

STRESS, GENES, AND GENERALIZABILITY ACROSS GENDER: EFFECTS OF MAOA AND STRESS SENSITIVITY ON CRIME AND DELINQUENCY*

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KEYWORDS: stress, antisocial behavior, biosocial, monoamine oxidase A (MAOA) gene

In the current study, we extend the gene-by-environment interaction (cGxE) literature by examining how a widely studied polymorphism, the MAOA upstream variable number tandem repeat (MAOA-uVNTR) interacts with distal and proximal stressors to explain variation in crime and delinquency. Prior research findings have revealed that MAOA-uVNTR interacts with single indicators of environmental adversity to explain criminal behavior in general-population and incarcerated samples. Nevertheless, the genetically moderated stress sensitivity hypothesis suggests that increased risk for criminal behavior associated with variation in the MAOA-uVNTR can be best understood in the context of both distal stress during childhood and proximal stress in adulthood. Therefore, we employed Tobit regression analyses to examine a gene–distal–proximal environment (CGxExE) interaction across gender in a sample of university students (n = 267) and with data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; n = 1,294). The results across both sets of analyses demonstrate that variation in the MAOA-uVNTR interacts with distal and proximal stress to lead to increased risk for criminal behavior in males. Although proximal life stress is associated with an increase in crime and delinquency, this effect is more pronounced among MAOA-L allele carriers that have experienced distal stress.

* Additional supporting information can be found in the listing for this article in the Wiley Online Library at <http://onlinelibrary.wiley.com/doi/10.1111/crim.2017.55.issue-3/issuetoc>.

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This study was supported by an Enhancement Grant for Professional Development from the Office of Research and Sponsored Programs at Sam Houston State University. Sponsors were not involved in the development of study methodology or in any aspect of study implementation, data analysis, or report writing. Data were taken from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill and funded by Grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human

In heritability studies, researchers have found that approximately 50 percent of the variance in antisocial behavior is attributable to genetic factors (Rhee and Waldman, 2002). Nevertheless, much work is still needed to uncover the precise developmental pathways through which genetic variation contributes to risk for antisocial behavior, including criminal behavior. One method to capture developmental influences is through the study of candidate gene-by-environment (cGxE) interactions. Such interactions are consistent with the multilevel perspective within developmental psychopathology. This perspective suggests that development of diverse forms of psychopathology, including patterns of pronounced antisocial behavior, is influenced by complex interactions between systems operating at multiple levels, including biological and environmental processes (Cicchetti and Dawson, 2002; Cicchetti and Toth, 2009; Masten, 2007). Consistent with this perspective, most contemporary research into molecular genetic influences on antisocial behavior is aimed at testing these influences in the context of the cGxE model. Within the cGxE model, genetic influences on antisocial behavior result in increased risk for antisocial behavior in interaction with environmental variation. Among research testing the cGxE model is a large group of study results showing that a polymorphism in the monoamine oxidase A (MAOA) gene moderates the association between environmental adversity and increased risk for antisocial behavior, including crime (Byrd and Manuck, 2014; Ficks and Waldman, 2014).

Although the application of the cGxE model has shown that genetic risk for antisocial behavior is often entirely contingent on the co-occurrence of environmental risk, cGxE models may be limited as they mostly rely on a single, unitary measure of the environment. The treatment of environment as a unitary phenomenon is challenged by research results showing the independent contributions of stress at different stages of development (Hammen, Henry, and Daley, 2000) and study findings demonstrating that the neurodevelopmental consequences of early life (distal) stress may lead to increased sensitivity to later (proximal) stressors (Frodl and O'Keane, 2013). Generalized to the study of molecular genetic influences on psychopathology, the results of this research reveal a developmental pathway for psychopathology where genetic variation influences sensitivity to proximal stress through an interaction with distal stress. Essentially, this genetically moderated stress sensitivity hypothesis proposes a gene-by-distal environmental stress by proximal environment stress interaction, cGxE_{ExE} (Homberg and van den Hove, 2012). cGxE_{ExE} interactions have recently been found by two studies in which researchers examined the development of depression (Grabe et al., 2012; Starr et al., 2014). Here, we test the generalizability of the genetically moderated stress sensitization hypothesis to the explanation of crime and delinquency by focusing on MAOA-uVNTR¹ and both distal and proximal stressors in a sample of university students and in a sample from the National Longitudinal Study of Adolescent to Adult Health (Add Health).

Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due to Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>). No direct support was received from Grant P01-HD31921 for this analysis.

1. MAOA-uVNTR = monoamine oxidase A upstream variable number tandem repeat.

LITERATURE REVIEW

MAOA-uVNTR AND CRIMINAL BEHAVIOR

The MAOA-uVNTR is a repeat sequence of 30 base pairs in the monoamine oxidase A gene (Ficks and Waldman, 2014). MAOA catabolizes monoamine neurotransmitters and shows a preference for serotonin and norepinephrine (Cases et al., 1995; Weyler, Hsu, and Breakfield, 1990). The transcriptional efficacy of the MAOA gene varies with the length of the MAOA-uVNTR. Specifically, 2, 3, and 5 repeat sequences are associated with low MAOA functioning (MAOA-L), whereas 3.5 and 4 repeat sequences are associated with high MAOA functioning (MAOA-H; Sabol, Hu, and Hamer, 1998).²

The cGxE model was originally applied to the MAOA-uVNTR by Caspi and colleagues in 2002. In this application, the detrimental effects of childhood maltreatment on later crime and delinquency varied as a function of the MAOA-uVNTR polymorphism. Among those who had experienced childhood maltreatment, MAOA-L carriers showed increased rates of conduct disorder and convictions for violent offenses. Since the seminal work of Caspi et al. (2002), increasingly, more studies have been aimed at testing the association between antisocial behavior and the interaction of MAOA-uVNTR genotype and a variety of measures of adverse environments. The findings of two independent meta-analyses of this literature have recently revealed that the MAOA-uVNTR interacts with environmental adversity to predict variation in antisocial behavior (Byrd and Manuck, 2014; Ficks and Waldman, 2014). The findings from these meta-analyses also show that cGxE interactions have a stronger association with antisocial behavior among males and that the association between the MAOA-uVNTR and antisocial behavior is stronger in the context of measures of adversity that focus on childhood maltreatment (Byrd and Manuck, 2014; Ficks and Waldman, 2014).

Among studies relating the interaction of the MAOA-uVNTR and environmental adversity to antisocial behavior are results showing this interaction is related to criminal behavior in both general-population samples and in incarcerated samples (Armstrong et al., 2014; Åslund et al., 2011; Stetler et al., 2014). These study findings demonstrate that the interaction of the MAOA-uVNTR polymorphism and environmental adversity may contribute to time-stable, between-individual differences in the tendency to commit crime or criminal propensity. Although explanations of criminal behavior increasingly acknowledge some degree of stability in between-individual differences in the tendency to commit crime, they are often silent on the specific mechanisms that contribute to the development of these differences. Results showing that MAOA-uVNTR interacts with environmental adversity to explain criminal behavior begin to specify the developmental pathways that contribute to the cross-contextual risk for criminal behavior, the hallmark of elevated criminal propensity. Research findings on the neurodevelopmental consequences of variation in the MAOA-uVNTR polymorphism reveal that the contribution

2. Some variation in its characterization exists in the literature. For example, Deckert et al. (1999) found the 5 repeat sequence occurred in approximately 1.4 percent of subjects and characterized the allele as high-functioning, whereas Sabol, Hu, and Hamer (1998) found that it occurred in 1.4 percent of subjects and characterized the allele as low-functioning. In the current study, we follow the work of Sabol, Hu, and Hamer (1998) and classify the 5 repeat allele as low-functioning (see also Kim-Cohen et al., 2006; Wakschlag et al., 2010).

of this polymorphism to differences in criminal propensity may be conceptualized as occurring through changes to brain structure and function leading to the development of a hostile aggressive temperament (Buckholtz and Meyer-Lindenberg, 2008). Although the results of extant research on the interaction of the MAOA-uVNTR polymorphism and environmental adversity do begin to illuminate the developmental processes that may contribute to the development of criminal propensity, much of this research has been based on single indicators of environmental adversity without consideration of cumulative effects of proximal and distal stressors.

GENETIC INFLUENCES ON THE CUMULATIVE EFFECTS OF PROXIMAL AND DISTAL STRESS

In the context of the multisystems perspective of developmental psychopathology, the findings from cGxE studies based on the MAOA-uVNTR confirm the role of genetic variation in shaping developmental responses to environmental stressors. Nevertheless, recent research results demonstrate that the unitary conceptualization of environmental adversity in the cGxE model may need elaboration. Specifically, the impact of stressors proximal to the enactment of antisocial behavior may be amplified by neurodevelopmental consequences of prior experiences of stress in distal environments, including those experienced in childhood (Heim et al., 1997, 2000; Heim and Nemeroff, 2001). Exposure to childhood adversity leads to stress system hyperactivity in response to contemporary stressors through increases in hypothalamic–pituitary–adrenal (HPA) axis functioning (Heim and Nemeroff, 2001). The increase in HPA axis functioning likely occurs as a result of the attenuation of a negative feedback mechanism in the stress system resulting in dysregulated stress system activity (Frodl and O’Keane, 2013).³

The increased HPA axis function that occurs as a consequence of early childhood environmental adversity is captured by the stress sensitization hypothesis. This hypothesis holds that early exposure to environmental stressors in childhood can lead to increased response to contemporary stressors later in life and to resultant psychopathology. Examination of the sensitization hypothesis with regard to risk for depression reveals that exposure to childhood abuse increases risk for depression associated with later life stress (Hammen, Henry, and Daley, 2000; Kendler, Kuhn, and Prescott, 2004; McLaughlin et al., 2010, 2012; Slavich, Monroe, and Gotlib, 2011). Recent evidence in this line of research further suggests that stress sensitization may be more important for depression in adolescents than in adults and may be specific to emotional abuse (La Roque, Harkness, and Bagby, 2014). Evidence for stress sensitization is also present in results showing childhood abuse exposure is associated with a greater propensity to develop posttraumatic stress disorder (PTSD) after trauma later in life (Bremner et al., 1993; Breslau et al., 1999; Brewin, Andrews, and Valentine, 2000; but see Breslau, Peterson, and Schultz, 2008; McLaughlin et al., 2010; Shao et al., 2015). The results of additional

3. Stress results in a “hormonal cascade” where a stressor causes the release of corticotropin-releasing hormone (CRH) from the HPA axis, the secretion of adrenocorticotrophic hormone (ACTH), and the release of glucocorticoids including cortisol (Tarullo and Gunnar, 2006). Cortisol potentiates the stress response and poststress attenuation of HPA axis activity through a negative feedback loop that, over time, may damage the hippocampus. Prolonged childhood stress can impair “cortisol-induced feedback inhibition of the HPA axis” (Frodl and O’Keane, 2013: 25).

research have shown that the association between proximal life stress and intimate partner violence was stronger for males with high childhood adversity exposure than that of males who had no such exposure (Roberts et al., 2011). The cumulative effects of distal and proximal stress are also associated with alcohol use and its antecedents (Eames et al., 2014; Kim et al., 2014; but see Young-Wolff, Kendler, and Prescott, 2012). Collectively, these lines of research are consistent with a link between distal stress and problematic behavior through stress sensitization resulting in increased HPA axis responsivity.

There is some reason to believe that stress sensitization may play a role in the development of risk for criminal behavior. Although most work relating HPA axis activity to antisocial behavior has been focused on HPA under- or hypo-arousal as a key factor for the development of antisocial behavior underpinned by callous-unemotional (CU) traits (Hawes, Brennan, and Dadds, 2009; Shirtcliff et al., 2009; Van Goozen et al., 2007), it has recently been suggested that stress system over- or hyper-arousal may contribute to patterns of antisocial behavior typified by heightened emotional responsivity to stress and consequent criminal behavior (Yildirim and Derksen, 2012). A potential link between increased HPA axis activity and crime and delinquency is supported by study findings showing that HPA axis responsivity during stress is positively associated with reactive aggression, conduct disorder, and antisocial behavior, with this association being stronger among females (Böhnke et al., 2010; Geniole, Carré, and McCormick, 2011; Kobak, Zajac, and Levine, 2009; Lopez-Duran et al., 2009; McBurnett et al., 2005; Susman et al., 2010).

Stress sensitization resulting from early childhood adversity and its potential link to criminal behavior may be exacerbated by the MAOA-L genotype. Recent characterizations of the MAOA-uVNTR L alleles have suggested that these alleles may not be “risk” alleles as traditionally conceptualized, but instead they may confer a general sensitivity to environments (Belsky and Beaver, 2011; Belsky et al., 2009). In the case of good or ameliorative environments, the MAOA-uVNTR L alleles may result in improved outcomes, but in the case of adverse environments, these alleles may be associated with significant increases in risk for antisocial behavior through environmental sensitivity (Belsky and Pleuss, 2009). A specific association between MAOA-L and increased responsivity to stress was suggested by Buckholtz and Meyer-Lindenberg (2008). They found that MAOA-L is associated with hostile aggressive temperament underpinned by changes in the structure and function of the amygdala and the medial prefrontal cortex. These changes included increased connectivity between the amygdala and the ventromedial prefrontal cortex and increased amygdala activity during stress (Buckholtz et al., 2008). Given the direct role of the amygdala in stress responses, this finding demonstrates that the MAOA-L alleles may compound sensitivity to stressful environments that result from the neurobiological consequences of exposure to distal stress.

The results of the research reviewed earlier indicate that among those with the MAOA-L genotype, early exposure to environmental adversity may lead to increased sensitivity to proximal stressors and to increased risk for criminal behavior. This finding reveals that the association between the MAOA-L genotype and increased risk for criminal behavior in the context of environmental adversity may be better understood through the genetically moderated stress sensitivity model (cGxExE) rather than through a traditional cGxE model.

THE CURRENT STUDY

In the current work, we apply the cGxExE model to the study of molecular genetic influences on crime and delinquency. Previously, the cGxExE model has been used to study genetic influences on stress sensitization in the development of depression but has not yet been extended to antisocial behavior in general or to crime and delinquency in particular (Grabe et al., 2012; Starr et al., 2014). The current study is focused on the MAOA-uVNTR as a result of its association with increased risk for criminal behavior in cGxE studies and its association with neurobiological change that may compound the sensitizing effects of distal stressors on reactions to proximal stress. A consideration of the cumulative effects of distal and proximal stressors within the genetically moderated stress model may contribute to our understanding of the etiological pathways through which molecular genetic variation influences risk for criminal behavior in interaction with the environment. In light of research results showing gender differences in the association of the MAOA-uVNTR polymorphism with risk for antisocial behavior (Byrd and Manuck, 2014), in the current study, we also disaggregate the sample under consideration by gender and test the genetically moderated stress sensitization hypothesis among males and females separately.⁴ Furthermore, to ensure findings were not a statistical artifact or as a result of sample-specific characteristics, analyses were conducted with two distinct samples. The first set of analyses was based on original data collection conducted at a university in a southern state. The second set of analyses was conducted with data gathered as part of the National Longitudinal Study of Adolescent to Adult Health (Add Health).

METHOD

PARTICIPANTS

SAMPLE I

Data were collected from 521 volunteers attending undergraduate courses in criminal justice at a university in a southern state.⁵ After providing informed consent, participants completed a self-report survey and allowed researchers to collect a buccal swab for DNA analysis. The sample was 15.3 percent African American, 27.5 percent Hispanic, 47.8 percent Caucasian, and 6.5 percent Other, and it was approximately evenly split between males (50 percent) and females (50 percent). The mean age of the sample was 21.35 [standard deviation (SD) = 2.96]. Research findings demonstrate that analyses should be replicated across demographic groups.⁶ To support this replication, the

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4. In their meta-analysis, Byrd and Manuck (2014) initially found the MAOA-uVNTR H allele indicated risk for antisocial behavior among females. Nevertheless, this association was driven by the results of two influential studies and the association between the MAOA-uVNTR and antisocial behavior was attenuated after these studies were deleted from their meta-analysis.
 5. The protocol for data collection was approved by the university's internal review board.
 6. The necessity for replication across demographic groups is demonstrated by the racial stratification of risk alleles and by variation across race in the influence of the MAOA-uVNTR on behavior. For example, Stetler et al. (2014) found the association between the MAOA-uVNTR and criminal behavior was stronger among Caucasians than among African Americans. As racial stratification of MAOA functioning may potentially influence the results of the current study, all analyses were replicated with the samples restricted to Hispanics and Caucasians.

analytical sample was restricted to the two largest racial/ethnic groups, Caucasians and Hispanics.⁷

SAMPLE 2

Data from the Add Health were used to replicate analyses based on the university sample. The Add Health is a nationally representative study that began data collection in the United States in 1994 when participating adolescents were in grades 7 through 12. Since then, four waves of data collection have been conducted with the most recent wave occurring in 2008–2009. During the wave III interviews, 2,611 participants from the sibling pairs subsample provided their DNA for genotyping. More detailed descriptions of the Add Health research design and data collection procedures are presented elsewhere (see Harris, 2009; Kelley and Peterson, 1997).

To replicate the analyses from the university sample the best, data from the Add Health were restricted to wave III when participants were between 18 and 27 years of age and included only White and Hispanic participants. The mean age of participants was 21.93 (SD = 1.69) with 81.7 percent reporting being White and 62.6 percent reporting being male.

MEASURES

DISTAL ENVIRONMENTAL STRESS

Sample 1. Distal environmental stress was measured with two items indexing self-reported exposure to parental verbal and physical abuse. Respondents were asked to indicate how well the statements “my parents were verbally abusive” and “my parents were physically abusive” described their parents before the participants were 18 years old. Responses were recorded on a Likert scale (1 = describes them well, 2 = somewhat describes them, 3 = does not really describe them, 4 = does not describe them at all). Total abuse was then dichotomized as 0: experienced neither abuse type (57.8 percent) and as 1: experienced at least one type of abuse (42.2 percent). Sex-specific means and standard deviations for this measure and the others included in the study are presented in table 1. There was no significant difference in distal stress reported across gender ($t = .32; p > .05$).

Sample 2. Distal stress was measured by asking participants the following question: “By the time you started the sixth grade, how often had your parents or other adult caregivers slapped, hit, or kicked you?” Response categories were collapsed into 0 = never to 1 = at least once. Approximately 29 percent of the sample reported having experienced some form of physical abuse as a child.

7. The results were the same for the combined Hispanic and Caucasian samples and in the group-specific samples. We were statistically unable to replicate our analyses with the university sample restricted to African Americans as a result of the low samples size (male $n = 51$; female $n = 35$). As we could not ensure the generalizability of our results to this group, they were omitted from the analyses.

Table 1. Descriptive Statistics of the University and Add Health Samples

Variable	University Sample					Add Health				
	Mean (SD)	SD	Range	<i>t</i> Test	<i>p</i>	Mean (SD)	SD	Range	<i>t</i> Test	<i>p</i>
Distal Stress	42.2%			.32		28.8%			.11	
Male (yes)	41.5%					28.9%				
Female (yes)	43.4%					28.6%				
Proximal Stress	2.46	2.09	0–10.0	2.24	*	.60	1.04	0–8.0	–.49	
Male	2.26	1.99				.59	1.10			
Female	2.85	2.24				.62	.93			
Romantic Partner/Ever Married	69.0%			2.27	*	19.6%			–5.77	***
Male (yes)	64.5%					14.9%				
Female (yes)	77.6%					27.6%				
Criminal Behavior	.17	.25	0–1.4	–2.16	*	.06	.14	0–1.4	7.9	***
Male	.20	.28				.08	.17			
Female	.13	.19				.02	.06			

ABBREVIATIONS: MAOA-L = low-functioning MAOA; SD = standard deviation.

p* < .05; **p* < .001.

PROXIMAL ENVIRONMENTAL STRESS

Sample 1. Proximal environmental stress was measured with the List of Threatening Experiences Questionnaire (LTE; Brugha and Cragg, 1990). The LTE asks participants which of 15 items they had experienced in the past 12 months and includes questions concerning various life stressors related to familial, financial, and health problems (see appendix A in the online supporting information for a full list of items⁸). The index was calculated as the sum of all responses (1 = yes, 0 = no) where higher scores reflect more stress (mean = 2.46; SD = 2.09; Cronbach’s alpha = .99). Among males, 20.7 percent reported no proximal life stress while 11.2 percent of females reported no proximal life stress. Female participants reported significantly more proximal stress in the past year (*t* = 2.23; *p* < .05).

Sample 2. Proximal stress was measured with 14 items derived from the Add Health data. Items were selected based on similarity to questions from the List of Threatening Experiences Questionnaire used with the University sample (see appendix A for a complete list of items). Participants were asked whether they had experienced various personal-, financial-, and health-related stressors in the past 12 months (1 = yes, 0 = no). Proximal stress was calculated as the sum of the 14 items where higher scores reflected having experienced greater amounts of stressors (Cronbach’s alpha = .52).⁹ There were no significant differences in levels of proximal stress between males and females (*t* = –.49, *p* > .05).

8. Additional supporting information can be found in the listing for this article in the Wiley Online Library at <http://onlinelibrary.wiley.com/doi/10.1111/crim.2017.55.issue-3/issuetoc>.

9. We recognize that the value of the Cronbach’s alpha for this measure is minimal. Items for this measure were selected to create a measure of proximal stress in sample 1 that was as close as possible to the proximal stress measure for sample 2. To maximize the overlap between the two measures, we retained all items in the sample 2 proximal stress measure despite this low Cronbach’s alpha value.

CRIME AND DELINQUENCY

Sample 1. Crime and delinquency were measured via 18 items asking participants how often they had engaged in a variety of criminal and delinquent behaviors ranging from minor crime and delinquency such as “exceeded the speed limit by 10–20 mph” to serious crime and delinquency such as “attacked someone with the idea of seriously hurting or killing them.” As a result of the low seriousness and high frequency of reporting, three items were omitted from the scale (see appendix B in the on-line supporting information for specific items removed). Potential responses ranged from 0 = never to 8 = 2–3 times a day (for a full list of items and responses, see appendix B). The crime and delinquency scale was calculated as the average of all reported responses to the 15 items. Of the resulting index, the mean score was .17 with a standard deviation of .25 (Cronbach’s $\alpha = .94$). Male participants reported significantly greater crime and delinquency than did female participants ($t = -2.16$, $p < .05$).

Sample 2. Criminal behavior was measured by asking respondents their level of involvement in various nonviolent (e.g., “deliberately damaged property that didn’t belong to you”) and violent (e.g., “used a weapon in a fight”) forms of criminal activities (see appendix B for a complete list of items and responses). The crime and delinquency measure was created by taking the average of the 14 items with higher scores reflecting greater criminal involvement (Cronbach’s $\alpha = .64$). Similar to the university sample, males reported significantly more criminal behavior compared with females ($t = 7.96$, $p < .001$).

CONTROL VARIABLES

Sample 1. To ensure that the observed effects were not driven by spurious associations with other risk factors, analyses also included measures of constructs featured in socioenvironmental explanations of criminal behavior, including age, race/ethnicity, and romantic partners. The presence of a romantic partner was measured with a dichotomous variable indicating involvement with any romantic partner in the past year (0 = no, 1 = yes). Age was measured as the number of years at the time of survey administration. Race/ethnicity was coded dichotomously (0 = Caucasian, 1 = Hispanic).

Sample 2. Age, race/ethnicity, and intimate relationships were also included as control variables by using the Add Health data. Age was measured as a continuous variable in years, race/ethnicity was dichotomized into 0 (Caucasian) and 1 (Hispanic), and intimate relationships was captured by asking respondents whether they had ever been married (0 = no, 1 = yes).

MAOA

Sample 1. Participants were classified into high- and low-functioning MAOA-uVNTR allele groups based on previous research (see Byrd and Manuck, 2014). This resulted in 29.8 percent ($n = 57$) of male participants classified as low-functioning (2R, 3R, and 5R) and 70.2 percent ($n = 134$) of male participants classified as high-functioning (3.5R and 4R). Because MAOA is located on the X chromosome, and males carry only one copy of

the gene, males were classified based on their hemizygous genotype.¹⁰ Females, carrying two X-chromosomes, carry 2 alleles and thus were categorized only if their two alleles were of the same functional category (e.g., 3.5R/4R would be classified as MAOA-H, whereas a 3R/4R would be excluded; see Widom and Brzustowicz, 2006). As such, 77 female participants in the analytic sample were excluded from analysis. Of the remaining female participants, 28.3 percent ($n = 28$) were classified as low-functioning and 71.7 percent ($n = 71$) were classified as high-functioning (see table 2 for allelic distribution). Genotyping procedures are detailed in appendix C in the online supporting information.¹¹

Sample 2. Similar to the university sample, we followed Byrd and Manuck's (2014) approach of measuring MAOA by dichotomizing the variable into low- (2R, 3R, and 5R) and high- (3.5R and 4R) functioning groups. For males, 38.7 percent ($n = 326$) and 61.3 percent ($n = 516$) were classified as low- and high- functioning, respectively. Similar to the university sample, females with cross-functional alleles (e.g., 3.5R/5R) were excluded from the analytical sample ($n = 403$; see Widom and Brzustowicz, 2006). This resulted in 26.8 percent ($n = 135$) of females being classified as low-functioning and 73.2 percent ($n = 369$) as high-functioning. Allelic distributions are presented in table 2.

STATISTICAL ANALYSES

The extent to which MAOA-uVNTR interacts with distal and proximal stress to predict crime was tested with Tobit regression models.¹² The Tobit regression model is appropriate for endogenous variables with truncated distributions such as the crime and delinquency measure under consideration here (Long, 1997). For example, in the sample 1 data, 41.5 percent of females reported no crime or delinquency, whereas 33.0 percent of males reported no crime or delinquency. Similarly, in the sample 2 data, 790 (84.6 percent) of females reported no crime or delinquency, whereas 574 (63.7 percent) of males reported no crime or delinquency. To account for positive skew in the dependent variable distribution, the natural log of crime and delinquency was analyzed in addition to the use of robust standard errors (see Osgood, Finken, and McMorris, 2002). Prior to the estimation of regression models, the analysis began by testing the gene–proximal-stress and gene–distal-stress correlations. These correlations were estimated as a result of the possibility that environments are a function of genotype rather than an independent predictor

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10. In the female sample, the MAOA-uVNTR significantly deviated from Hardy Weinberg equilibrium, which tests the observed allele frequencies against that of a population without evolutionary pressures (likelihood ratio $\chi^2 = 21.21$, $p = .02$). This is likely as a result of the low frequency of the 3.5 and 5 repeat sequences.
 11. As a result of mixed findings concerning the functionality of the rarer MAOA-uVNTR variants (2R, 3.5R, and 5R), analyses were replicated restricting the samples to those 3R and 4R carriers. Three-way interactions remained significant for males (sample 1: $b = .11$, $SE = .04$, $p < .01$; sample 2: $b = .09$, $SE = .04$, $p < .05$) and not significant for females (sample 1: $b = .12$, $SE = .08$, $p > .05$; sample 2: $b = .01$, $SE = .06$, $p > .05$). These results are available upon request.
 12. To ensure that results were not the product of model choice, the results from Tobit models were compared with the results from negative binomial models. They were substantively similar with two minor exceptions: 1) The negative binomial models found a significant ExE association among males in model 1 of sample 2 that did not exist in Tobit models; and 2) the significance of the gxExE coefficient in model 2 of sample 1 was diminished to $p = .05$. The results are available upon request.

Table 2. MAOA Allele Distributions in University and Add Health Samples

Variable	University Sample					Add Health Sample							
	MAOA-L		MAOA-H			2R	MAOA-L			MAOA-H			
	3R	5R	3.5R	4R	3.5R/4R		2R/3R	3R	3R/5R	5R	3.5R	3.5R/4R	
Male													
Distal stress	23	0	0	57	—	—	—	80	—	7	4	152	—
No distal stress	34	2	1	74	—	2	—	229	—	8	4	356	—
Stress (mean[SD])	2.43 (1.79)			2.19 (2.07)				.58 (1.07)				.59 (1.11)	
Female													
Distal stress	12	0	0	30	1	—	1	44	1	—	—	93	5
No distal stress	16	0	0	40	0	—	1	81	7	—	—	264	7
Stress (mean[SD])	2.64 (2.61)			2.93 (2.08)				.59 (.85)				.63 (.96)	

NOTES: University sample: MAOA-H reporting no proximal stress ($n = 38, 19.2\%$); MAOA-L reporting no proximal stress ($n = 11, 13.1\%$); no significant MAOA-proximal stress ($r = .04, p = .51$) or MAOA-distal stress correlation was detected ($r = -.02, p = .68$). Add Health sample: MAOA-H reporting no proximal stress ($n = 545, 61.6\%$); MAOA-L reporting no proximal stress ($n = 285, 61.8\%$); no significant MAOA-proximal stress ($r = -.01, p = .67$) or MAOA-distal stress correlation was detected ($r = .002, p = .95$).

of the dependent variable of interest. The lack of statistical significance indicated that the MAOA-uVNTR genotype did not predict exposure to proximal or distal stress (sample 1: MAOA-distal: $r = -.02$, $p = .68$, MAOA-proximal: $r = .04$, $p = .51$; sample 2: MAOA-distal: $r = .00$, $p = .95$, MAOA-proximal: $r = -.01$, $p = .67$). This finding demonstrates that any association between cGxE interactions or cGxExE interactions and crime and delinquency is not the result of gene–environment correlation (Scarr and McCartney, 1983).

Initial regression models were used to test the stress sensitization hypothesis by exploring the interactive effect of distal and proximal stress on crime and delinquency. These models included the direct influences of distal and proximal stress terms and the interaction between distal and proximal stress. Next, the genetically moderated stress sensitivity model was tested. For this test, interaction terms representing the interactions between the MAOA-uVNTR polymorphism and the distal and proximal stress measures were added to the regression model. Interaction terms included two cGxE interactions and the cGxExE interaction. The cGxE interaction terms included the interaction between MAOA genotype and distal stress, as well as the interaction between MAOA genotype and proximal stress. To avoid multicollinearity in models, distal and proximal stress were mean-centered, resulting in no tolerance values below the common .10 threshold (Hair et al., 2006). Finally, the models tested the cGxExE interaction while controlling for the effects of age, race/ethnicity, and romantic partners. Each model was estimated in sex-specific subsamples.

RESULTS

The results for gender-specific Tobit models in sample 1 are presented in table 3. In model 1, we tested the interactive effects of distal and proximal stress without genetic moderation. In these models, proximal life stress was directly associated with crime and delinquency in both males [$b = .06$, standard error (SE) = .01, $p < .001$] and females ($b = .03$, SE = .01, $p < .001$). Distal stress was also positively associated with crime and delinquency among females ($b = .10$, SE = .05, $p < .05$). The interaction between distal and proximal stress was not associated with crime and delinquency among males ($b = -.02$, SE = .02, $p > .05$) or females ($b = -.00$, SE = .02, $p > .05$). These results fail to confirm the stress sensitivity hypothesis, absent consideration of genotype.

The results of replication analyses in the Add Health wave III data can be observed in table 4. Again, proximal stress was significantly associated with an increase in crime in delinquency in both males ($b = .07$, SE = .01, $p < .001$) and females ($b = .06$, SE = .01, $p < .001$). In this sample, distal stress was also associated with criminal behavior among both males ($b = .09$, SE = .02, $p < .001$) and females ($b = .06$, SE = .03, $p < .05$). An association between distal stress and criminal behavior among males was not present in the earlier results from the university sample. In sample 2, no significant distal $\times$ proximal stress association with offending was found (male: $b = -.01$, SE = .02, $p > .05$; female: $b = -.04$, SE = .03, $p > .05$), which mirrors the results from sample 1.

In model 2, we tested the genetically moderated stress sensitivity hypothesis. We explored the possibility that the effects of distal and proximal stress both directly and in interaction with each other are moderated by MAOA-uVNTR genotype. In sample 1 (table 3), no two-way interactions between the MAOA-uVNTR and distal ($b = -.01$, SE = .10, $p > .05$) or proximal stress ($b = .02$, SE = .02, $p > .05$) were found. The results show that the cGxExE interaction among the MAOA-uVNTR, distal stress, and

Table 3. Tobit Regression Models by Sex—University Sample

Variable	Males									Females								
	Model 1			Model 2			Model 3			Model 1			Model 2			Model 3		
	<i>b</i>	SE	<i>p</i>	<i>b</i>	SE	<i>p</i>	<i>b</i>	SE	<i>P</i>	<i>b</i>	SE	<i>p</i>	<i>b</i>	SE	<i>p</i>	<i>b</i>	SE	<i>p</i>
Age	-.01	.01		-.01	.00		-.01	.00		-.01	.01		-.01	.01		-.00	.01	
Hispanic	.00	.04		.02	.04		-.00	.04		.00	.04		.02	.04		.02	.04	
Distal Stress	.06	.04		.06	.04		.06	.04		.10	.05	*	.14	.06	*	.14	.06	*
Proximal Stress	.06	.01	***	.05	.01	***	.05	.01	***	.03	.01	***	.04	.01	***	.03	.01	***
Proximal Stress × Distal Stress	-.02	.02		-.05	.02	**	-.05	.02	*	-.00	.02		.01	.02		.02	.02	
MAOA-L				-.00	.05		-.02	.05					.05	.07		.04	.08	
MAOA-L × Proximal Stress				.01	.02		.02	.02					.05	.04		.04	.04	
MAOA-L × Distal Stress				-.01	.10		-.06	.11					-.26	.15		-.24	.15	
MAOA-L × Proximal Stress × Distal Stress				.11	.04	**	.13	.04	**				-.12	.08		-.11	.08	
Romantic Relationships							.07	.04								.08	.05	
<i>R</i> ²		.37			.44			.47			.47			.59			.63	
<i>N</i>		175			175			173			92			92			91	

ABBREVIATIONS: MAOA-L = low-functioning MAOA; SE = standard error.

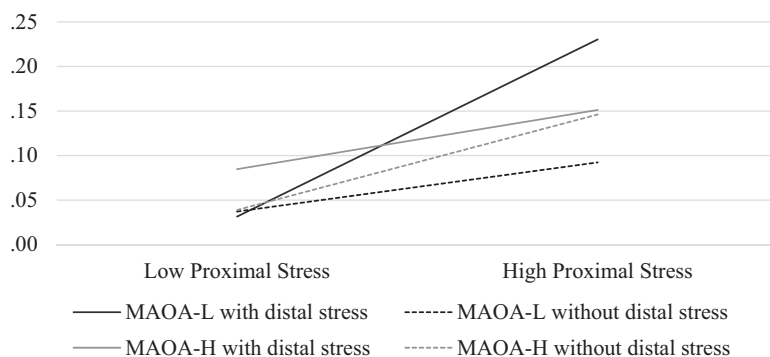
p* < .05; *p* < .01; ****p* < .001.

Table 4. Tobit Regression Models by Sex—Add Health Wave III

Variable	Males									Females								
	Model 1			Model 2			Model 3			Model 1			Model 2			Model 3		
	b	SE	p	b	SE	p	b	SE	p	b	SE	p	b	SE	p	b	SE	p
Age	-.03	.01	***	-.03	.01	***	-.02	.01	***	-.03	.01	***	-.03	.01	***	-.03	.01	***
Hispanic	-.06	.03	*	-.06	.03	*	-.05	.02	*	.05	.03		.05	.03		.05	.03	
Distal Stress	.09	.02	***	.08	.03	**	.08	.03	**	.06	.03	*	.02	.04		.02	.04	
Proximal Stress	.07	.01	***	.08	.02	***	.08	.02	***	.06	.01	***	.06	.02	***	.06	.02	***
Proximal Stress × Distal Stress	-.01	.02		-.04	.02		-.03	.02		-.04	.03		-.04	.03		-.04	.03	
MAOA-L				-.03	.02		-.03	.02					.05	.03		.05	.03	
MAOA-L × Proximal Stress				-.01	.02		-.01	.02					.02	.03		.01	.03	
MAOA-L × Distal Stress				-.00	.04		.00	.04					.09	.06		.09	.06	
MAOA-L × Proximal Stress × Distal Stress				.08	.04	*	.09	.03	*				.00	.06		.01	.06	
Romantic Relationships							-.16	.03	***							-.03	.03	
R ²	.22			.23			.27			.19			.22			.22		
N	809			809			808			485			485			484		

ABBREVIATIONS: MAOA-L = low-functioning MAOA; SE = standard error.
* $p < .05$; ** $p < .01$; *** $p < .001$.

Figure 1. Association of Proximal Stress on Crime and Delinquency Stratified by MAOA and Distal Stress Among Males (Before Log Transformation)



proximal stress was significantly associated with crime and delinquency among males ($b = .11$, $SE = .04$, $p < .01$) but not among females ($b = -.12$, $SE = .08$, $p > .05$). This result was replicated in sample 2 (table 4). As in sample 1, no two-way interactions between the MAOA-uVNTR and distal ($b = -.00$, $SE = .04$, $p > .05$) or proximal stress ($b = -.01$, $SE = .02$, $p > .05$) were found. A significant cGxE interaction was found among males ($b = .08$, $SE = .04$, $p < .05$) but not among females ($b = .00$, $SE = .06$, $p > .05$).

Next, through the models, we tested the predictive validity of the cGxE interaction by adding measures controlling for the presence of romantic partners. The same pattern of results emerged wherein the cGxE was associated with crime and delinquency in the male subsamples (sample 1: $b = .13$, $SE = .04$, $p < .01$; sample 2: $b = .09$, $SE = .03$, $p < .05$) but not in the female subsamples (sample 1: $b = -.11$, $SE = .08$, $p > .05$; sample 2: $b = .01$, $SE = .06$, $p > .05$). This finding suggests that the interactive effects of distal and proximal stress play a role in the etiology of crime and delinquency among males with the MAOA-L genotype.

The association among MAOA-uVNTR genotype, distal stress, and proximal stress in sample 2 is graphed in figures 1 and 2.¹³ Figure 1 shows that among males, even though exposure to proximal stress increases crime and delinquency regardless of distal stress exposure or genotype, those with the MAOA-L genotype and distal stress have a more pronounced increase in crime and delinquency than do those with MAOA-L carriers without distal stress exposure. This effect is not observed in those with the MAOA-H genotype. Among the group with the MAOA-H genotype, exposure to distal stress did not increase the association between proximal stress and crime and delinquency. Figure 2 shows that among females, there is no genetically moderated stress sensitization effect as suggested by regression models.

13. Figures and stratified analyses are limited to sample 2 as the number of participants in the sample was much larger, allowing for more reliable point estimates and regression coefficients (study 1: males with abuse $n = 72$, males without abuse $n = 103$, females with abuse $n = 39$, females without abuse $n = 53$). The results from sample 1 were substantively identical and are available upon request.

Figure 2. Association of Proximal Stress on Crime and Delinquency Stratified by MAOA and Distal Stress Exposure Among Females (Before Log Transformation)

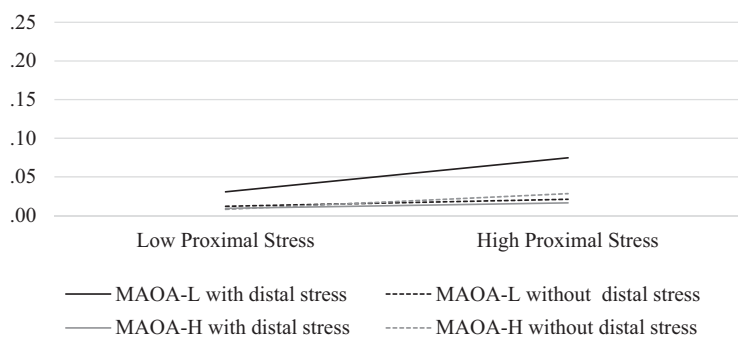


Table 5. Tobit Regression Stratified Sample Models—Add Health Wave III

Variable	Males						Females					
	Abused			Not Abused			Abused			Not Abused		
	<i>b</i>	SE	<i>p</i>	<i>b</i>	SE	<i>p</i>	<i>b</i>	SE	<i>p</i>	<i>b</i>	SE	<i>p</i>
Age	−.02	.01	*	−.03	.01	***	−.02	.01	†	−.03	.01	**
Hispanic	−.03	.04		−.08	.04	*	.02	.05		.07	.04	†
Stress	.05	.01	***	.09	.02	***	.02	.02		.07	.02	**
MAOA-L	−.03	.04		−.03	.03		.10	.04	*	.02	.04	
MAOA-L × Stress	.05	.02	*	−.04	.03		.02	.03		.02	.05	
R ²		.31			.15			.20			.21	
N		229			580			137			348	

ABBREVIATIONS: MAOA-L = low-functioning MAOA; SE = standard error.
†*p* < .10; **p* < .05; ***p* < .01; ****p* < .001.

To illustrate further the association between MAOA genotype and proximal stress in the context of prior stress, sample 2 was stratified by sex and distal stress exposure and the interactive effects of genotype and proximal stress were tested within sex/distal stress exposure groups. The results presented in table 5 indicate that the MAOA–proximal stress interaction is present only among males that had been exposed to distal stress (*b* = .05, SE = .02, *p* < .05). As such, only among males that had experienced early stress did the presence of a low-functioning MAOA alleles lead to a stronger, positive association between proximal stress and crime and delinquency. This finding is supportive of a genetically moderated stress sensitization mechanism, where early exposure to stress leads to a stronger association between proximal stress and crime and delinquency, but only among males with the MAOA-L genotype.

DISCUSSION

The results of the current work suggest that future inquiry into the association between the interactive effects of proximal and distal stress on criminal behavior should

emphasize both genotype and gender differences. Specifically, these results show that among males with the MAOA-L genotype, exposure to distal stress strengthens the association between proximal stress and criminal behavior. This result is consistent with an emerging conceptualization of the MAOA-uVNTR polymorphism as a marker for environmental sensitivity rather than as a “risk” allele that simply conveys an increased likelihood of crime and delinquency. In the case of the current results, the environmental sensitivity imbued by the MAOA-L genotype compounds the interaction between distal and proximal stress leading to increased risk for criminal behavior. This result extends the findings of a large body of research that show that the interaction between the MAOA-uVNTR polymorphism and environmental adversity is associated with antisocial behavior. The current results also extend the outcomes of a smaller group of studies showing that the interaction between MAOA-uVNTR genotype and environmental adversity is relevant to the explanation of criminal behavior. We built on this prior research by advancing the conceptualization of the environment offered by the traditional cGxE model to a cGxE_E model consistent with genetically moderated stress sensitivity. The cGxE_E (MAOA-L genotype $\times$ distal stress $\times$ proximal stress) found here challenges the unitary conceptualization of the environment offered in the traditional cGxE model and suggests that genetic variation may moderate the cumulative influence of distal and proximal stress on criminal behavior.

The current results can be framed in the context of the multisystems perspective in developmental psychopathology. This perspective incorporates aspects of biology and the environment and their interactive effects on the development of psychopathology (Cicchetti and Dawson, 2002; Cicchetti and Blender, 2004; Cicchetti and Toth, 2009; Masten, 2007). The dynamics implied by the multisystems perspective, particularly the interaction between biology and the environment, are absent from many criminological theories but are well captured by Moffitt’s (1993) dual taxonomy. Within this taxonomy, differences in life-course–persistent antisocial behaviors are driven by differences in executive function that result from exposure to an adverse rearing context along with neuropsychological risk. Here we find that a marker of neuropsychological risk (low-functioning MAOA-uVNTR genotype) interacts with distal abuse and proximal stress to predict crime and delinquency in males. That is, the environmental sensitivity to later life stress is most pronounced among those with both traumatic early life experiences and with biological risk factors, thus, leading to a greater likelihood of criminal and delinquent behavior. This finding supports the proposition that the interaction of biological and environmental risk is an important predictor of crime and delinquency, but given the sample used in this study, the current results also demonstrate that this interaction may be relevant for both life-course–persistent antisocial behavior and for patterns of antisocial behavior that are less pronounced, including patterns of antisocial behavior such as those shown by Moffitt’s adolescence-limited antisocial behavior group.

In the context of prior research, the current results offer some suggestion that a hostile aggressive temperament plays a role in explaining stable between-individual differences in the tendency to commit crime. In an assessment of the neurodevelopmental consequences of the MAOA-uVNTR polymorphism, Buckholtz and Meyer-Lindenberg (2008) have argued that the changes in brain structure and function associated with the MAOA-L genotype result in increased risk for antisocial behavior through the development of a hostile aggressive temperament. In the case of the cGxE_E model, this is

potentially compounded by the stress sensitization that occurs when distal stress leads to neurobiological changes that in turn lead to increased arousal attendant to proximal stressors. Applied to the idea of criminal propensity, the unique genetic and neurobiological antecedents of a hostile reactive temperament suggest that such a temperament is an independent trait that makes a unique contribution to stable between-individual differences in the tendency to engage in acts of crime and delinquency. The independence of a hostile reactive temperament is contrary to both narrowly construed definitions of criminal propensity (see Hirschi, 2004) and to broad conceptualizations that subsume differences in hostile reactivity along with other traits including risk seeking and impulsivity in a single unitary criminal propensity (see Gottfredson and Hirschi, 1990).

If a hostile reactive temperament is central to the influence of the MAOA-uVNTR on the relationship between environmental stressors and crime and delinquency, then the current results are also consistent with general strain theory's emphasis on trait anger in the etiology of criminal and delinquent behavior (see Agnew, 1992, 2001). General strain theory holds that the association between strain and criminal and antisocial behavior is mediated by emotional reactivity (anger) to strain. It also allows that trait anger—the tendency to react to strains with hostile negative emotional arousal—may lead to a general increased risk for criminal behavior. The findings of the current study reveal that, among males, reactivity to proximal stress is most pronounced among those with both the MAOA-L genotype and the prior exposure to distal stress. This indicates that the association between strain and crime and delinquency may be strongest among those with both the MAOA-L genotype and the exposure to distal stress, in part through increases in trait anger.

Gender differences in the current study parallel differences found in tests of general strain theory. Tests of general strain theory find that although anger follows strain for both males and females (Piquero and Sealock, 2004; Sigfusdottir and Silver, 2009), the likelihood that these strains lead to antisocial behavior is increased among males (Agnew, 2001; Broidy and Agnew, 1997; Broidy, 2001; Sigfusdottir, Farkas, and Silver, 2004). Our results indicate that some gender differences in the association between strain and antisocial behavior may result from increased stress sensitization among males with the MAOA-L genotype. Differences in the association between stressors and criminal behavior across genotype and gender also suggest that a consideration of genetic and gender differences may help to specify the association between strain and crime and delinquency further.

The results presented in tables 3 and 4 show that a consideration of genetic influences can be critical for tests of stress sensitization. Developed primarily within the study of depression, the stress sensitization hypothesis suggests that the neurobiological consequences of distal stressors experienced during childhood result in increased sensitivity to proximal stressors in the immediate environment (Heim et al., 1997, 2000; Heim and Nemeroff, 2001). Nevertheless, in both the university sample and in the Add Health data, the initial interaction of distal and proximal stress in model 1 was not statistically significant, whereas the interaction of MAOA-L genotype with distal and proximal stress in models 2 and 3 was. Thus, prior to a consideration of genotype, we did not find evidence for stress sensitization. It should be recognized that the importance of genotype for stress sensitization may be unique to a consideration of the effects of stress sensitization on risk for criminal behavior.

The neurobiological mechanisms that underpin the association between crime and delinquency and the genetically moderated interactive influence of distal and proximal stress are somewhat speculative. In previous research, it has been reported that distal stress is associated with increased HPA axis reactivity as measured by cortisol levels (Guerry and Hastings, 2011; Southwick, Vythilingam, and Charney, 2005), and other work has also reported an association between increased HPA activity and antisocial behavior (Böhnke et al., 2010; Geniole, Carré, and McCormick, 2011; Kobak, Zajac, and Levine, 2009; Lopez-Duran et al., 2009; McBurnett et al., 2005; Susman et al., 2010). Nevertheless, to date, no single study has resulted in finding a link between distal stress and changes in antisocial behavior through increased HPA axis reactivity. Nonetheless it is possible that changes to HPA axis function caused by stress sensitivity are compounded by changes in brain structure and function among MAOA-L carriers. Buckholtz et al. (2008) found the MAOA-L genotype was associated with increased amygdala reactivity and increased connectivity between the prefrontal cortex and the amygdala. Amygdala reactivity is positively associated with stress system responses including activity in the HPA axis (Herman and Cullinan, 1997; Herman et al., 2005). Thus, the MAOA-uVNTR L genotype may compound increased stress sensitization driven by the neurobiological consequences of exposure to distal stress.

Conclusions based on the current results are shaped by the methodological features of the current work and by the broader context of candidate gene-by-environment (cGxE) research. The results of cGxE studies have been sharply criticized for their failure to replicate (Duncan and Keller, 2011). Yet, even the strongest of these critics, rather than suggesting that the cGxE methodology should be abandoned, instead calls for the implementation of more rigorous standards for research employing the cGxE methodology (see for example Dick et al., 2015). Foremost among these standards is the replication of novel findings in more than one data set. In the current work, we meet this standard by replicating findings from a proprietary sample in an independent sample using a vastly different sampling approach. We also meet additional criteria for sound cGxE research, including the selection of variation within a gene with known phenotypical consequences. In this case, the impact of the MAOA-uVNTR on neurological development is well understood. Furthermore, in the current study, we use distinct but overlapping measures of environmental adversity including distal and proximal stress. Although aspects of our methodology support its rigor, we acknowledge that criticism of cGxE research is persuasive and we suggest the conclusions regarding the robustness and generalizability of the current result await further replication.

Specific considerations regarding the methodological features of the current study include the measurement of stress and level of criminal activity in the respective samples. The results of a recent meta-analysis showed that interactions between MAOA-uVNTR genotype and measures of adversity specific to childhood maltreatment have a stronger association with antisocial behavior than do interactions between the MAOA-uVNTR and general adversity measures, ranging from sociodemographic characteristics to peer behavior, to measures of parenting style (Byrd and Manuck, 2014). In the current work, distal stress or adversity is measured as physical or verbal abuse before the age of 18 in sample 1 and physical abuse before the sixth grade in sample 2. Proximal stress is measured with the List of Threatening Experiences Questionnaire (LTE), a scale

indexing exposure to 15 different stressors in the past year in sample 1 and with a close approximation in sample 2. While the LTE has established psychometric properties the ad hoc measure of proximal stress derived from the Add Health data had a low Cronbach's alpha (.52) and our measure is limited in that it lacks specificity regarding the timing of abuse. Lack of specificity regarding the time at which abuse occurred is somewhat problematic as the impact of childhood adversity on neurological differences is influenced by the age at which adversity is experienced (Heim and Binder, 2012; Riem et al., 2015). Results of studies in this area vary somewhat but agree that environmental adversity occurring before adolescence has a more pronounced effect on neurobiological development associated with stress regulation (Carpenter et al., 2004, 2009; Heim et al., 2008; Riem et al., 2015). This suggests that measure of adversity specific to abuse occurring before adolescence may capture instances of maltreatment that are more likely to be associated with differences in stress system reactivity.

The weight given the current results is conditioned by the low rates of criminal behavior among both samples used in the current work. There is some evidence that the developmental processes leading to antisocial behavior are different across groups with different levels of risk for antisocial behavior. Nevertheless, it should be noted that some models specifying different developmental antecedents for different trajectories of antisocial behavior suggest that biological risk factors are more important among groups with more pronounced patterns of antisocial behavior.

CONCLUSION

Through the current work, we further our understanding of the developmental etiology of crime and delinquency by generalizing the genetically moderated stress sensitization hypothesis to the explanation of criminal and delinquent behavior. The genetically moderated stress sensitization suggests that genetic variation may convey increased risk for changes in stress system responsivity that result from distal stress. Generalized to the explanation of criminal behavior, increased stress system responsivity potentially results in increased emotional arousal from proximal stress and in increased risk for acts of crime and delinquency resulting from a hostile aggressive temperament. These results support perspectives emphasizing the interaction of biology and the environment in the explanation of criminal behavior and perspectives suggesting that a tendency toward anger is an important part of the explanation of time-stable, between-individual differences in the tendency to commit crime. Although these results contribute to our knowledge regarding the development of risk for crime and delinquency, they do leave key questions unanswered. These questions may be addressed by future work that is aimed at building on the current results by exploring generalizability to samples at high risk for criminal behavior. Further work may also be used to extend the current results with measures of distal stressors that specify the time period in which abuse occurred and with measures of traits that likely mediate the association between cGxExE interactions and acts of crime and delinquency.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher's web site:

Appendix A. List of Threatening Experiences Questionnaire and Proximal Stress Survey Items

Appendix B. Criminal Behavior Survey Items

Appendix C. University Sample