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

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Running title: MAOA and criminal violence

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The main enzyme for serotonin degradation, monoamine oxidase (MAO) A, has recently emerged as a key biological factor in the predisposition towards violence. In particular, impulsive aggression in males has been associated with the interaction of child maltreatment and low-activity variants of the main functional polymorphism of the MAOA gene (MAOA-uVNTR). Based on this background, we hypothesized that the same gene-environment interplay may also predict a higher proclivity to engage in 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 association between low-activity MAOA-uVNTR alleles and violent crime, irrespectively of aggression and impulsivity levels. The same genotype predicted for high-impulsivity trait and interacted with the CTQ score for physical abuse with respect to the BPAQ anger scale scores. Violent crime charges, however, were not associated with CTQ, BIS-11 and BPAQ scores. Furthermore, a robust correlation was found between BIS-11 and scores, irrespective of the violent nature of crime, MAOA-uVNTR genotype and ethnicity. In summary, these preliminary findings support the role of MAOA gene as a prominent genetic determinant to the predisposition to violence in criminals. Further studies are required to confirm these results in larger samples of inmates.

**Keywords:** Monoamine oxidase A, criminal violence, childhood maltreatment, impulsivity, aggression
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 largely 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 actions in response to minor stressors (Brunner et al., 1993). The role of MAO A in the neurobiological bases of violence is also confirmed by animal studies, which 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-Lindentberg, 2008), as well as maladaptive processing of affect (Lee and Ham, 2008). In addition, several independent studies have 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 could be predicted by the interaction of early trauma and 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 the association between this gene x environment interplay may be moderated by other psychological traits related to violence, namely impulsivity and aggression.
Methods

Participants. The original sample of participants consisted of 49 violent and 42 age-matched non-violent (controls) male inmates at the Lansing Correctional Facility (LCF), located in Lansing, KS. The study was limited to males 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, in comparable proportions (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. Two non-violent participants were excluded from the study because they had a schizophrenia diagnosis; thus, the final analyses included 40 non-violent inmates.
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’- GAACGGACGCTCCATTGCAGA-3’. PCR reactions contained 25 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 5X 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. To study the distribution of high
impulsivity in our sample with respect to genotype and violent crime, the top quartile of BIS-11 total score was used as a cut-off value to differentiate between high- and low-impulsivity individuals, as previously indicated (Baca-Garcia et al., 2004; Maloney et al., 2009). The same procedure, based on BPAQ Total scores, was used to study the distribution of highly aggressive subjects in our sample.

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. Homogeneity-of-slopes designs were used to test for interactions between continuous and categorical predictors.

The analysis of gene - environment interactions with respect to BIS-11 and BPAQ scores, as well as criminal violence, was conducted by mixed general-linear model (GLM) designs, incorporating factorial regression analyses for continuous variables. MAOA genotype (treated as a dichotomous variable, dummy-coded as 0=2-or 3-repeat alleles and 1=4-repeat alleles), and CTQ scores were used as predictors, ethnicity as a categorical variable and either BPAQ or BIS-11 total scores as dependent, continuous variables.

Significance threshold was set at 0.05. However, statistical trends were reported for p<0.10. False discovery rate 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 convicts.** Low-activity MAOA variants were displayed by 61.22% of violent and 20% of non-violent offenders, indicating a robust association of these alleles with violent crime (P<0.0001; Fisher's exact test) (Fig.1A). The odds ratio and relative risk for violent behavior in convicts with 2 or 3-repeat MAOA alleles were 6.31 and 2.12, respectively. Subsequent analyses, however, showed that the distribution of the individuals in the top quartiles of the BPAQ and BIS-11 scores was comparable across violent and non-violent convicts. Notably, a statistical trend (P<0.10) towards a disproportionate distribution of the low-activity genotypes was also found in African American (55.88%) convicts in comparison with Caucasian inmates (34.55%). In separate analyses, we also found that the low-activity MAOA genotype was significantly associated with high-impulsivity (P<0.05) (Fig.1B), but not high-aggression trait (Fig. 1C).

**Comparisons and correlations of impulsivity, aggression and early-trauma scores.** ANOVAs 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 (Table 2 and supplementary Figure 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 score [F(1,85)=3.51; P=0.06; Main effect] and BIS-11 motor score [F(1,85)=3.15; P=0.08] (Supplementary Fig.1 G-I). In line with previous results (Garcia-Forero et al., 2009), all BIS-11 scores, with the only exception of those related to non-planning impulsivity, were significantly correlated with all BPAQ scores (Table 3). ANCOVAs identified that the correlation was not affected by the criminal charge (Fig.2C), MAOA genotype (Fig.2F) or ethnicity (data not shown). CTQ
total scores did not correlate with either BIS-11 (Figs.3A and 3D) or BPAQ total scores (Figs.2B and 2F); however, the CTQ scores for emotional neglect were significantly correlated with all BIS-11 scores with the only exception of those related to motor impulsivity. The CTQ score for physical neglect was also correlated with BIS-11 score for non-planning impulsivity. The CTQ score for emotional abuse was correlated with the BIS-motor impulsivity score (Table 3). None of these correlations was affected by the violent nature of the crime, MAOA-uVNTR alleles or ethnicity (data not shown).

**Interactive effects of MAOA genotype, age and early trauma in aggression, impulsivity and violent crime.** GLM factorial regression designs were used to study the potential interactions of MAOA genotype and CTQ scores with respect to BIS-11 and BPAQ total scores. The results of these analyses indicated a significant interaction between the CTQ score for physical abuse and low MAOA genotype, which resulted in higher BPAQ anger scores \( [F(1,85)=4.05, P<0.05; \beta=0.56\pm0.28] \). A statistical trend was also found for an interaction between the CTQ score for physical abuse and low-activity MAOA genotype, resulting in higher physical violence scores \( [F(1,85)=2.77, P<0.10; \beta=0.46\pm0.27] \). No other interaction for CTQ scores and MAOA genotype was found with respect to other BPAQ or BIS-11 scores. No significant differences were found with respect to ethnicity (data not shown).
**Discussion**

The main results of the present study documented that, in a sample of male Caucasian and African-American offenders, violent crime charges were significantly more frequent in carriers of $L$-MAOA alleles; however, criminal violence was not associated with either impulsivity or aggression scores. Low-activity $MAOA-uVNTR$ variants were also found to predispose to high impulsivity and to interact significantly with the severity of physical abuse in childhood to predict for higher intensity of anger, irrespective of the violent nature of the crime or the ethnicity. The multiple, mutually independent associations of the $L$-MAOA genotype with violent crime, impulsivity and anger (through the interplay with early physical abuse) suggests that $MAOA$ exerts a multifaceted influence on the predisposition to different behavioral outcomes, through the interplay with diverse environmental vulnerability factors.

The high frequency of $L$-MAOA alleles in violent offenders is in line with previous reports documenting low catalytic activity of MAO in violent criminals (Davis et al., 1983). Additionally, other studies found that $L$-MAOA alleles increased the 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).

The role of MAOA in violence may reflect homeostatic alterations in the regulation of the serotonergic system and/or dysfunctions in the regulation of dopamine and norepinephrine neurotransmission, in consideration of the involvement of these systems in the modulation of aggression (Bortolato and Shih, 2011). Recent evidence on MAOA-deficient mice have 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 \textit{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).

Irrespective of the potential neurobiological mechanisms, our data strongly suggest that, in criminal offenders, MAOA may be a key genetic factor in the predisposition to criminal violence. Nevertheless, it is worth noting that the association between \textit{L-MAOA} genotype and criminal violence is likely to hold true only in relation to offenders, and not to the general population. Indeed, a plausible interpretation of this result is that \textit{L-MAOA} allele may predispose to criminal violence by interacting with other known environmental factors linked to a higher predisposition to commit crimes, such as a permissive parenting style in the family of origin, abuse of substances etc. In our study, the potential influence of these sociological variables on violence was clearly reduced by the selection bias of our design, which was limited to a sample of inmates. Future studies on larger samples in the general population will be necessary to identify the factors that may interact with MAOA to predict for a higher risk of criminal violence.

Although previous studies have occasionally shown a direct association between aggression and \textit{L-MAOA} alleles (Aslund et al., 2011; Sjöberg et al., 2007; Prom-Wormley et al., 2009), this relation was not apparent in our sample. Indeed, previous studies failed to identify an influence of \textit{MAOA-uVNTR} genotype on BPAQ scores (Hurd et al., 2011). Additionally, it has been shown that \textit{L-MAOA} alleles do not inherently confer predisposition to aggression (Fowler et al., 2007), but their influence on this trait is likely mediated by interactions with the early exposure to traumatic experiences (Caspi et al, 2002; Kim-Cohen et al., 2006; Williams et al., 2009; Fergusson et al., 2011).
In this respect, it is worth noting that, in our sample of incarcerated offenders, \textit{L-MAOA} alleles interacted only with early physical abuse to predict for the severity of anger (and possibly physical aggression, as suggested by a marginal statistical trend). The fact that the interaction of the \textit{L-MAOA} genotype was limited to physical abuse but did not involve other aspects of child maltreatment may be reflective of specific characteristics of delinquent individuals, such as the low socio-economic status of the family of origin (Lipsey and Derzon, 1998).

The lack of differences between any dimensions of aggression and the type of criminal charge is in line with previous results by Williams et al (1996), who failed to identify any significant differences in BPAQ scores between violent and non-violent offenders. This result is at odds with previous findings on the general population, in which the BPAQ total scores predict the proclivity to engage in fights and violent acts (Buss and Perry, 1992; Archer et al., 1995); however, 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 reports which have shown 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 significantly 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).

\textit{L-MAOA} variants have been typically associated with reactive aggression (McDermott et al, 2009), a trait characterized by anger, irritability, reduced self-control, high impulsivity, 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 emotionless 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 a 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). Another problem of our study is that the ratio of African American and Caucasian inmates was
significantly higher among inmates incarcerated for violent crimes in comparison with the non-violent counterparts. However, it is highly unlikely that this difference may have skewed our results, in consideration of the lack of any statistical effect for ethnicity across our findings.

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 \textit{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 \textit{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 \textit{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.
Acknowledgements

We thank the inmates and personnel at LCF for their participation and assistance. The conclusions, interpretations and recommendations expressed in this work are those of the authors and do not necessarily reflect the position or policy of the Kansas Department of Corrections.
### Table 1. Demographic characteristics of study participants

<table>
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<tr>
<th></th>
<th>Violent</th>
<th>Non-violent</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>49</td>
<td>40</td>
<td>89</td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>32.63 (11.57)</td>
<td>30.93 (8.71)</td>
<td>31.81 (10.27)</td>
</tr>
<tr>
<td>Median age</td>
<td>28</td>
<td>30</td>
<td>29</td>
</tr>
<tr>
<td>Range</td>
<td>18-77</td>
<td>20-50</td>
<td>18-77</td>
</tr>
<tr>
<td><strong>Race/ethnicity</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>44.90%</td>
<td>30.00%</td>
<td>38.20%</td>
</tr>
<tr>
<td>Caucasian</td>
<td>55.10%</td>
<td>70.00%</td>
<td>61.80%</td>
</tr>
<tr>
<td><strong>Type of offense</strong></td>
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<tr>
<td>Murder/Voluntary manslaughter</td>
<td>10.20%</td>
<td>n/a</td>
<td>5.62%</td>
</tr>
<tr>
<td>Assault/battery</td>
<td>55.10%</td>
<td>n/a</td>
<td>30.34%</td>
</tr>
<tr>
<td>Sexual abuse/Rape</td>
<td>14.29%</td>
<td>n/a</td>
<td>7.87%</td>
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<td>Kidnapping</td>
<td>8.16%</td>
<td>n/a</td>
<td>4.49%</td>
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<td>Indecent liberties with children</td>
<td>40.82%</td>
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<tr>
<td>Drug manufacturing/delivery/possession</td>
<td>34.69%</td>
<td>30.00%</td>
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<tr>
<td>Burglary/Theft/Robbery</td>
<td>87.76%</td>
<td>77.50%</td>
<td>83.15%</td>
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<tr>
<td>Fraud/Forgery</td>
<td>10.20%</td>
<td>12.50%</td>
<td>11.24%</td>
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<tr>
<td>Involuntary manslaughter</td>
<td>2.04%</td>
<td>2.50%</td>
<td>2.25%</td>
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### Table 2. Psychometric characteristics of study participants

<table>
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<th></th>
<th>Violent</th>
<th>Non-violent</th>
<th>Overall</th>
</tr>
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<tbody>
<tr>
<td><strong>Number</strong></td>
<td>49</td>
<td>40</td>
<td>89</td>
</tr>
<tr>
<td><strong>CTQ Scores (Mean ± SD)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>49.68 ± 19.53</td>
<td>47.26 ± 16.88</td>
<td>48.51 ± 18.24</td>
</tr>
<tr>
<td>Emotional Abuse</td>
<td>10.47 ± 5.59</td>
<td>10.49 ± 5.45</td>
<td>10.48 ± 5.49</td>
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<tr>
<td>Physical Abuse</td>
<td>10.32 ± 4.58</td>
<td>9.81 ± 4.39</td>
<td>10.08 ± 4.47</td>
</tr>
<tr>
<td>Sexual Abuse</td>
<td>7.65 ± 5.32</td>
<td>7.09 ± 4.31</td>
<td>7.38 ± 4.84</td>
</tr>
<tr>
<td>Emotional Neglect</td>
<td>11.78 ± 5.70</td>
<td>11.40 ± 5.20</td>
<td>11.60 ± 5.44</td>
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<tr>
<td>Physical Neglect</td>
<td>9.43 ± 4.37</td>
<td>8.467 ± 3.42</td>
<td>8.97 ± 3.95</td>
</tr>
<tr>
<td><strong>BIS-11 Scores (Mean ± SD)</strong></td>
<td></td>
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<td></td>
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<tr>
<td>Total</td>
<td>71 ± 13.38</td>
<td>69.09 ± 12.03</td>
<td>70.08 ± 12.71</td>
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<tr>
<td>Attentional</td>
<td>19.43 ± 3.79</td>
<td>18.79 ± 4.05</td>
<td>19.12 ± 3.91</td>
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<tr>
<td>Motor</td>
<td>22.87 ± 6.16</td>
<td>23.09 ± 5.65</td>
<td>22.98 ± 5.88</td>
</tr>
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<td>Non-planning</td>
<td>28.70 ± 5.92</td>
<td>27.21 ± 5.48</td>
<td>27.98 ± 5.73</td>
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<tr>
<td><strong>BPAQ Scores (Mean ± SD)</strong></td>
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<td></td>
</tr>
<tr>
<td>Total</td>
<td>95.70 ± 20.78</td>
<td>91.58 ± 19.36</td>
<td>93.71 ± 20.10</td>
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<tr>
<td>Physical</td>
<td>30.67 ± 7.27</td>
<td>28.12 ± 7.35</td>
<td>29.44 ± 7.38</td>
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<tr>
<td>Verbal</td>
<td>17.41 ± 4.40</td>
<td>17 ± 4.08</td>
<td>17.22 ± 4.23</td>
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<tr>
<td>Anger</td>
<td>22.04 ± 5.35</td>
<td>21.19 ± 5.32</td>
<td>21.63 ± 5.32</td>
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<tr>
<td>Hostility</td>
<td>25.57 ± 7.19</td>
<td>25.28 ± 6.81</td>
<td>25.43 ± 6.97</td>
</tr>
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</table>
### Table 3

Synoptic tables of correlations between CTQ, BIS-11 and BPAQ scale scores. Significant correlations (P<0.05) 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.

<table>
<thead>
<tr>
<th></th>
<th>CTQ TOT</th>
<th>CTQ EA</th>
<th>CTQ PA</th>
<th>CTQ SA</th>
<th>CTQ EN</th>
<th>CTQ PN</th>
<th>BIS ATT</th>
<th>BIS MOT</th>
<th>BIS NP</th>
<th>BPAQ TOT</th>
<th>BPAQ PHYS</th>
<th>BPAQ VOC</th>
<th>BPAQ ANG</th>
<th>BPAQ HOST</th>
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<td>CTQ TOT</td>
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<tr>
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<td>BIS ATT</td>
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<td>0.11</td>
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<td>0.43</td>
<td>0.40</td>
<td>0.54</td>
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<tr>
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<td>0.43</td>
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<td>0.11</td>
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</table>
Figure Legends

**Fig.1** Comparisons of the frequencies of MAOA-uVNTR allele carriers with respect to violent crime, impulsivity and aggression. *L-MAOA*, low-activity *MAOA-uVNTR* variants (2 and 3 repeats); *H-MAOA*, high-activity *MAOA-uVNTR* variants (4 repeats). *, $P<0.05$; ***, $P<0.001$ in comparison with *L-MAOA*. For more details, see text.

**Fig. 2** Correlations of total CTQ, BIS-11 and BPAQ scores across *MAOA-uVNTR* genotype and violence of the crime charge. For more details, see text.

**Supplementary Fig.1** Comparisons of CTQ, BIS-11 and BPAQ scores across *MAOA-uVNTR* genotype and violence of the crime charge. V, violent offenders; NV, non-violent offenders; *L-MAOA*, low-activity *MAOA-uVNTR* variants (2 and 3 repeats); *H-MAOA*, high-activity *MAOA-uVNTR* variants (4 repeats). All values are represented as means ± SEM. For more details, see text.
References


Beaver KM, DeLisi M, Vaughn MG, Barnes JC. Monoamine oxidase A genotype is associated with gang membership and weapon use. Comprehensive Psychiatry 2010; 51:130-134.


Brunner HG, Nelen M, Breakefield XO, RPersonality HH, van Oost BA. Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. Science 1993; 262: 578-580.


Huang YY, Cate SP, Battistuzzi C, Oquendo MA, Brent D, Mann JJ. An association between a functional polymorphism in the monoamine oxidase a gene promoter, impulsive traits and early abuse experiences. Neuropsychopharmacology 2004; 29:1498-1505.

Hurd PL, Vaillancourt KL, Dinsdale NL. Aggression, digit ratio and variation in androgen receptor and monoamine oxidase a genes in men. Behavioral Genetics 2011; 41:543-556.


Little TD, Jones SM, Henrich CC, Hawley PH. Disentangling the "whys" from the "whats" of aggressive behavior. International Journal of Behavioral Development 2003; 27: 122–133


