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Association of low-activity *MAOA* allelic variants with violent crime in incarcerated offenders

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Abstract

The main enzyme for serotonin degradation, monoamine oxidase (MAO) A, has recently emerged as a key biological factor in the predisposition to impulsive aggression. Male carriers of low-activity variants of the main functional polymorphism of the *MAOA* gene (*MAOA-uVNTR*) have been shown to exhibit a greater proclivity to engage in violent acts. Thus, we hypothesized that low-activity *MAOA-uVNTR* alleles may be associated with a higher risk for criminal violence among male offenders. To test this possibility, we analyzed the *MAOA-uVNTR* variants of violent (n=49) and non-violent (n=40) male Caucasian and African-American convicts in a correctional facility. All participants were also tested with the Childhood Trauma Questionnaire (CTQ), Barratt Impulsivity Scale (BIS-11) and Buss-Perry Aggression Questionnaire (BPAQ) to assess their levels of childhood trauma exposure, impulsivity and aggression, respectively. Our results revealed a robust ($P<0.0001$) association between low-activity *MAOA-uVNTR* alleles and violent crime. This association was replicated in the group of Caucasian violent offenders ($P<0.01$), but reached only a marginal trend ($P=0.08$) in their African American counterparts. While violent crime charges were not associated with CTQ, BIS-11 and BPAQ scores, carriers of low-activity alleles exhibited a mild, yet significant ($P<0.05$) increase in BIS-11 total and attentional-impulsiveness scores. In summary, these findings support the role of *MAOA* gene as a prominent genetic determinant for criminal violence. Further studies are required to confirm these results in larger samples of inmates and evaluate potential interactions between *MAOA* alleles and environmental vulnerability factors.

Keywords

Monoamine oxidase A; criminal violence; childhood maltreatment; impulsivity; aggression

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Introduction

Criminal violence is one of the most burdensome public-health issues worldwide, with staggering socio-economic repercussions (Krug et al. 2002). The urgent need to develop effective strategies for the prevention of criminal violence has recently given impetus to new research efforts aimed at identifying its psychobiological causes. These investigations have revealed that the propensity towards criminal violence is underpinned by a complex interplay of genetic and socio-environmental factors (Rhee and Waldman, 2002; Gottschalk and Ellis, 2009); the exact nature of these interactions, however, remains poorly understood, also in consideration of the ethical, legal and logistic concerns raised by genetic studies in criminal offenders.

Among the genes that have been implicated in the predisposition to violence, emerging evidence has highlighted a key role for *MAOA*, which encodes for monoamine oxidase A. This enzyme serves a primary role in the metabolism of neurotransmitters implicated in the regulation of aggression, such as serotonin, norepinephrine and dopamine (Bortolato et al. 2008). A loss-of-function mutation of *MAOA* gene has been shown to result in a clinical syndrome characterized by overt proclivity to engage in violent acts in response to minor stressors (Brunner et al. 1993).

The role of MAO A in the neurobiological bases of violence is also confirmed by preclinical studies. In particular, these lines of research have shown that, in mice, the deficiency of this enzyme leads to marked aggressiveness, maladaptive defensive reactivity, defects in information processing and perseverative responses (Cases et al. 1995; Bortolato et al. 2011; Godar et al. 2011; Bortolato et al. 2012; Bortolato et al. 2013).

The link between *MAOA* and violent behavior has been further investigated with respect to its allelic variants, and in particular *MAOA-uVNTR*, a 30-bp functional polymorphism located upstream of its transcription initiation site (Sabol et al. 1998). Six *MAOA-uVNTR* variants have been characterized, based on their different number of repeats (2, 3, 3.5, 4, 5 and 6) (Huang et al. 2004); of these, the 2- and 3-repeat variants are associated to lower transcriptional efficiency and enzymatic activity (Sabol et al. 1998; Deckert et al. 1999; Denney et al. 1999). The low-activity alleles (*L-MAOA*) have been linked to a higher risk of impulsive aggression (Oreland et al. 2007; Buckholtz and Meyer-Lindenberg, 2008), as well as maladaptive processing of affect (Lee and Ham, 2008). In particular, the 2-repeat variant, albeit extremely rare in the general population, has been associated with delinquent and violent behavior (Guo et al. 2008; Beaver et al. 2013; Beaver et al. in press). Several independent studies have also shown that carriers of *L-MAOA* variants with a history of maltreatment during childhood have a significantly higher risk to develop impulsive aggression (Caspi et al. 2002; Kim-Cohen et al. 2006; Williams et al. 2009; Fergusson et al. 2011; but see Haberstick et al. 2014 for conflicting results).

Recently, Beaver and colleagues (2010) documented the association of *L-MAOA* variants with multiple aspects of violent criminal activity, such as gang membership and weapon use. This background suggests that *L-MAOA* alleles may play a role in the predisposition to criminal violence. To the best of our knowledge, however, no study has examined whether

criminal violence in prison inmates could be predicted by *MAOA-uVNTR* genotype. Thus, here we investigated this possibility in a sample of male convicts incarcerated for violent and non-violent acts, and verified whether this relation may be paralleled by alterations in other psychological traits related to violence, namely impulsivity and aggression.

Methods

Participants

The original sample of participants consisted of 49 violent and 40 age-matched non-violent (controls) male inmates at the Lansing Correctional Facility (LCF), located in Lansing, KS. The study was only performed on male offenders because of the well-consolidated association between low-activity *MAOA* variants and aggression in males, but not in females (Sjöberg et al. 2007; Prom-Wormley et al. 2009; Aslund et al. 2011). Each sample consisted of Caucasian and African-American individuals (see Table 1 for a demographic description of the two samples). Violent offenders were defined based on the category of the crime for which they were convicted, and included inmates convicted for 1st and 2nd degree murder, aggravated assault, domestic and non-domestic battery, voluntary manslaughter, aggravated kidnapping, rape and indecent liberties with children. Non-violent crimes included forgery, burglary/robbery/theft, sale and possession of drugs, and DUI manslaughter. All individuals were screened for mental status and potential psychiatric disorders by licensed psychiatrists and trained psychologists of the LCF staff. None of the participants had a history of schizophrenia and/or antisocial personality disorder (based on the diagnostic criteria of the DSM-IV TR). All participants were explained the scope and procedure of the study, and gave oral and written informed consent, under guidelines approved by the Human Subjects Committee of the University of Kansas as well as by the Secretary of Corrections of the Kansas, Department of Corrections. All participants completed psychological and psychiatric evaluations by trained LCF staff upon admission to the facility.

Measures

All participants completed the following psychometric self-report measures: 1) the Childhood Trauma Questionnaire (CTQ) (Bernstein et al. 1994; Bernstein et al. 1997) for the assessment of childhood abuse, neglect or other forms of maltreatment; 2) the Barratt Impulsivity Scale -11 (BIS-11) (Patton et al. 1995), for the assessment of impulsivity; and 3) the Buss-Perry Aggression Questionnaire (BPAQ) (Buss and Perry, 1992), for the assessment of aggressiveness.

Collection of DNA and genotyping

DNA was extracted from buccal swab samples, using the QuickExtract solution and protocol from Epicentre (Madison, WI). *MAOA-uVNTR* allelic variants were genotyped using PCR-based amplification, with the following primers: forward, 5'-ACAGCCTGACCGTGGAGAAG-3'; and reverse, 5'-GAACGGACGCTCCATTCGGA-3'. PCR reactions contained 50 ng of template DNA, 1.0 U GoTaq Flexi DNA polymerase (Promega), 1.0 μM of each primer, 0.3 mM dNTP, 2.0 mM MgCl2, and 5 μl of 5× Green Reaction Buffer (Promega) in a total volume of 25 μl. volume. After 2 min at 95°C, 35 cycles were carried out at 95°C for 1 min, at 59°C for 1 min, and at 72°C for 1 min, with a

final extension at 72°C for 5 min. PCR products were separated on 3% agarose gels and visualized by ethidium bromide staining. All laboratory procedures were carried out blind to the case-control status. All participants were found to harbor 3-repeat (*L-MAOA*) or 4-repeat variants (*H-MAOA*), with the exception of one carrier of the 2-repeat variant, who was added to the *L-MAOA* group (Sabol et al. 1998) for all statistical analyses.

Data analysis

The frequency of *MAOA-uVNTR* allelic variants was compared between violent and non-violent offenders and across Caucasian and African-American participants by two-sided Fisher's Exact Tests. Normality and homoscedasticity of the distribution of continuous variables were verified by Kolmogorov-Smirnov and Bartlett's tests. Thus, the differences of BIS-11, BPAQ and CTQ scores among different groups were tested by three-way ANOVAs, with *MAOA* genotype, violence of the crime and ethnicity as independent factors. Correlations between BIS-11, BPAQ and CTQ scores across violent and nonviolent offenders (as well as genotype groups) were studied by ANCOVAs. Interactions between variables were not tested in the present study, given that the small sample size failed to confer sufficient statistical power for these analyses.

Significance threshold was set at 0.05. However, statistical trends were reported for $P<0.10$. Bonferroni corrections for multiple testing were consistently applied throughout the study. All statistical analyses were performed by STATISTICA 9 software (Statsoft, Tulsa, OK).

Results

Distribution of MAOA genotypes across races and violent convicts

In substantial agreement with previous data on the *MAOA* allelic distribution in the general population (Sabol et al. 1998), we found a trend (P=0.08) toward a significantly higher frequency of African-American carriers of low-activity *MAOA* variants, as compared with their Caucasian counterparts (Fig. 1A). Notably, low-activity *MAOA* variants were displayed by 61.22% violent and 20% non-violent offenders, indicating a robust association of these alleles with violent crime (P<0.0001; Fisher's exact test) (Fig.1B). The odds ratio and relative risk for violent behavior in convicts with low-activity *MAOA* alleles were 6.31 and 2.12, respectively. Fisher's exact test revealed a significantly disproportionate distribution of *MAOA* alleles between violent and non-violent convicts in the Caucasian subgroup (P<0.01) (Fig. 1C). Conversely, only a marginally significant difference (P=0.08) was found in the proportion of low-activity *MAOA* alleles in African-Americans violent and non-violent convicts (Fig. 1D).

Comparisons and correlations of impulsivity, aggression and early-trauma scores

The comparison between genotypes revealed that carriers of low-activity *MAOA* variants exhibited a weak, yet significant increase in BIS-11 total scores (Fig. 2A) [Main effect for genotype: $F(1,85)= 4.52$, $P<0.05$; partial $\eta^2=0.05$] and BIS-11 attentional-impulsiveness subscale scores (Fig. 2B) [Main effect for genotype: $F(1,85)= 4.76$, $P<0.05$; partial $\eta^2=0.05$]. No main effects for ethnicity or race × genotype interactions were identified. Furthermore, no other statistically significant effects were found for other BIS-11 scores,

BPAQ and CTQ scores (Fig. 2 and Suppl. Table 1). ANOVA failed to reveal any significant difference across the four subgroup defined by the combinations of violent crime and *MAOA* genotype with respect to CTQ, BIS-11 and BPAQ scores (Supplementary Fig. 1). Nevertheless, statistical trends were found for the effect of *MAOA* genotype on BIS-11 total score [$F(1,85)=3.63$; $P=0.06$; Main effect], BIS-11 attentional-impulsiveness subscale score [$F(1,85)=3.51$; $P=0.06$; Main effect] and BIS-11 motor-impulsiveness subscale score [$F(1,85)=3.15$; $P=0.08$] (Supplementary Fig.1 G–I). In line with previous results (Garcia-Forero et al. 2009), total BIS-11 and motor-impulsiveness substance scores were significantly correlated with BPAQ total scores, as well as Anger and Hostility subscale scores ($P<0.0002$ for each correlation; Bonferroni correction for multiple testing) (Table 2). ANCOVA identified that none of these correlations was influenced by violent crime charge (Fig.3C), *MAOA* genotype (Fig.3F) or ethnicity (data not shown). CTQ scores did not correlate with either BIS-11 or BPAQ scores (Table 2 and Fig. 3).

Discussion

The main result of the present study was that, in a sample of incarcerated male offenders, violent crime charges were significantly more frequent in carriers of *L-MAOA* alleles. This association was replicated in Caucasian inmates, while it only reached a marginal statistical trend in African-American convicts; this apparent divergence, however, is likely to be due to the demographic characteristics of our sample, which featured a majority of Caucasian convicts. The high frequency of *L-MAOA* alleles in violent offenders is in line with previous reports documenting that *L-MAOA* alleles are associated with an increased risk to commit violent acts (Reif et al. 2007). Furthermore, our results are in keeping with recent evidence highlighting *L-MAOA* as a vulnerability genetic factor for use of weapons and proclivity to violence among members of criminal gangs (Beaver et al. 2010). While our data strongly suggest the implication of *MAOA* in the genetic bases of the predisposition to criminal violence, it is worth noting that this association is likely to hold only in relation to offenders, and not to the general population. *L-MAOA* alleles may predispose to criminal violence by interacting with other known environmental factors linked to a higher predisposition to commit crimes, such as uninvolved and/or permissive parenting style in the family of origin, exposure to early traumas and/or specific forms of maltreatment, abuse of substances etc. Given the extremely small number of subjects in our sample, we could not reach a sufficient statistical power needed to detect interactions between *MAOA* alleles and environmental factors; future studies on much larger samples of inmates and non-convicted subjects will be necessary to identify the environmental factors that may interact with *MAOA* to predict for a higher risk of criminal violence.

The role of *MAOA* in violence may reflect neurodevelopmental alterations in the homeostatic regulation of serotonin, dopamine and norepinephrine neurotransmission, in consideration of the involvement of these systems in the early regulation of aggression circuitry (Bortolato and Shih, 2011). Recent evidence on *MAOA*-deficient mice has shown that the role of MAO A in aggression is mediated by alterations in N-methyl-D-aspartate glutamate receptor (Bortolato et al. 2012) in the prefrontal cortex, which may underpin the impairments in social information processing and environmental reactivity. Accordingly, male carriers of *L-MAOA* alleles have been shown to exhibit pronounced anatomical and

functional alterations of the prefrontal cortex and its limbic connections (Meyer-Lindenberg et al. 2006; Buckholtz et al. 2008), which likely lead to a negative cognitive bias in the interpretation of ambiguous social cues (Buckholtz and Meyer-Lindenberg, 2008), as well as to exaggerated responses to provocation (Kuepper et al. 2013).

Carriers of low-activity *MAOA-uVNTR* variants were also found to exhibit a very mild, yet significant enhancement in BIS-11 total and attention-impulsiveness subscale scores. The very small effect size of these differences underscores the need for further investigations to confirm the association between *MAOA* genotype and impulsivity in offenders. It is worth noting that, in line with previous reports (Barratt, 2000), the average BIS-11 scores in our sample of male inmates (Total: 70.1 ± 12.7; Attentional: 19.1 ± 3.9; Motor: 23.0 ± 5.9; Non-planning: 28.0 ± 5.7) were markedly higher than those reported in the general population (Barratt, 2000), and therefore potentially less suitable to capture subtle differences due to genetic contributions. Nevertheless, previous reports showed that, in the general population, BIS-11 scores are not predicted by L-*MAOA* alleles, but rather by their epistatic interaction variants of the 5-HTTLPR polymorphism of the *SLC6A4* gene (encoding for the serotonin transporter) (Stoltenberg et al. 2012). This finding is particularly interesting, in consideration of the role of the 5-HTTLPR as a genetic determinant for antisocial personality in convicts (Garcia et al. 2010).

In contrast with the associations of the *L-MAOA* genotypes with both violent crime and impulsivity, the lack of correlations between these two latter factors suggests that *MAOA* exerts multiple, mutually independent influences on these behavioral outcomes, possibly through the interplay with different genetic and/or environmental vulnerability factors.

Although previous studies have occasionally shown a direct association between aggression and *L-MAOA* alleles (Aslund et al. 2011; Sjöberg et al. 2007; Prom-Wormley et al. 2009), this relation was not apparent in our sample. These findings are in line with previous studies, which failed to identify an influence of *MAOA-uVNTR* genotype on BPAQ scores (Hurd et al. 2011). Although previous evidence has documented an association between *L-MAOA* alleles and high indices of antisocial personality, including anger and hostility (Yang et al. 2007; McDermott et al. 2009; Williams et al. 2009), ample evidence has shown that *L-MAOA* alleles do not confer an inherent predisposition to aggression (Fowler et al. 2007), but their influence on this trait is likely mediated by interactions with early exposure to traumatic experiences (Caspi et al. 2002; Kim-Cohen et al. 2006; Williams et al. 2009; Fergusson et al. 2011) and/or other concurrent risk factors (Beaver et al. 2011), including parenting style (Pickles et al. 2013).

Our analyses revealed a lack of association between criminal violence and either impulsivity or aggression scores. These results are in keeping with previous findings by Williams et al (1996), who failed to identify any significant differences in BPAQ scores between violent and non-violent offenders. While BPAQ total scores have been shown to predict the proclivity to engage in fights and violent acts in the general population (Buss and Perry, 1992; Archer et al. 1995), it should be noted that the nature of the crime does not accurately reflect the criminal history and pattern of aggressive conduct of inmates. This idea is indirectly supported by previous evidence documenting that, while the intensity of bullying

behavior in incarcerated offenders is associated with BPAQ total scores (Palmer and Thakordas, 2005), it is not strongly predicted by the nature of the criminal charge (Ireland and Ireland, 2000). In addition, it should be observed that the relation between aggression levels and the violent nature of the crime may be modified by the actual exposure to the prison environment, which has been shown to significantly affect the social behavior of inmates (Bottoms, 1999; Edgar et al. 2003; Homel and Thompson, 2005).

L-MAOA variants have been typically associated with reactive aggression (McDermott et al. 2009), a trait characterized by irritability, reduced self-control, as well as maladaptive perceptions of ambiguous social cues and information processing deficits (Dodge and Coie, 1987; Dodge, 1991; Crick and Dodge, 1996; Scarpa and Raine, 1997; Volavka, 1999; Coccaro et al. 2007; Wilkowski and Robinson, 2008). Conversely, *L-MAOA* variants in inmates may be negatively associated with proactive aggression (Tikkanen et al. 2011), which features callous and unemotional conduct and instrumental violence (Frick and Ellis, 1999; Frick and White, 2008). Given the predominance of proactive-aggressive traits in violent offenders (Hare and McPherson, 1984), this background is apparently at variance with the robust association between *L-MAOA* and criminal violence documented by our results. Nevertheless, it should be noted that reactive and proactive aggression are often correlated (Vitaro and Brendgen, 2005) and share common genetic bases (Baker et al. 2008). Indeed, these overlapping heritable factors may be specifically related to the predisposition to physical aggression, a common underlying form between reactive and aggression (Little et al. 2003; Brendgen et al. 2006). Capitalizing on this notion, our results may indicate that the endophenotypic anomalies associated with *L-MAOA* alleles may lead to a higher predisposition for exteriorizing aspects of physical violence, which may be common to both reactive and proactive aggression.

Several limitations should be considered in the interpretation of the results of this study, such as the exclusive employment of self-reported measures, which may raise issues on the reliability of our findings. In addition, the conclusions of the study are limited by the relatively small sample size and the inclusion of only male convicts; nevertheless, it should be noted that recruitment of prison inmates for research purposes is extremely problematic, in view of significant legal and logistical hurdles, as well as important ethical limitations (Gostin et al. 2007). From this perspective, the number of subjects recruited for our study was comparable or higher than other recent genetic and investigations in this population (Garcia et al. 2010; Aluja et al. 2011).

While these caveats need to be addressed in future larger studies, it is important to underscore that our results provide the first demonstration of an association between *L-MAOA* variants and criminal violence in incarcerated offenders. While this evidence is in support of the importance of heritable biological components in violent crime, future studies on larger cohorts of subjects and non-convicted individuals are necessary to clarify the role of *MAOA* polymorphism in the predisposition to violence. Until then, it is clear that any application of our findings to the criminal justice system should be done with extreme caution, particularly in view of recent controversial judicial decisions in Italy and USA, in which genetic evidence on *MAOA-uVNTR* variants was used to justify sentence reductions (Baum, 2013). This type of interpretation appears premature, given our poor understanding

of the neurobiological and cognitive underpinnings of violence predisposition. Thus, we strongly advocate extreme caution against any potential misrepresentations of the present results. Nevertheless, it appears that the present data support the idea that a significant fraction of violent crimes might be related to genetic predispositions, involving genes involved in serotonergic and dopaminergic neurotransmission. The incorporation of this concept in our current criminological framework could lead to a significant improvement in the development of interventional strategies for violent crime. In particular, the identification of potential biomarkers for risk of criminal violence may help enact preventive programs for highly predisposed youth, and result in a significant reduction of the staggering socio-economic burden of this important problem.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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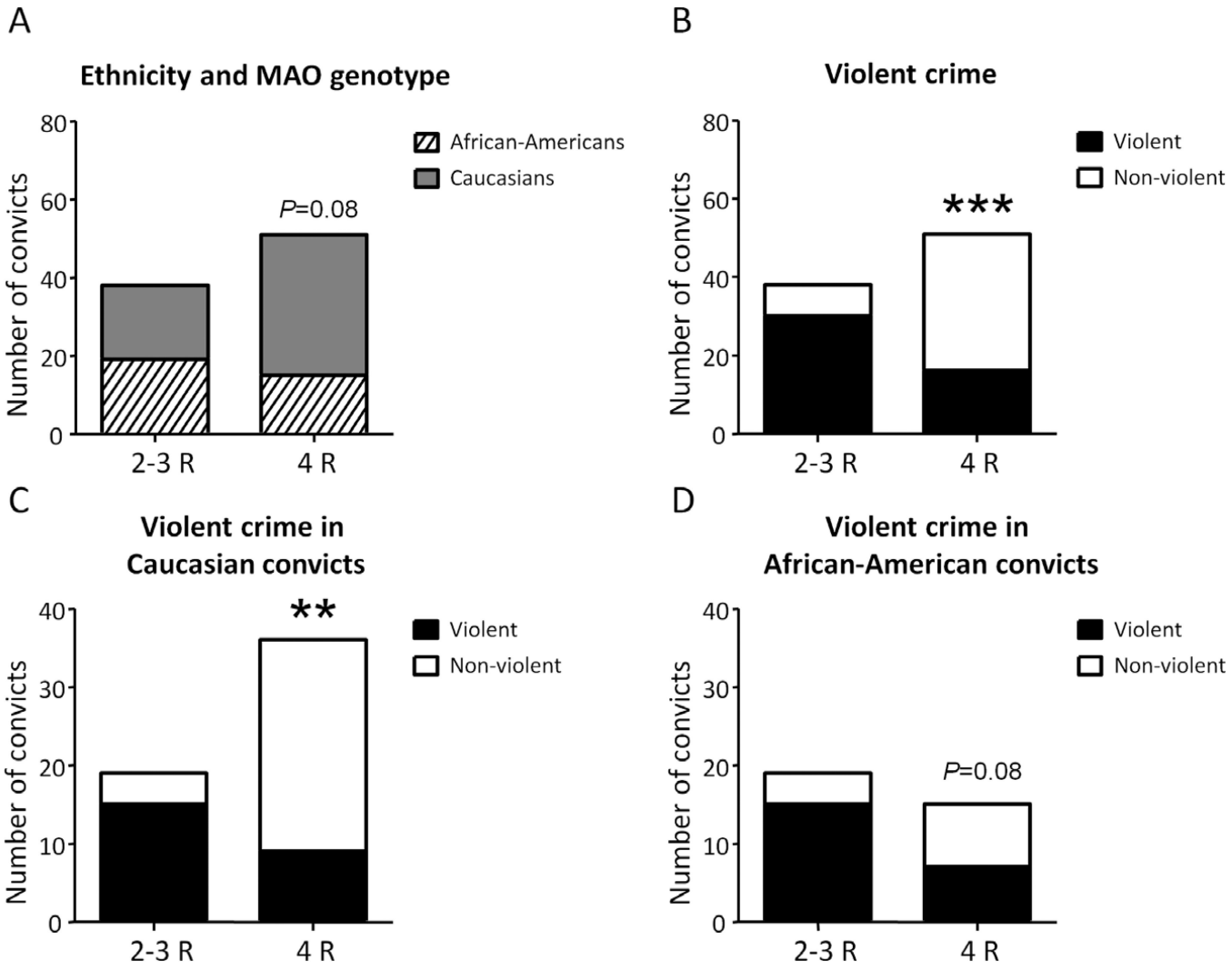

Fig.1.

Comparisons of the frequencies of *MAOA*-uVNTR allele carriers with respect to violent crime and ethnicity. *L-MAOA*, low-activity *MAOA-uVNT*R variants (2 and 3 repeats); *H-MAOA*, high-activity *MAOA-uVNTR* variants (4 repeats).**, $P<0.01$; ***, $P<0.001$ in comparison with *L-MAOA*. For more details, see text.

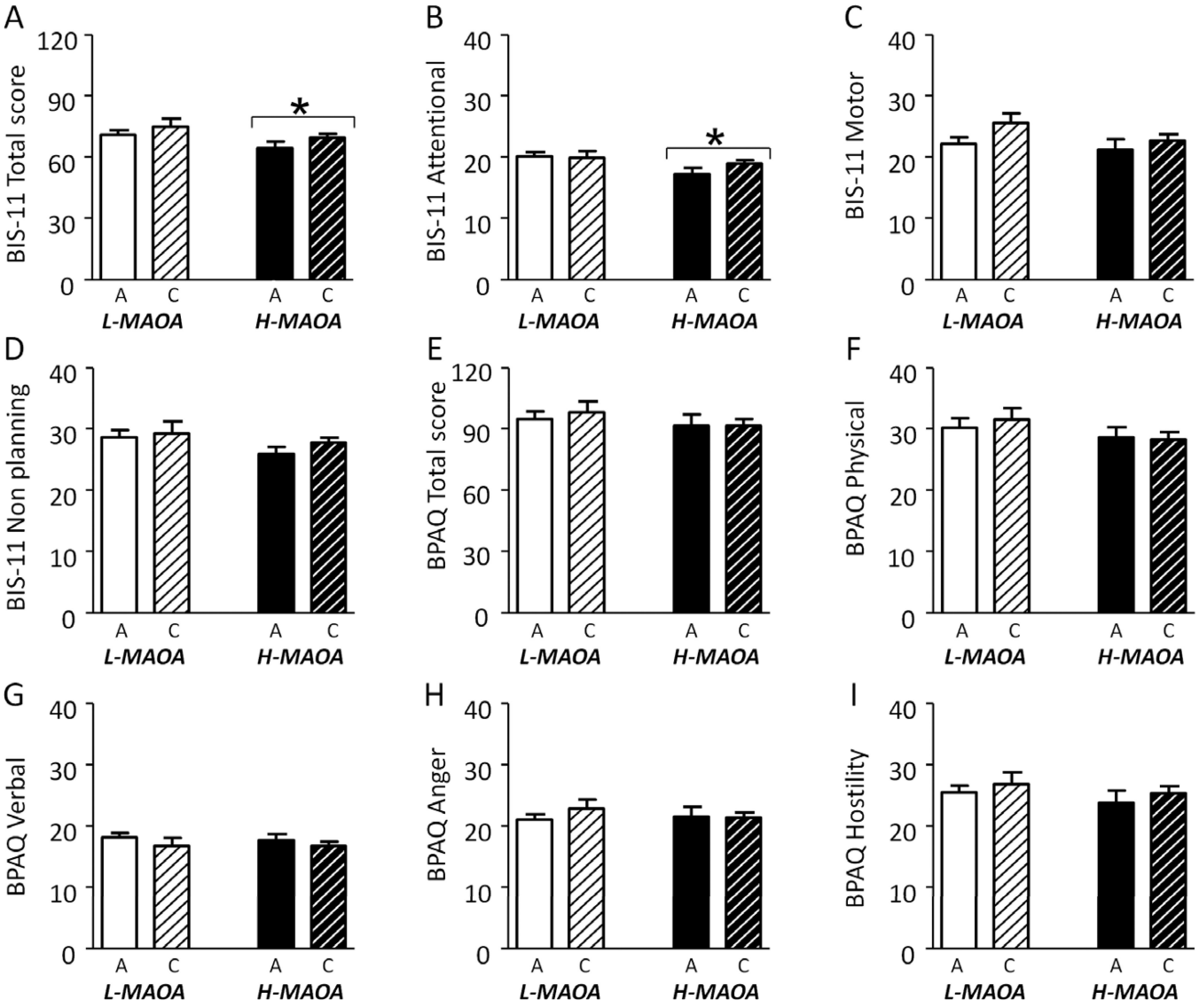

Fig.2.

Comparisons of BIS-11 and BPAQ scores across MAOA-uVNTR genotype and ethnicity. A, African-Americans; C, Caucasians. *L-MAOA*, low-activity *MAOA-uVNT*R variants (2 and 3 repeats); *H-MAOA*, high-activity *MAOA-uVNTR* variants (4 repeats). *, $P<0.05$ for comparisons with L-MAOA variants (Main effect). All values are represented as means ± SEM. For more details, see text.

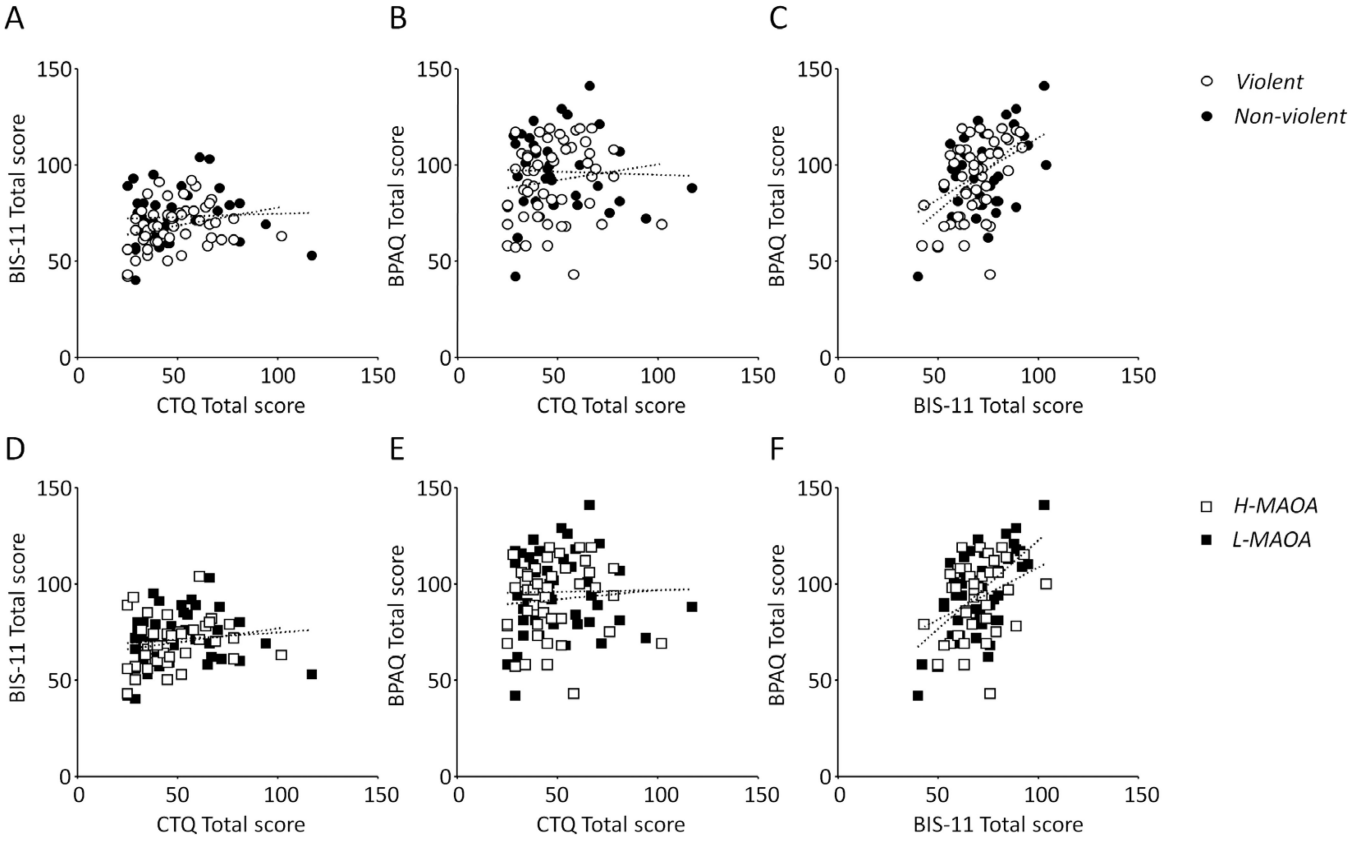

Fig.3.

Correlations of total CTQ, BIS-11 and BPAQ scores across *MAOA-uVNTR* genotypes and violence of the crime charge. For more details, see text.

Table 1

Demographic characteristics of study participants

	Violent	Non-violent	Overall
Number	**49**	**40**	**89**
Mean age (SD)	32.63 (11.57)	30.93 (8.71)	31.81 (10.27)
Median age	28	30	29
Range	18–77	20–50	18–77
Race/ethnicity			
African American	44.90%	30.00%	38.20%
Caucasian	55.10%	70.00%	61.80%
Type of offense			
Murder/Voluntary manslaughter	10.20%	n/a	5.62%
Assault/battery	55.10%	n/a	30.34%
Sexual abuse/Rape	14.29%	n/a	7.87%
Kidnapping	8.16%	n/a	4.49%
Indecent liberties with children	40.82%	n/a	22.47%
Drug manufacturing/delivery/possession	34.69%	30.00%	32.58%
Burglary/Theft/Robbery	87.76%	77.50%	83.15%
Fraud/Forgery	10.20%	12.50%	11.24%
Involuntary manslaughter	2.04%	2.50%	2.25%

Table 2

Synoptic tables of correlations between CTQ, BIS-11 and BPAQ scale scores for all participants (irrespective of genotype and violent crime).

CTQ TOT															
CTQ EA	**0.82**														
CTQ PA	**0.82**	**0.66**													
CTQ SA	**0.72**	**0.49**	**0.65**												
CTQ EN	**0.73**	**0.50**	0.33	**0.30**											
CTQ PN	**0.67**	0.34	**0.46**	**0.25**	**0.58**										
BIS-11 TOT	0.15	0.19	0.09	−0.15	0.23	0.20									
BIS-11 ATT	0.18	0.19	0.10	−0.05	0.24	0.18	**0.87**								
BIS-11 MOT	0.08	0.23	0.06	−0.16	0.11	0.01	**0.81**	**0.63**							
BIS-11 NP	0.13	0.05	0.06	−0.14	0.23	0.32	**0.79**	**0.59**	0.35						
BPAQ TOT	0.05	0.21	0.13	−0.14	0.03	−0.05	**0.48**	**0.45**	**0.56**	0.20					
BPAQ PHYS	0.05	0.15	0.14	−0.11	0.00	−0.01	0.37	0.36	**0.43**	0.14	**0.88**				
BPAQ VOC	−0.05	0.14	0.10	−0.14	−0.10	−0.23	0.29	0.25	0.36	0.10	**0.78**	**0.60**			
BPAQ ANG	0.03	0.09	0.11	−0.10	0.04	−0.02	**0.52**	**0.48**	**0.54**	0.26	**0.86**	**0.76**	**0.51**		
BPAQ HOST	0.11	0.28	0.09	−0.13	0.11	0.01	**0.43**	0.38	**0.53**	0.15	**0.83**	**0.52**	**0.63**	**0.59**	
TOTAL	**CTQ TOT**	**CTQ EA**	**CTQ PA**	**CTQ SA**	**CTQ EN**	**CTQ PN**	**BIS11 TOT**	**BIS ATT**	**BIS MOT**	**BIS NP**	**BNAQ TOT**	**BNAQ PHYS**	**BPAQ VOC**	**BPAQ ANG**	**BPAQ HOST**

Significant correlations ($P<0.0002$; Bonferroni correction for multiple testing) are marked in bold. The values indicated in the tables are the β coefficients of each correlation.

Abbreviations: TOT: total score; EA, emotional abuse; PA, physical abuse; SA, sexual abuse; EN, emotional neglect; PN, physical neglect; ATT, attentional; MOT, motor; NP, non-planning; PHYS, physical aggression; VOC, vocal aggression; ANG, anger; HOST, hostility.