samples using the same methodology (e.g., Dodge, Laird, Lochman, & Zelli, 2002), children in the present sample attributed hostile intent to the majority of peer provocateurs. Perhaps because of the low-SES status of our participants, the attribution of hostility was a normative tendency, making it difficult to identify a family risk factor that contributed uniquely to later HAB. Future research to resolve these discrepancies is needed, with important considerations of racial, socioeconomic, developmental, and cultural contexts.

Limitations

A few caveats to this study warrant further consideration. First, the present study included only modest numbers of young men relative to large-scale epidemiological investigations. However, power to detect associations was enhanced because the sample was at high risk for maternal punitiveness and research suggests that studies with samples smaller than Caspi et al.'s (2002; $N = 1{,}037$) are no less likely to replicate their G × E findings (Byrd & Manuck, 2013). A smaller sample permitted longitudinal measurement spanning over 20 years, investigation of multiple developmental periods, and rigorous assessment of parenting using observational methods, SIP using a laboratory paradigm, and AB using

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press

both self-reports and court record data. These advantages have posed longstanding challenges in the genetic epidemiology of complex phenotypes. Nonetheless, the small sample may have limited our ability to detect an indirect effect of maternal punitiveness on L-*MAOA* men's AB in early adulthood through SIP. In addition, while a subgroup approach to combining moderation and mediation is recommended in structural equation modeling, the use of subgroups inherently results in lower statistical power (Edwards & Lambert, 2007). Thus, the associations reported herein necessitate cross-validation in independent samples. Despite sample size limitations, use of a stringent false discovery rate controlling procedure protects against the likelihood of Type 1 error and increases confidence in our findings.

Second, the current sample was originally recruited from Women, Infants, and Children Program nutrition supplement centers in a single metropolitan area, and thus, families faced financial hardships and other correlates of financial adversity (e.g., living in poor neighborhoods). The present sample was recruited because male children from low-SES backgrounds are at a greater risk for showing meaningful levels of AB, but findings may not generalize to children from nonurban, higher SES families. Third and relatedly, the sample only included men. Relative to males, there are far fewer data relating to *MAOA* functional variants in females, possibly resulting from their extensive variability in X-linked gene expression (Carrel & Willard, 2005). Research suggests a possible reversal of allelic associations in females such that the high-activity, rather than the low-activity, *MAOA* genotype may interact with childhood maltreatment to confer increased risk for antisocial outcomes (Byrd & Manuck, 2013). In addition, the nature of SIP has received less empirical attention in girls than in boys, although existing literature suggests that girls who do demonstrate maladaptive patterns of SIP are comparably at risk for the same aggressive outcomes as their male counterparts (Dodge et al., 2003; Zelli, Dodge, Lochman, & Laird, 1999). Thus, examining whether current findings also generalize to girls is warranted. Nonetheless, keeping this limitation in mind, the use of an all-male sample was deemed suitable as males continue to outnumber females in frequency and seriousness of AB (American Psychiatric Association, 2013).

Third, although use of a racially diverse sample is advantageous in several ways, this heterogeneity may also pose limitations in genetic association studies. Like the current investigation, other studies have also reported racial–ethnic differences in both *MAOA* allele distribution and the effect of *MAOA* on youth outcomes (Reti et al., 2011; Sabol et al., 1998). This may confound tests of G × E interactions in mixed-race samples (Hutchison, Stallings, McGeary, & Bryan, 2004) and account for discrepant findings in the literature. While various strategies are employed to detect and account for racial–ethnic admixture, race-specific analyses reduce confounds of racial–ethnic variation in allele frequencies (Zintzaras & Lau, 2008).

Clinical implications and future directions

In spite of these limitations, few studies have examined the mechanisms by which genes and environments interact to predict the development of AB. To our knowledge, this is the first study to provide empirical evidence suggesting that maladaptive patterns of processing social information may be a mechanism by which this risk is conferred. The current study was designed to maximize important considerations in developmental and G × E research, including use of multiple assessment methods, a prospective longitudinal design, and high levels of retention over 20 years.

Generally, once a stable aggressive behavioral response pattern or HAB has developed, forces operate to maintain these patterns across development (Dodge, 2006). Nonetheless, intervention programs focusing explicitly on modifying attributional styles show promise (e.g., Sukhodolsky, Golub, Stone, & Orban, 2005), and the present findings encourage further development and implementation of attributional and response retraining interventions. While effective interventions for AB have been developed, targeting specific processes, particularly social–cognitive skills related to the development of AB, may lead to more cost-effective and robust intervention efforts in reducing aggressive and antisocial tendencies, especially among individuals at high risk due to gene–environment risk profiles. Future work is needed to corroborate the current findings and to identity additional neural and cognitive mechanisms to provide a clearer picture of the sensitivities that mediate G × E interactions.

Supplementary Material

To view the supplementary material for this article, please visit https://doi.org/10.1017/S0954579416001279.

References

Aguilar, B., Sroufe, L. A., Egeland, B., & Carlson, E. (2000). Distinguishing the early-onset/persistent and adolescence-onset antisocial behavior types: From birth to 16 years. *Development and Psychopathology*, *12*, 109–132.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Bandura, A. (1973). *Aggression: A social learning analysis*. Englewood Cliffs, NJ: Prentice-Hall.

Beaver, K. M., DeLisi, M., Vaughn, M. G., & Barnes, J. C. (2010). Monoamine oxidase A genotype is associated with gang membership and weapon use. *Comprehensive Psychiatry*, *51*, 130–134.

Beaver, K. M., Nedelec, J. L., Wilde, M., Lippoff, C., & Jackson, D. (2011). Examining the association between MAOA genotype and incarceration, anger and hostility: The moderating influences of risk and protective factors. *Journal of Research in Personality*, *45*, 279–284.

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society*, *57*, 289–300.

Bongers, I. L., Koot, H. M., van der Ende, J., & Verhulst, F. C. (2004). Developmental trajectories of externalizing behaviors in childhood and adolescence. *Child Development*, *75*, 1523–1537.

Burnette, M. L., Oshri, A., Lax, R., Richards, D., & Ragbeer, S. N. (2012). Pathways from harsh parenting to adolescent antisocial behavior: A multidomain test of gender moderation. *Development and Psychopathology*, *24*, 857–870.

Burt, S. A., & Mikolajewski, A. J. (2008). Preliminary evidence that specific candidate genes are associated with adolescent-onset antisocial behavior. *Aggressive Behavior*, *34*, 437–445.

Byrd, A. L., & Manuck, S. B. (2013). MAOA, childhood maltreatment, and antisocial behavior: Meta-analysis of a gene-environment interaction. *Biological Psychiatry*, *75*, 9–17.

Campbell, S. B., Shaw, D. S., & Gilliom, M. (2000). Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Development and Psychopathology*, *12*, 467–488.

Carrel, L., & Willard, H. F. (2005). X-inactivation profile reveals extensive variability in X-linked gene expression in females. *Nature*, *434*, 400–404.

Caspi, A., McClay, J., Moffitt, T., Mill, J., Martin, J., Craig, I. W., . . . Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 851–854.

Chen, E., Langer, D. A., Raphaelson, Y. E., & Matthews, K. A. (2004). Socioeconomic status and health in adolescents: The role of stress interpretations. *Child Development*, *75*, 1039–1052.

Choe, D. E., Shaw, D. S., & Forbes, E. E. (2015). Maladaptive social information processing in childhood predicts young men's atypical amygdala reactivity to threat. *Journal of Child Psychology and Psychiatry*, *56*, 549–557.

Choe, D. E., Shaw, D. S., Hyde, L. W., & Forbes, E. E. (2014). Interactions between monoamine oxidase A and punitive discipline in African American and Caucasian men's antisocial behavior. *Clinical Psychological Science*, *2*, 591–601.

Coie, J. D., & Dodge, K. A. (1998). Aggression and antisocial behavior. In W. Damon & N. Eisenberg (Eds.), *Handbook of child psychology: Social, emotional, and personality development* (Vol. 3, pp. 779–862). Toronto: Wiley.

Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin*, *115*, 74–101.

Crittenden, P. M., & Ainsworth, M. D. S. (1989). Child maltreatment and attachment theory. In D. Cicchetti & V. Carlson (Eds.), *Handbook of child maltreatment* (pp. 432–463). New York: Cambridge University Press.

Cummings, E. M., & Davies, P. T. (1994). Maternal depression and child development. *Child Psychology and Psychiatry, and Allied Disciplines*, *35*, 73–112.

DNA Genotek Inc. (2006). *Oragene*™ *product brochure*. Ottawa: Author.

Dodge, K. A. (2006). Translational science in action: Hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology*, *18*, 791–814.

Dodge, K. A. (2009). Mechanisms of the gene-environment interaction effects in the development of conduct disorder. *Perspectives on Psychological Science*, *4*, 408–414.

Dodge, K. A. (2011). Social information processing patterns as mediators of the interaction between genetic factors and life experiences in the development of aggressive behavior. In M. Mikulincer & P. R. Shaver (Eds.), *Human aggression and violence: Causes, manifestations, and consequences* (pp. 165–185). Washington, DC: American Psychological Association.

Dodge, K. A., Laird, R., Lochman, J. E., & Zelli, A. (2002). Multidimensional latent-construct analysis of children's social information processing patterns: Correlations with aggressive behavior problems. *Psychological Assessment*, *14*, 60–73.

Dodge, K. A., Lansford, J. E., Burks, V. S., Bates, J. E., Pettit, G. S., Fontaine, R., & Price, J. M. (2003). Peer rejection and social information-processing factors in the development of aggressive behavior problems in children. *Child Development*, *74*, 374–393.

Dodge, K. A., & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology*, *39*, 349–371.

Dodge, K. A., Pettit, G. S., & Bates, J. E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development*, *65*, 649–665.

Dodge, K. A., Pettit, G. S., Bates, J. E., & Valente, E. (1995). Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. *Journal of Abnormal Psychology*, *104*, 632–643.

Dodge, K. A., Price, J. M., Bachorowski, J., & Newman, J. P. (1990). Hostile attributional biases in severely aggressive adolescents. *Journal of Abnormal Psychology*, *99*, 385–392.

Dodge, K. A., & Somberg, D. R. (1987). Hostile attributional biases among aggressive boys are exacerbated under conditions of threats to the self. *Child Development*, *58*, 213–224.

Downey, G., & Walker, E. (1989). Social cognition and adjustment in children at risk for psychopathology. *Developmental Psychology*, *25*, 835–845.

Duncan, G. J., Brooks-Gunn, J., & Klebanov, P. K. (1994). Economic deprivation and early childhood development. *Child Development*, *65*, 296–318.

Edens, J. F., Skopp, N. A., & Cahill, M. A. (2008). Psychopathic features moderate the relationship between harsh and inconsistent parental discipline and adolescent antisocial behavior. *Journal of Clinical Child and Adolescent Psychology*, *37*, 472–476.

Edwards, J. R., & Lambert, L. S. (2007). Methods for integrating moderation and mediation: A general analytical framework using moderated path analysis. *Psychological Methods*, *12*, 1.

Egeland, B., Kalkoske, M., Gottesman, N., & Erickson, M. F. (1990). Preschool behavior problems: Stability and factors accounting for change. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *31*, 891–909.

Eisenberger, N. I., Way, B. M., Taylor, S. E., Welch, W. T., & Lieberman, M. D. (2007). Understanding genetic risk for aggression: Clues from the brain's response to social exclusion. *Biological Psychiatry*, *61*, 1100–1108.

Elliott, D. S., Huizinga, D., & Ageton, S. S. (1985). *Explaining delinquency and drug use*. Thousand Oaks, CA: Sage.

Enoch, M., Shen, P., Xu, K., Hodgkinson, C., & Goldman, D. (2006). Using ancestry-informative markers to define populations and detect population stratification. *Journal of Psychopharmacology*, *20*, 19–26.

Ferguson, C. J. (2010). Genetic contributions to antisocial personality and behavior: A meta-analytic review from an evolutionary perspective. *Journal of Social Psychology*, *150*, 160–180.

Fergusson, D. M., Horwood, L. J., & Ridder, E. M. (2005). Show me the child at seven: The consequences of conduct problems in childhood for psychosocial functioning in adulthood. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *46*, 837–849.

Frazzetto, G., Di Lorenzo, G., Carola, V., Proietti, L., Sokolowska, E., Siracusano, A., . . . Troisi, A. (2007). Early trauma and increased risk for physical aggression during adulthood: The moderating role of MAOA genotype. *PLOS ONE*, *2*, e486.

Funk, J. B., Elliott, R., Urman, M. L., Flores, G. T., & Mock, R. M. (1999). The attitudes towards violence scale: A measure for adolescents. *Journal of Interpersonal Violence*, *14*, 1123–1136.

Gershoff, E. T. (2002). Corporal punishment by parents and associated child behaviors and experiences: A meta-analytic and theoretical review. *Psychological Bulletin*, *128*, 539–579.

Guerra, N. G., & Slaby, R. G. (1989). Evaluative factors in social problem solving by aggressive boys. *Journal of Abnormal Child Psychology*, *17*, 277–289.

Gunter, T. D., Vaughn, M. G., & Philibert, R. A. (2010). Behavioral genetics in antisocial spectrum disorders and psychopathy: A review of the recent literature. *Behavioral Sciences and the Law*, *28*, 148–173.

Guo, G., Roettger, M. E., & Shih, J. C. (2007). Contributions of the DAT1 and DRD2 genes to serious and violent delinquency among adolescents and young adults. *Human Genetics*, *121*, 125–136.

Hayes, A. F. (2009). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication Monographs*, *76*, 408–420.

Hodgkinson, C. A., Yuan, Q., Xu, K., Pei-hong, S., Heinz, E., Lobos, E. A., . . . Goldman, D. (2008). Addictions biology: Haplotype-based analysis for 130 candidate genes on a single array. *Alcohol and Alcoholisms*, *43*, 505–515.

Hollingshead, A. (1975). *Four Factor Index of Social Status*. Unpublished manuscript, Yale University.

Hutchison, K. E., Stallings, M., McGeary, J., & Bryan, A. (2004). Population stratification in the candidate gene study: Fatal threat or red herring? *Psychological Bulletin*, *130*, 66–79.

Hyde, L. W., Shaw, D. S., & Moilanen, K. L. (2010). Developmental precursors of moral disengagement and the role of moral disengagement in the development of antisocial behavior. *Journal of Abnormal Child Psychology*, *38*, 197–209.

Kendler, K. S., & Eaves, L. J. (1986). Models for the joint effect of genotype and environment on liability to psychiatric illness. *American Journal of Psychiatry*, *143*, 279–289.

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., . . . Kendler, K. S. (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States: Results from the national comorbidity study. *Archives of General Psychiatry*, *51*, 8–19.

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W., & Moffitt, T. E. (2006). MAOA, maltreatment, and gene-environment interaction predicting children's mental health: New evidence and a meta-analysis. *Molecular Psychiatry*, *11*, 903–913.

Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2nd ed.). New York: Guilford Press.

Leventhal, T., & Brooks-Gunn, J. (2000). The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin*, *126*, 309–337.

Levy, E. (1989). Localization of human monoamine oxidase-A gene to Xp11.23-11.4 by in situ hybridization: Implications for Norrie disease. *Genomics*, *5*, 368–370.

Lochman, J. E. (1987). Self- and peer perceptions and attributional biases of aggressive and nonaggressive boys in dyadic interactions. *Journal of Consulting and Clinical Psychology*, *55*, 404–410.

Loeber, R. (1982). The stability of antisocial and delinquent child behavior: A review. *Child Development*, *53*, 1431–1446.

Loeber, R., & Stouthamer-Loeber, M. (1998). Development of juvenile aggression and violence. *American Psychologist*, *53*, 242–259.

Meyer-Lindenberg, A., Buckholtz, J. W., Kolachana, B., Hariri, A. R., Pezawas, L., Blasi, G., . . . Weinberger, D. R. (2006). Neural mechanisms of genetic risk for impulsivity and violence in humans. *Proceedings of the National Academy of Sciences*, *103*, 6269–6274.

Moffitt, T. E. (2005). The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. *Psychological Bulletin*, *131*, 533–554.

Muscatell, K. A., Morelli, S. A., Falk, E. B., Way, B. M., Pfeifer, J. H., Galinsky, A. D., . . . Eisenberger, N. I. (2012). Social status modulates neural activity in the mentalizing network. *NeuroImage*, *60*, 1771–1777.

Odgers, C. L., Moffitt, T. E., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., . . . Caspi, A. (2008). Female and male antisocial trajectories: From childhood origins to adult outcomes. *Development and Psychopathology*, *20*, 673–716.

Orobio de Castro, B., Veerman, J. W., Koops, W., Bosch, J. D., & Monshouwer, H. J. (2002). Hostile attribution of intent and aggressive behavior: A meta-analysis. *Child Development*, *73*, 916–934.

Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. *Developmental Psychology*, *36*, 679–688.

Pollak, S. D., Cicchetti, D., Klorman, R., & Brumaghim, J. T. (1997). Cognitive brain event-related potentials and emotion processing in maltreated children. *Child Development*, *68*, 773–787.

Pollak, S. D., & Tolley-Schell, S. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology*, *112*, 323–338.

Reid, M. J., Webster-Stratton, C., & Baydar, N. (2004). Halting the development of conduct problems in Head Start children: The effects of parent training. *Journal of Clinical Child and Adolescent Psychology*, *33*, 279–291.

Reif, A., Rösler, M., Freitag, C. M., Schneider, M., Eujen, A., Kissling, C., . . . Retz, W. (2007). Nature and nurture predispose to violent behavior: Serotonergic genes and adverse childhood environment. *Neuropsychopharmacology*, *32*, 2375–2383.

Reti, I. M., Xu, J. Z., Yanofski, J., McKibben, J., Uhart, M., Cheng, Y., . . . Nestadt, G. (2011). Monoamine oxidase a regulates antisocial personality in whites with no history of physical abuse. *Comprehensive Psychiatry*, *52*, 188–194.

Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin*, *128*, 490–529.

Rose, R. J., Dick, D. M., Viken, R. J., Pulkkinen, L., & Kaprio, J. (2004). Genetic and environmental effects on conduct disorder and alcohol dependence symptoms and their covariation at age 14. *Alcoholism: Clinical and Experimental Research*, *28*, 1541–1548.

Sabol, S. Z., Hu, S., & Hamer, D. (1998). A functional polymorphism in the monoamine oxidase a gene promoter. *Human Genetics*, *103*, 273–279.

Satorra, A., & Bentler, P. M. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika*, *66*, 507–514.

Schultz, D., Izard, C. E., & Ackerman, B. P. (2000). Children's anger attribution bias: Relations to family environment and social adjustment. *Social Development*, *9*, 284–301.

Schultz, D., & Shaw, D. S. (2003). Boys' maladaptive social information processing, family emotional climate, and pathways to early conduct problems. *Social Development*, *12*, 440–460.

Schwartz, D., & Proctor, L. J. (2000). Community violence exposure and children's social adjustment in the school peer group: The mediating roles of emotion regulation and social cognition. *Journal of Consulting and Clinical Psychology*, *68*, 670–683.

Shaw, D. S., Bell, R. Q., & Gilliom, M. (2000). A truly early starter model of antisocial behavior revisited. *Clinical Child and Family Psychology Review*, *3*, 155–172.

Shaw, D. S., Criss, M. M., Schonberg, M. A., & Beck, J. E. (2004). The development of family hierarchies and their relation to children's conduct problems. *Development and Psychopathology*, *16*, 483–500.

Shaw, D. S., Gilliom, M., Ingoldsby, E. M., & Nagin, D. S. (2003). Trajectories leading to school-age conduct problems. *Developmental Psychology*, *39*, 189–200.

Shaw, D. S., & Gross, H. (2008). Early childhood and the development of delinquency: What we have learned from longitudinal research. In A. Lieberman (Ed.), *The long view of crime: A synthesis of longitudinal research* (pp. 79–127). New York: Springer.

Shaw, D. S., Hyde, L. W., & Brennan, L. M. (2012). Early predictors of boys' antisocial trajectories. *Development and Psychopathology*, *24*, 871–888.

Shaw, D. S., Winslow, E. B., Owens, E. B., Vondra, J. I., Cohn, J. F., & Bell, R. Q. (1998). The development of early externalizing problems among children from low-income families: A transformational perspective. *Journal of Abnormal Child Psychology*, *26*, 95–107.

Slaby, R. G., & Guerra, N. G. (1988). Cognitive mediators of aggression in adolescent offenders: I. Assessment. *Developmental Psychology*, *24*, 580–588.

Sukhodolsky, D. G., Golub, A., Stone, E. C., & Orban, L. (2005). Dismantling anger control training for children: A randomized pilot study of social problem-solving versus social skills training components. *Behavior Therapy*, *36*, 15–23.

Taylor, A., & Kim-Cohen, J. (2007). Meta-analysis of gene–environment interactions in developmental psychopathology. *Development and Psychopathology*, *19*, 1029–1037.

Thomas, D. E., Bierman, K. L., Thompson, C., Powers, C. J., & Conduct Problems Prevention Research Goup. (2008). Double jeopardy: Child and school characteristics that predict aggressive-disruptive behavior in first grade. *School Psychology Review*, *37*, 516–532.

Trentacosta, C. J., & Shaw, D. S. (2009). Emotional self-regulation, peer rejection, and antisocial behavior: Developmental associations from early childhood to early adolescence. *Journal of Applied Developmental Psychology*, *30*, 356–365.

Waldman, I. D., & Rhee, S. H. (2006). Genetic and environmental influences on psychopathy and antisocial behavior. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 205–228). New York: Guilford Press.

Weiss, B., Dodge, K. A., Bates, J. E., & Pettit, G. S. (1992). Some consequences of early harsh discipline: Child aggression and a maladaptive social information processing style. *Child Development*, *63*, 1321–1335.

Widom, C. S., & Brzustowicz, L. M. (2006). MAOA and the "cycle of violence": Childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. *Biological Psychiatry*, *60*, 684–689.

Winslow, E., & Shaw, D.S. (1995). *Early Parenting Coding System*. Unpublished manuscript, University of Pittsburgh.

Yung-yu, H., Cate, S. P., Battistuzzi, C., Oquendo, M. A., Brent, D., & Mann, J. J. (2004). An association between a functional polymorphism in the monoamine oxidase A gene promoter, impulsive traits and early abuse experiences. *Neuropsychopharmacology*, *29*, 1498–1505.

Zahn-Waxler, C., Iannotti, R. J., Cummings, E. M., & Denham, S. (1990). Antecedents of problem behaviors in children of depressed mothers. *Development and Psychopathology*, *2*, 271–291.

Zelli, A., Dodge, K. A., Lochman, J. E., & Laird, R. D. (1999). The distinction between beliefs legitimizing aggression and deviant processing of social cues: Testing measurement validity and the hypothesis that biased processing mediates the effects of beliefs on aggression. Conduct Problems Prevention Research Group. *Journal of Personality and Social Psychology*, *77*, 150–166.

Zintzaras, E., & Lau, J. (2008). Synthesis of genetic association studies for pertinent gene-disease associations requires appropriate methodological and statistical approaches. *Journal of Clinical Epidemiology*, *61*, 634–645.

Appendix A

Racial group differences in study variables

Racial Group	1. MP at Age 1.5	2. HAB at Age 10	3. ARG at Age 10	4. VA at Age 17	5. OAR in EA	6. AB at Ages 20 and 22
1. African American men	$M = 1.15$, $SD = 0.38$	$M = 5.35$, $SD = 1.81$	$M = 2.05$, $SD = 1.72$	$M = 35.60$, $SD = 10.84$	$M = 0.20$, $SD = 0.28$	$M = 10.57$, $SD = 7.67$
2. Caucasian American men	$M = 1.12$, $SD = 0.33$	$M = 4.33$, $SD = 2.07$	$M = 1.35$, $SD = 1.57$	$M = 33.79$, $SD = 8.28$	$M = 0.09$, $SD = 0.19$	$M = 9.03$, $SD = 8.23$
t-test score	t (173) = –.47, $p = .642$	t (139) = –3.00, $p = .003$	t (139) = –2.51, $p = .013$	t (122.25) = −1.18, $p = .241$	t (123.39) = −3.19, $p = .002$	t (171) = 1.26, $p = .210$
95% CI	[–0.13, 0.08]	[–1.69, –0.35]	[–1.26, –0.15]	[–4.85, 1.23]	[–0.19, –0.05]	[–0.87, 3.93]

Note: Degrees of freedom (*df*) that include decimals indicate *t* tests that violated the homogeneity of variance assumption, which we corrected for with the use of Welch *t* tests. MP, Maternal punitiveness; HAB, hostile attributional bias; ARG, aggressive response generation; VA, violent attitudes; OAR, official arrest record; EA, early adulthood; AB, antisocial behavior.

Appendix B

Monoamine oxidase A genotype differences in study variables for African American and Caucasian men

Racial Group	1. MP at Age 1.5	2. HAB at Age 10	3. ARG at Age 10	5. VA at Age 17	6. OAR in EA	7. AB at Ages 20 and 22
1. AA men						
L-*MAOA*	$M = 1.21$, $SD = .49$	$M = 5.22$, $SD = 1.99$	$M = 2.15$, $SD = 1.92$	$M = 34.55$, $SD = 11.50$	$M = 0.20$, $SD = 0.29$	$M = 10.95$, $SD = 10.59$
H-*MAOA*	$M = 1.09$, $SD = 0.23$	$M = 5.46$, $SD = 1.64$	$M = 1.96$, $SD = 1.53$	$M = 36.54$, $SD = 10.28$	$M = 0.20$, $SD = 0.28$	$M = 7.45$, $SD = 5.23$
t-test score	t (46.70) = 1.38, $p = .174$	t (53) = –0.49, $p = .624$	t (53) = 0.39, $p = .695$	t (68) = –0.77, $p = .446$	t (74) = 0.04, $p = .970$	t (44.67) = 1.74, $p = .090$
95% CI	[–0.06, 0.31]	[–1.23, 0.74]	[–0.75, 1.12]	[–7.19, 3.20]	[–0.13, 0.13]	[–0.57, 7.57]
2. CA men						
L-*MAOA*	$M = 1.10$, $SD = 0.33$	$M = 5.17$, $SD = 1.75$	$M = 1.83$, $SD = 1.54$	$M = 34.57$, $SD = 9.10$	$M = 0.11$, $SD = 0.23$	$M = 11.60$, $SD = 7.02$
H-*MAOA*	$M = 1.14$, $SD = 0.33$	$M = 3.89$, $SD = 2.09$	$M = 1.11$, $SD = 1.54$	$M = 33.46$, $SD = 7.95$	$M = 0.07$, $SD = 0.17$	$M = 10.12$, $SD = 7.94$
t-test score	t (99) = –0.55, $p = .584$	t (84) = 2.82, $p = .006$	t (84) = 2.06, $p = .043$	t (99) = 0.61, $p = .544$	t (46.09) = 1.04, $p = .304$	t (98) = 0.88, $p = .380$
95% CI	[–0.18, 0.10]	[0.38, 2.18]	[0.02, 1.42]	[–2.49, 4.69]	[–0.04, 0.14]	[–1.85, 4.80]

Note: Degrees of freedom (*df*) that include decimals indicate *t*-tests that violated the homogeneity of variance assumption, which we corrected with the use of Welch *t*-test. *MAOA*, Monoamine oxidase A genotype; MP, maternal punitiveness; HAB, hostile attributional bias; ARG, aggressive response generation; VA, violent attitudes; OAR, official arrest record; EA, early adulthood; AB, antisocial behavior; AA, African American; CA, Caucasian American.

https://doi.org/10.1017/S0954579416001279 Published online by Cambridge University Press