Effects of Childhood Adversity and Genes on Aggression in Male Prisoners: A Postprint

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Abstract

Violent recidivism risk assessment constitutes a priority in contemporary recidivism risk evaluation, with aggression representing a stable individual factor underlying violent reoffending among offenders. Investigating aggression facilitates prevention and reduction of violent recidivism risk during parole or post-release, thereby contributing to long-term social stability. Research demonstrates that childhood adversity exposure and carriage of susceptibility genes (e.g., low-activity alleles of MAOA-uVNTR) are significant contributors to aggressive behavior. However, extant research is limited by: (1) scoring methods for childhood adversity restricted to simple linear summation; (2) statistical models that neglect interactive and nonlinear relationships among adversity dimensions; and (3) offender aggression assessments that overlook aggression subtypes while predominantly employing self-report measures—these issues collectively constrain predictive validity. This study proposes to establish latent class models to identify childhood adversity subtypes among male offenders and general adult populations; utilizing experimental and questionnaire measures alongside judicial behavioral records as aggression indicators, we will elucidate how childhood adversity influences proactive aggression, reactive aggression, and violent criminal conduct. Specifically, we will examine the effects of latent childhood adversity classes on proactive and reactive aggression, and the moderating roles of MAOA-uVNTR, COMT Val158Met, and 5-HTTLPR genetic polymorphisms. Findings will facilitate identification of biological-genetic markers for high-aggression individuals, enabling detection of populations susceptible to childhood adversity effects, and provide theoretical and empirical foundations for violent behavior risk prediction, behavioral correction of violent aggression, and related pharmacological design, thereby enhancing intervention efficacy.

Full Text

The Effect of Childhood Adversity and Genetic Factors on Male Prisoners' Aggression

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Abstract

Violent recidivism risk assessment is a critical component of contemporary correctional evaluation work, with aggression representing a stable individual factor underlying violent reoffending among prisoners. Investigating aggression is essential for preventing and reducing the risk of violent recidivism after parole or release, thereby contributing to long-term social stability. Research has demonstrated that experiencing childhood adversity and carrying susceptibility genes (such as the low-activity allele of MAOA-uVNTR) are important contributors to aggressive behavior. However, existing studies have limitations: childhood adversity is typically scored through simple linear summation or statistical models that ignore interactions among adversity dimensions and nonlinear relationships; assessments of prisoner aggression have not considered aggression subtypes and have predominantly relied on self-report questionnaires, which constrains predictive validity. This study proposes to establish latent class models to analyze subtypes of childhood adversity among male prisoners and ordinary adults; to reveal how childhood adversity influences proactive aggression, reactive aggression, and violent criminal behavior using experimental and questionnaire measures alongside judicial behavioral records; and to examine the differential effects of childhood adversity latent classes on proactive versus reactive aggression, as well as the moderating role of MAOA-uVNTR, COMT Val158Met, and 5-HTTLPR polymorphisms. The findings will help identify biological-genetic markers of highly aggressive individuals and detect susceptible populations affected by childhood adversity, providing theoretical and empirical references for violent behavior risk prediction, behavioral correction interventions, and related pharmacological design.

Keywords: childhood adversity; gene; aggression; latent class analysis
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1 Introduction

Aggression refers to the internal psychological characteristics that underlie aggressive behavior (Ye, 2003), though some scholars conceptualize aggression as the behavior itself—purposeful, intentional acts that harm or attempt to harm another individual's psychological or physical condition, or destroy other targets (objects or social norms) (Crick & Grotpeter, 1995). Violent behavior involves using violent means or threats of violence to cause harmful consequences or danger to others (Zhang, 2011), representing an extreme form of aggressive behavior. Violent criminal behavior refers to criminal acts fundamentally characterized by violence, such as assault, homicide, and robbery (Zhang, 2014). Aggressive behavior, violent behavior, and violent criminal behavior all reflect individual aggression. Violent aggression is a complex social behavior in human society, a common feature of many mental disorders, and represents serious social maladjustment. Despite civilized contexts, various violent incidents frequently appear in media reports, attracting widespread social attention. For example, in October 2018, an illegal gathering in Pingdu City, Shandong Province, involved individuals with criminal records who committed violent acts against police, vandalized vehicles, and engaged in other serious criminal behaviors, causing severe personal injury, significant economic losses, and major threats to public order. Thus, investigating the causes of violent aggression is crucial for preventing and intervening in such incidents and holds important socioeconomic significance.

The etiology of aggression can be divided into environmental and genetic factors. Childhood adversity represents a critical environmental contributor to aggression. Numerous studies have shown that individuals who experienced childhood abuse exhibit higher levels of aggressive behavior (Connor, Steingard, Cunningham, Melloni, & Anderson, 2004; Li et al., 2010). On the other hand, molecular genetics research demonstrates that MAOA, COMT, and 5-HTT genes are closely associated with the development of violent aggression (Singh, Volavka, Czobor, & Van Dorn, 2012; Ficks & Waldman, 2014). In recent years, increasing attention has focused on gene-environment (G×E) interactions in aggressive behavior (Byrd & Manuck, 2014; Tielbeek et al., 2016). However, these studies have several limitations: (1) Childhood adversity encompasses abuse, neglect, and family dysfunction, with different types potentially exerting different effects on aggression. Yet existing research typically uses simple summation of adversity scores or counts (Cui et al., 2013; Auslander et al., 2016), thereby ignoring qualitative differences in childhood adversity and obscuring individuals' cumulative and combinative patterns across adversity types. (2) Based on the perpetrator' s emotional arousal and precipitating factors, aggression can be divided into proactive and reactive aggression. These subtypes have important relationships with their underlying mechanisms and behavioral outcomes, yet most studies have not addressed this distinction. (3) Since aggressive behavior is socially undesirable, previous research has predominantly used self-report questionnaires, which may be susceptible to response bias and impression management (Sun & Yang, 2010). Given these limitations, this study will employ

exploratory latent class analysis to investigate childhood adversity among male prisoners and ordinary adults, revealing heterogeneity in adversity experiences across these populations. We will examine MAOA-uVNTR, COMT Val158Met, and 5-HTTLPR polymorphisms, combining experimental methods, questionnaires, and behavioral indicators to investigate the effects of these genetic polymorphisms and childhood adversity on proactive aggression, reactive aggression, and violent criminal behavior.

2.1 Effects of Childhood Adversity and Genes on Aggression

Childhood Adversity (Adverse Childhood Experiences, ACE) refers to actual or potential harm to health, survival, development, or psychological/physical well-being experienced before age 18 (Felitti et al., 1998). Research indicates that childhood adversity experiences contributing to violent aggression include abuse (Lansford et al., 2007; Ford, Fraleigh, & Connor, 2009; Shackman & Pollak, 2014), neglect (Van Wert, Mishna, Trocme, & Fallon, 2017; Li et al., 2010), and family dysfunction (Sternberg, Lamb, Guterman, & Abbott, 2006; Connor et al., 2004; Raine et al., 2006). The more childhood adversity individuals experience, the more frequent their aggressive behavior (Cui et al., 2013; Auslander et al., 2016).

Regarding genetic factors, previous reviews and meta-analyses have identified three important candidate genes influencing aggression: MAOA (Monoamine Oxidase A), COMT (catechol-O-methyltransferase), and 5-HTT (serotonin transporter). The low-activity MAOA-uVNTR allele (hereinafter referred to as the MAOA gene) is associated with aggression (Veroude et al., 2016), violent crime (Tiihonen et al., 2015; Stetler et al., 2014), and antisocial behavior (Ficks & Waldman, 2014). The low-activity Met allele of the COMT Val158Met polymorphism is linked to violent aggression (Wang et al., 2014; Singh et al., 2012; Wang & Zhang, 2010). The S allele of the 5-HTTLPR polymorphism positively correlates with aggression and antisocial behavior (Vassos et al., 2014; Ficks & Waldman, 2014). However, some Chinese sample studies have found no significant relationships between low-activity MAOA alleles (Nie, 2017), COMT Val158Met (Huang et al., 2010), or 5-HTTLPR (Cao et al., 2011) and violent aggression. A meta-analysis by Wang et al. (2014) found that COMT Val158Met polymorphism is associated with violent behavior in Asian schizophrenia patients but not in European or American populations. These discrepant findings suggest that conclusions from Western countries may not directly apply to Chinese populations, necessitating research with Chinese samples. Moreover, contradictory results may stem from systematic differences in environmental factors across samples, with gene-aggression associations potentially existing only in specific environments, making it essential to investigate gene-environment interactions in aggressive behavior.

2.2 Interactive Effects of Childhood Adversity and Genes on Aggression

Recent research has revealed gene-environment (G×E) interactions in aggression (Weeland, Overbeek, de Castro, & Matthys, 2015), particularly between the MAOA gene and childhood maltreatment in influencing aggression or antisocial behavior (including aggression, violence, delinquency, and conduct disorders) (Buades-Rotger & Gallardo-Pujol, 2014; Liu et al., 2017). In a 26-year longitudinal study, Caspi et al. (2002) found no main effect of MAOA genotype, but a significant interaction between MAOA and childhood maltreatment: among males who experienced childhood maltreatment, those carrying the low-activity MAOA allele were more likely to exhibit antisocial and even criminal behavior in adulthood compared to those with high-activity alleles. This finding has been replicated in numerous studies (Reti et al., 2011; Frazzetto et al., 2007) and validated by meta-analyses (Byrd & Manuck, 2014). However, some studies have failed to find G×E interactions between MAOA and childhood adversity (Reif et al., 2007), and one study (Tikkanen et al., 2010) reported opposite results: among males who experienced childhood abuse, those with high-activity MAOA alleles were more likely to commit violent crimes than those with low-activity alleles.

Regarding COMT Val158Met polymorphism, research has found that individuals with Val/Val genotype or Val allele carriers who experienced childhood stress, neglect, or sexual abuse show higher aggression (Hygen et al., 2015; Perroud et al., 2010) and greater probability of violent behavior (Andersson, 2014). However, Wagner et al. (2010) found that among female borderline personality disorder patients with Val/Val genotype, childhood sexual abuse and cumulative serious life events were associated with lower impulsive aggression. Tuvblad et al. (2016) also found that Val/Val carriers showed lower physical aggression when experiencing domestic violence combined with positive parent-child relationships. Additionally, Met allele carriers exhibited higher aggression under conditions of parental divorce or low positive parenting (Zhang, Cao, Wang, Ji, & Cao, 2016; Nederhof, Belsky, Ormel, & Oldehinkel, 2012). Overall, these studies suggest that COMT Val158Met polymorphism interacts with childhood adversity to influence aggression, but fail to clarify which genotype is more susceptible to environmental influence. Research has also found that males carrying the S allele of 5-HTTLPR polymorphism are more likely to exhibit violent aggression after experiencing high adversity or sexual abuse (Reif et al., 2007; Zhang et al., 2012). Meta-analytic results show that 5-HTTLPR polymorphism interacts with childhood adversity in relation to antisocial behavior (including violent aggression), though they do not specify which allele carries higher risk (Tielbeek et al., 2016).

2.3.1 The Necessity of Distinguishing Aggression and Adversity Types

Based on the perpetrator' s emotional arousal and precipitating factors, researchers have further distinguished proactive and reactive aggression (Dodge & Coie, 1987; Wrangham, 2018). Grounded in social learning theory, proactive aggression refers to deliberate, purposeful aggressive acts implemented without provocation. Based on frustration theory, reactive aggression refers to angry, defensive responses to perceived provocation or frustration (Dodge & Coie, 1987). Empirical research demonstrates that proactive and reactive aggressors differ significantly in social cognition, behavior, emotion, personality, family, and peer relationships (Fite, Raine, Stouthamer-Loeber, Loeber, & Pardini, 2010; Dodge & Coie, 1987; Zhou & Feng, 2014; Cao, Wang, Zhang, & Chen, 2012). For instance, in social cognition, proactive aggression is associated with positive expectations about aggressive outcomes (Xu & Zhang, 2008), whereas reactive aggression is associated with hostile attribution bias (Dodge & Coie, 1987). In peer relationships, proactive aggressive individuals hold higher peer status, demonstrating leadership and humor (Dodge & Coie, 1987; Price & Dodge, 1989), while reactive aggressive individuals have lower peer status and are more vulnerable to peer victimization (Xu & Zhang, 2008). Distinguishing between these aggression types is important for legal adjudication, prediction of maladaptive behavior patterns, and prevention and intervention (Ramírez, 2010).

Different types of childhood adversity also exert differential effects on the two aggression types. Overall, reactive aggression is associated with abuse experiences, such as physical abuse (Dodge, Lochman, Harnish, Bates, & Pettit, 1997; Kolla et al., 2013; Ford et al., 2009) and sexual abuse (Connor et al., 2004). In contrast, proactive aggression is associated with family dysfunction, such as parental substance abuse (Connor et al., 2004; Raine et al., 2006), domestic violence (Connor et al., 2004), and parental divorce (Raine et al., 2006). However, many studies have only examined the effect of adversity quantity on aggression, using simple summation or averaging across adversity types (Cui et al., 2013; Auslander et al., 2016). This approach obscures individuals' cumulative and combinative patterns across adversity types and ignores interactions among adversities (Connor et al., 2004; Li et al., 2010). Investigating typical combinations of adversities can more specifically examine how various adversities interactively influence the two aggression types.

2.3.2 Discussion of Analytical Methods for G×E Interactions

As noted above, examining relationships between adversity and aggression requires consideration of adversity types and their interactions. Investigating genetic moderation of these relationships introduces higher-order interactions that make statistical analysis cumbersome and computationally difficult. Con-

sequently, many studies treat adversity as a unidimensional variable when examining G×E interactions, which substantially reduces the variance explained by statistical models. In this study, examining interactions between ten dimensions of childhood adversity and genes would require testing 1,024 (2^1) combinations of adversity presence/absence with each gene, dramatically increasing computational load and the number of statistical tests, thereby reducing statistical power (Lanza & Rhoades, 2013; Merz & Roesch, 2011). Thus, novel methods are needed to address the complex interactions among childhood adversity types and between adversity and genes.

Latent Class Analysis (LCA; Zhang, Jiao, & Zhang, 2010) can explain associations among observed variables through a small number of mutually exclusive latent classes, thereby simplifying complex interactions among manifest variables (Lanza & Rhoades, 2013). In practice, not every combination has substantive meaning, making it necessary to first use LCA to classify individuals into several adversity types based on their experiences. This approach better explains qualitative differences in individuals' adversity profiles (Marsh, Lüdtke, Trautwein, & Morin, 2009) and simplifies subsequent interaction analyses with genes. Moreover, compared to traditional cluster analysis, LCA offers more objective criteria for selecting the number of classes and can estimate classification error, enhancing the accuracy of subsequent statistical models (Zhang, Zhang, & Li, 2017). Previous research has applied LCA to childhood maltreatment experiences and examined differences across criminal behaviors. For example, among male adolescent or adult offenders, emotional/physical abuse and poly-victimization classes show higher recidivism and violence rates than no/low abuse classes (Aebi et al., 2015; Debowska & Boduszek, 2017). Zhang and Zheng (2018) reported similar findings in Chinese offender populations, additionally identifying a high neglect class with more severe Cluster A personality disorder symptoms than the low abuse class, though with lower recidivism and violence rates than the emotional/physical abuse class. These findings demonstrate that childhood adversity experiences exert type-specific and qualitative effects on behavior, and that simple summation of adversity scores or counts obscures the distinct impact patterns of different adversity types. However, these LCA studies have not incorporated family dysfunction—a crucial factor influencing aggression—nor distinguished aggression subtypes, necessitating improved classification indicators and more refined outcome measures.

2.3.3 Discussion of Aggression Measurement Methods

Most previous studies have measured aggression using self-report questionnaires (e.g., Kolla et al., 2013; Connor et al., 2004). This method has limited empirical validity and is susceptible to response bias and impression management (Sun & Yang, 2010). Only a few studies have used competitive reaction time tasks and the hot sauce paradigm to examine gene-reactive aggression relationships (McDermott et al., 2009; Kuepper, Grant, Wielpuetz, & Hennig, 2013). Adapted competitive reaction time tasks can also effectively measure proactive

aggression (Fan, 2017). These experimental paradigms more closely approximate real-world situations and are more likely to elicit reactive aggression (e.g., angry expression or hostile retaliation) and proactive aggression (intimidation, control, risky behavior) (Sun & Yang, 2010). No studies to date have used experimental paradigms to investigate G×E interactions between childhood adversity and genes on aggression. Additionally, using judicial behavioral data as an aggression indicator has important theoretical and practical significance. Research shows that physical abuse, emotional abuse, and sexual abuse are risk factors for violent crime (Brewer-Smyth, Cornelius, & Pickelsimer, 2015; Wang et al., 2014). Childhood maltreatment may lead to violent crime by influencing individuals' reactive aggression. Judicial records serve as important behavioral indicators with high ecological validity, enabling cross-validation of aggressive behavior and further identification of violent crime risk factors, providing crucial empirical evidence for violence prevention.

3 Research Plan

This study will explore the effects and interactions of different childhood adversity types and genes on aggression and violent criminal behavior, with three objectives: (1) to identify common and distinct latent classes of childhood adversity among male prisoners and ordinary populations; (2) to examine the effects of different childhood adversity types on proactive and reactive aggression and the moderating role of genes in these pathways; and (3) to verify the effects of childhood adversity types and genes on violent criminal behavior.

3.1 Study 1: Latent Class Analysis of Childhood Adversity Among Male Prisoners and Ordinary Adults

Study 1 will use latent class analysis to classify childhood adversity among male prisoners and ordinary adults. Participants will include 1,000 ordinary adult males and 600 newly incarcerated adult male prisoners from Guangdong Province, with childhood adversity measured through questionnaires. The ordinary adult sample of 1,000 ensures that each adversity type in Study 2 will have at least 50 participants; if any adversity type in the ordinary population falls below 50, we will expand the ordinary adult sample further. A multi-group latent class model will be established based on scores across ten dimensions of childhood adversity. Empirical LCA studies of childhood adversity have identified low abuse, high emotional/physical abuse, high neglect, sexual abuse, and poly-victimization classes in both populations, with prisoners showing higher proportions in severe adversity classes (Debowska, Willmott, Boduszek, & Jones, 2017). Since family dysfunction influences proactive aggression, this study will incorporate family dysfunction into the adversity classification. Based on this, we propose:

Hypothesis 1: Both male prisoners and ordinary adult populations will exhibit distinct subtypes of childhood adversity, such as low abuse, high neglect, and

poly-victimization classes. Compared to ordinary adults, prisoners will show higher proportions in severe adversity classes.

3.2 Study 2: Effects of Childhood Adversity Types and Genes on Aggression

Building on Study 1' s classification, Study 2 will examine the effects of childhood adversity latent classes and genes on aggression. Participants will complete the Reactive-Proactive Aggression Questionnaire (RPQ; Raine et al., 2006) and a demographic questionnaire, while providing blood samples for genotyping of MAOA-uVNTR, COMT Val158Met, and 5-HTTLPR polymorphisms. Given that prisoners show significantly higher aggression levels than ordinary populations (Zhang et al., 2019; Li, 2008), the data likely represent a mixture distribution. Analyzing both samples together (e.g., using ANOVA or regression) would likely violate normality assumptions (Zhang, 2010). Therefore, this study will test main and interactive effects of genetic polymorphisms and childhood adversity on both aggression types separately within prisoner and ordinary adult samples. Behavioral experiments will then further verify these effects. For the experimental component, 50 participants from each childhood adversity type will complete an adapted competitive reaction time task measuring proactive and reactive aggression. In the proactive aggression experiment, participants are randomly paired with a pseudo-opponent (unbeknownst to them) for an auditory reaction time competition, with winners receiving monetary rewards. One participant (the disruptor) has the privilege to interfere with the opponent during the reaction phase without receiving interference, while the other (the disrupted) has no such privilege and receives interference. Before each round, the disruptor selects an interference level for the opponent. In reality, all participants are assigned the disruptor role. Proactive aggression is assessed by the noise levels participants select under high and low monetary reward conditions (Fan, 2017). In the reactive aggression experiment, participants compete in reaction time races against two pseudo-opponents, with only one opponent appearing per round. The slower responder receives a noise blast from the faster responder. Before each round, participants and opponents select noise punishment levels. In reality, win/loss ratios, sequence, and opponent-selected noise intensity are pre-programmed. Reactive aggression is assessed by noise levels selected under high and low provocation conditions (Krämer, Jansma, Tempelmann, & Münte, 2007).

Research has confirmed that abuse experiences relate to reactive aggression, while family dysfunction relates to proactive aggression (Dodge et al., 1997; Kolla et al., 2013; Connor et al., 2004; Raine et al., 2006). Individuals carrying low-activity MAOA alleles or 5-HTTLPR S alleles show higher aggression after childhood abuse (Byrd & Manuck, 2014; Hygen et al., 2015; Tielbeek et al., 2016). Given contradictory findings regarding COMT Val158Met polymorphism' s interaction with childhood adversity, this study will explore this relationship without specific hypotheses. Based on previous research, we pro-

pose:

Hypothesis 2: Childhood adversity experiences positively predict aggression, with differential effects across adversity types.

Hypothesis 3: Genetic polymorphisms moderate the effects of childhood adversity types on aggression.

3.3 Study 3: Effects of Childhood Adversity Types and Genes on Violent Criminal Behavior

Building on the first two studies, Study 3 will use judicial records to employ crime type as an objective behavioral outcome measure of aggression. Ordinary individuals will be coded as 0, non-violent offenders as 1, and violent offenders as 2, with a multinomial logistic regression model testing main and interactive effects of childhood adversity latent classes and genes on criminal behavior.

Previous research shows that individuals with adversity experiences are more likely to commit violent crimes (Debowska & Boduszek, 2017; Zhang & Zheng, 2018), and most studies find that individuals carrying low-activity MAOA alleles or 5-HTTLPR S alleles are more likely to commit violent crimes after childhood abuse (Byrd & Manuck, 2014; Reif et al., 2007). Based on this, we propose:

Hypothesis 4: Childhood adversity experiences positively predict violent criminal behavior, with differential effects across adversity types.

Hypothesis 5: Genetic polymorphisms moderate the effects of childhood adversity types on violent criminal behavior.

4 Theoretical Framework and Innovation

Over the past half-century, researchers have proposed numerous theories to explain the origins of aggression. Reactive aggression is grounded in frustration-aggression theory (Dollard, Doob, Miller, Mowrer, & Sears, 1939), which posits that frustration increases the likelihood of angry and aggressive responses, with aggression representing angry or defensive reactions to perceived frustration, threat, or provocation. Whether aggression ultimately occurs depends on the individual's interpretation of events and their response to frustration (Berkowitz, 1989). In environments characterized by parental coldness or abuse, individuals tend to attend to negative information in situations (Pollak & Tolley-Schell, 2003; Shackman & Pollak, 2014), miss relevant social cues, and develop hostile attribution biases (Dodge, 2006), making them more prone to reactive aggression (Dodge & Coie, 1987; Vitaro, Brendgen, & Baker, 2006). Proactive aggression is grounded in social learning theory (Bandura, 1973), which posits that aggression can be learned through operant conditioning or observational learning of models, representing a tool-like behavior controlled by expected outcomes. The precipitating factor is positive expectations about behavioral consequences, with proactive aggressive individuals typically expecting to achieve

goals or desires rather than punishment through aggression. Aggressive behavior is a learned social behavior acquired through continuous observation of parental modeling and family environment.

Increasing evidence indicates that aggression is not determined by environmental factors alone but results from interactions between environmental and genetic factors. Based on the diathesis-stress model (Rosenthal, 1963; Bleuler, 1966), individuals carrying certain "risk"or "susceptibility"genes are more vulnerable to adverse environmental influences, leading to psychological or behavioral problems. In other words, problem behavior emerges from the combined effects of individual risk diathesis and negative environmental experiences. Among individuals who experienced childhood abuse or family dysfunction, those carrying such "susceptibility" genes are more likely to exhibit aggressive behavior. As aggression is a complex social behavior, understanding how genetic and environmental factors influence it represents an important research question. This study integrates social learning theory, frustration-aggression theory, the diathesis-stress model, and factors influencing proactive and reactive aggression to examine the mechanisms through which childhood adversity types and genes affect both aggression types, constructing a theoretical framework (see figure below).

Note: Solid lines represent hypothesized relationships based on previous research; dashed lines represent exploratory hypotheses for relationships not previously established.

[Figure 1: see original paper] Theoretical Framework of This Study

First, individuals whose childhood adversity primarily involves abuse (physical, emotional, sexual) and who carry low-activity alleles will show higher reactive aggression. Previous research has found that childhood abuse relates to reactive but not proactive aggression (Dodge et al., 1997; Kolla et al., 2013). Thus, individuals with risk genes who experienced childhood abuse may be more likely to develop hostile attribution biases and show aggressive responses to frustration in adulthood, potentially exhibiting higher reactive aggression. Second, individuals whose childhood adversity primarily involves family dysfunction (domestic violence, familial substance abuse, parental divorce, familial mental illness or criminal records) and who carry low-activity alleles will show higher proactive aggression. Previous research indicates that proactive aggression is more strongly influenced by family factors such as parental substance abuse (Connor et al., 2004; Raine et al., 2006), domestic violence (Connor et al., 2014), and parental divorce (Raine et al., 2006), suggesting that families serve as "models" influencing individual development. Individuals with risk genes exposed to dysfunctional family environments may exhibit higher proactive aggression.

In summary, this study integrates biological-genetic and environmental factors of aggression within a unified theoretical framework, using questionnaires, behavioral experiments, and objective behavioral indicators to examine and validate the effects of genes and childhood adversity types on both aggression

types. The goal is to identify the environmental causes of different aggression types and genetically susceptible populations. Verifying this theoretical framework will help reveal the formation mechanisms of both aggression types from multiple perspectives. Theoretically, this study provides empirical support for G×E interactions in predicting prisoner aggression and offers an interdisciplinary perspective for future aggression research. Methodologically, it incorporates novel statistical models to provide more effective predictive models for aggression. Practically, it aims to identify biological-genetic markers of highly aggressive individuals and detect susceptible populations with childhood adversity experiences, providing theoretical and empirical references for violent behavior risk prediction, behavioral correction interventions, and pharmacological design, thereby improving intervention efficiency and holding important socioeconomic significance.

References

Cao, C., Wang, M., Zhang, W., & Chen, G. (2012). A review of genetic basis of proactive and reactive aggression. *Advances in Psychological Science*, 20(12), 2001–2010.

Cao, Y., Li, L., Zhao, X., & Zhang, Y. (2011). Association analysis of childhood aggressive behavior with COMT Val158Met and 5-HTTLPR polymorphisms. *Chinese Journal of Contemporary Pediatrics*, 13(5), 361–364.

Cui, N., Cao, F., Li, Y., Long, Z., Li, J., & Dong, F. (2013). Cumulative effects of adverse childhood experiences and aggressive behavior in medical freshmen. *Chinese Mental Health Journal*, 27(3), 213–214.

Fan, L. (2017). *Is it worth attacking? The relationship between consideration of attack outcomes and proactive aggression and its fMRI evidence* (Master' s thesis). Southwest University, Chongqing.

Huang, X., Jiang, N., Lin, Z., Deng, H., Chen, R., Cao, L., ···Li, Y. (2010). Association between COMT gene polymorphism and violent aggressive behavior in schizophrenia. *Guangdong Medical Journal*, (1), 82–83.

Li, B., Wang, B., Zhang, J., Zhang, Z., Liu, G., & Hu, J. (2010). Impulsivity, aggressive personality traits in male violent offenders and their relationship with childhood abuse. *Journal of Psychiatry*, 23(2), 119–122.

Li, J. (2008). *Moral emotions in juvenile delinquents and their relationship with social behavior* (Master' s thesis). Shandong Normal University.

Liu, L., Tian, X., Zhang, W., & Wang, M. (2017). MAOA gene-environment interaction on antisocial behavior and its possible neural mechanisms. *Advances in Psychological Science*, 25(6), 970–979.

Nie, A. (2017). *Study on the correlation between MAOA-uVNTR polymorphism and physical aggressive behavior in Yunnan Han and Yi populations* (Master' s thesis). Kunming Medical University.

Sun, L., & Yang, Z. (2010). Experimental research paradigms for aggression. *Psychological Science*, 33(6), 1436–1438.

Wang, M., & Zhang, W. (2010). The relationship between COMT gene polymorphism and aggressive behavior. *Advances in Psychological Science*, 18(8), 1256–1263.

Wang, L., Yang, J., Yang, Z., Zuo, L., Hao, Y., Cao, Y., & Xu, J. (2014). Preliminary investigation of psychosocial factors in violent and property offenders. *Chinese Mental Health Journal*, 28(9), 713–717.

Wang, Y., Wang, J., Gang, Q., Liu, J., & Wang, J. (2014). Meta-analysis of association between COMT gene Val158Met polymorphism and violent behavior in schizophrenia patients. *Chinese Mental Health Journal*, 28(7), 492–498.

Ye, M. (2003). Research methods for aggression. *Psychological Science*, 26(2), 334–336.

Zhang, J., Jiao, C., & Zhang, M. (2010). Application of latent class analysis in psychological research. *Advances in Psychological Science*, 18(12), 1991–1998.

Zhang, J., Zhang, J., Qiu, W., Wen, C., Cui, H., & Jiao, C. (2019). Relationship between mindfulness and aggression in male prisoners. *Chinese Mental Health Journal*, 33(3), 238–240.

Zhang, J., Zhang, M., & Li, G. (2017). Subsequent analysis of latent profile models—improving classification analysis bias. *Psychological Exploration*, 37(5), 434–440.

Zhang, M. (2011). *Principles of Criminal Law*. Beijing: The Commercial Press.

Zhang, M. (2010). *Educational and Psychological Statistics* (3rd ed., pp. 181–182). Beijing: People' s Education Press.

Zhang, W. (2014). *Research on violent behavior in criminal law* (Master' s thesis). Southwest University of Political Science and Law, Chongqing.

Zhang, Y., Ming, Q., Yao, S., & Chen, W. (2012). Moderating effect of serotonin transporter gene polymorphism on the relationship between childhood abuse and adolescent aggression. *Journal of Neuroscience and Mental Health*, 12(6), 541–545.

Zhou, G., & Feng, L. (2014). Distinguishing two types of aggression: Reactive and proactive aggression. *Psychological Development and Education*, 30(1), 108–116.

Aebi, M., Linhart, S., Thunhohenstein, L., Bessler, C., Steinhausen, H. C., & Plattner, B. (2015). Detained male adolescent offender' s emotional, physical and sexual maltreatment profiles and their associations to psychiatric disorders and criminal behaviors. *Journal of Abnormal Child Psychology*, 43(5), 999–1009.

Andersson, A. (2014). *Catechol-o-Methyltransferase (COMT), Earlier Abuse, Gene-Environment Interaction in the Prediction of Violence* (Unpublished master' s thesis). Örebro University.

Auslander, W., Sterzing, P., Threlfall, J., Gerke, D., & Edmond, T. (2016). Childhood abuse and aggression in adolescent girls involved in child welfare: The role of depression and posttraumatic stress. *Journal of Child & Adolescent Trauma*, 9(4), 1.

Bandura, A. (1973). *Aggression: A social learning analysis*. American Journal of Sociology, 26(5), 1-3.

Berkowitz, L. (1989). Frustration-aggression hypothesis: Examination and reformulation. *Psychological Bulletin*, 106(1), 59.

Bleuler, M. (1966). Conception of schizophrenia within the last fifty years and today. *Proceedings of the Royal Society of Medicine*, 56(10), 945-952.

Brewer-Smyth, K., Cornelius, M. E., & Pickelsimer, E. E. (2015). Childhood adversity, mental health, and violent crime. *Journal of Forensic Nursing*, 11(1), 4-14.

Buades-Rotger, M., & Gallardo-Pujol, D. (2014). The role of the monoamine oxidase A gene in moderating the response to adversity and associated antisocial behavior: A review. *Psychology Research and Behavior Management*, 7, 185.

Byrd, A. L., & Manuck, S. B. (2014). MAOA, childhood maltreatment, and antisocial behavior: Meta-analysis of a gene-environment interaction. *Biological Psychiatry*, 75(1), 9-17.

Caspi, A., Mcclay, J., Moffitt, T. E., Mill, J., Martin, J., & Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, 297(5582), 851-854.

Connor, D. F., Steingard, R. J., Cunningham, J. A., Melloni Jr, R. H., & Anderson, J. J. (2004). Proactive and reactive aggression in referred children and adolescents. *American Journal of Orthopsychiatry*, 74(2), 129-136.

Crick, N. R., & Grotpeter, J. K. (1995). Relational aggression, gender, and social-psychological adjustment. *Child Development*, 66(3), 710-722.

Debowska, A., & Boduszek, D. (2017). Child abuse and neglect profiles and their psychosocial consequences in a large sample of incarcerated males. *Child Abuse & Neglect*, 65, 266-277.

Debowska, A., Willmott, D., Boduszek, D., & Jones, A. D. (2017). What do we know about child abuse and neglect patterns of co-occurrence? A systematic review of profiling studies and recommendations for future research. *Child Abuse & Neglect*, 70, 100-111.

Dollard, John, Miller, Neal E, Doob, Leonard W, Mowrer, O. H, & Sears, Robert R. (1939). Frustration and aggression. *American Journal of Sociology*, 92(7),

1654–1667.

Dodge, K. A. (2006). Translational science in action: Hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology*, 18(3), 791–814.

Dodge, K. A., & Coie, J. D. (1987). Social-information-processing factors in reactive and proactive aggression in children's peer groups. *Journal of Personality & Social Psychology*, 53(6), 1146.

Dodge, K. A., Lochman, J. E., Harnish, J. D., Bates, J. E., & Pettit, G. S. (1997). Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. *Journal of Abnormal Psychology*, 106(1), 37.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ···& Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine*, 14(4), 245–258.

Ficks, C. A., & Waldman, I. D. (2014). Candidate genes for aggression and antisocial behavior: A meta-analysis of association studies of the 5HTTLPR and MAOA-uVNTR. *Behavior Genetics*, 44(5), 427–444.

Fite, P. J., Raine, A., Stouthamer-Loeber, M., Loeber, R., & Pardini, D. A. (2010). Reactive and proactive aggression in adolescent males: Examining differential outcomes 10 years later in early adulthood. *Criminal Justice and Behavior*, 37(2), 141–157.

Ford, J. D., Fraleigh, L. A., & Connor, D. F. (2009). Child abuse and aggression among seriously emotionally disturbed children. *Journal of Clinical Child & Adolescent Psychology*, 39(1), 44–54.

Frazzetto, G., Lorenzo, G. D., Carola, V., Proietti, L., Sokolowska, E., & Siracusano, A., et al. (2007). Early trauma and increased risk for physical aggression during adulthood: The moderating role of MAOA genotype. *Plos One*, 2(5), e486.

Hygen, B. W., Belsky, J., Stenseng, F., Lydersen, S., Guzey, I. C., & Wichstrøm, L. (2015). Child exposure to serious life events, COMT, and aggression: Testing differential susceptibility theory. *Developmental Psychology*, 51(8), 1098.

Kolla, N. J., Malcolm, C., Attard, S., Arenovich, T., Blackwood, N., & Hodgins, S. (2013). Childhood maltreatment and aggressive behaviour in violent offenders with psychopathy. *The Canadian Journal of Psychiatry*, 58(9), 487–494.

Kuepper, Y., Grant, P., Wielpuetz, C., & Hennig, J. (2013). MAOA-uVNTR genotype predicts interindividual differences in experimental aggressiveness as a function of the degree of provocation. *Behavioural Brain Research*, 247(12), 73–78.

Krämer, U. M., Jansma, H., Tempelmann, C., & Münte, T. F. (2007). Tit-for-tat: The neural basis of reactive aggression. *Neuroimage*, 38(1), 203-211.

Lansford, J. E., Millerjohnson, S., Berlin, L. J., Dodge, K. A., Bates, J. E., & Pettit, G. S. (2007). Early physical abuse and later violent delinquency: A prospective longitudinal study. *Child Maltreatment*, 12(3), 233-245.

Lanza, S. T., & Rhoades, B. L. (2013). Latent class analysis: An alternative perspective on subgroup analysis in prevention and treatment. *Prevention Science*, 14(2), 157-168.

Marsh, H. W., Lüdtke, O., Trautwein, U., & Morin, A. J. (2009). Classical latent profile analysis of academic self-concept dimensions: Synergy of person-and variable-centered approaches to theoretical models of self-concept. *Structural Equation Modeling*, 16(2), 191-225.

McDermott, R., Tingley, D., Cowden, J., Frazzetto, G., & Johnson, D. D. (2009). Monoamine oxidase A gene (MAOA) predicts behavioral aggression following provocation. *Proceedings of the National Academy of Sciences*, 106(7), 2118-2123.

Merz, E. L., & Roesch, S. C. (2011). A latent profile analysis of the Five Factor Model of personality: Modeling trait interactions. *Personality and Individual Differences*, 51(8), 915-919.

Nederhof, E., Belsky, J., Ormel, J., & Oldehinkel, A. J. (2012). Effects of divorce on Dutch boys' and girls' externalizing behavior in Gene× Environment perspective: Diathesis stress or differential susceptibility in the Dutch Tracking Adolescents' Individual Lives Survey study?. *Development and Psychopathology*, 24(3), 929-939.

Perroud, N., Jaussent, I., Guillaume, S., Bellivier, F., Baud, P., Jollant, F., ···& Courtet, P. (2010). COMT but not serotonin-related genes modulates the influence of childhood abuse on anger traits. *Genes, Brain and Behavior*, 9(2), 193-202.

Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology*, 112(3), 323-338.

Price, J. M., & Dodge, K. A. (1989). Reactive and proactive aggression in childhood: Relations to peer status and social context dimensions. *Journal of Abnormal Child Psychology*, 17(4), 455-472.

Raine, A., Dodge, K., Loeber, R., Gatzke-Kopp, L., Lynam, D., Reynolds, C., ···& Liu, J. (2006). The reactive-proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. *Aggressive Behavior*, 32(2), 159-171.

Ramírez, J. M. (2010). The usefulness of distinguishing types of aggression by function. *International Social Science Journal*, 61(200-201), 263-272.

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

Reti, I. M., Xu, J. Z., Yanofski, J., McKibben, J., Uhart, M., Cheng, Y. J., ···& Kasch-Semenza, L. (2011). Monoamine oxidase A regulates antisocial personality in whites with no history of physical abuse. *Comprehensive Psychiatry*, 52(2), 188–194.

Rosenthal, D. (1963). Theoretical overview: A suggested conceptual framework. In D. Rosenthal (Ed.), *The Genain quadruplets: A case study and theoretical analysis of heredity and environment in schizophrenia* (pp. 505–516). New York: Basic Books Inc.

Shackman, J. E., & Pollak, S. D. (2014). Impact of physical maltreatment on the regulation of negative affect and aggression. *Development & Psychopathology*, 26(1), 1021–1033.

Singh, J. P., Volavka, J., Czobor, P., & Van Dorn, R. A. (2012). A meta-analysis of the Val158Met COMT polymorphism and violent behavior in schizophrenia. *Plos One*, 7(8), e43423.

Sternberg, K. J., Lamb, M. E., Guterman, E., & Abbott, C. B. (2006). Effects of early and later family violence on children' s behavior problems and depression: A longitudinal, multi-informant perspective. *Child Abuse & Neglect*, 30(3), 283–306.

Stetler, D. A., Davis, C., Leavitt, K., Schriger, I., Benson, K., & Bhakta, S., et al. (2014). Association of low-activity MAOA allelic variants with violent crime in incarcerated offenders. *Journal of Psychiatric Research*, 58, 69–75.

Tielbeek, J. J., Karlsson, L. R., Beers, K., Posthuma, D., Popma, A., & Polderman, T. J. (2016). Meta-analysis of the serotonin transporter promoter variant (5-HTTLPR) in relation to adverse environment and antisocial behavior. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 171(5), 748–760.

Tikkanen, R., Ducci, F., Goldman, D., Holi, M., Lindberg, N., Tiihonen, J., & Virkkunen, M. (2010). MAOA alters the effects of heavy drinking and childhood physical abuse on risk for severe impulsive acts of violence among alcoholic violent offenders. *Alcoholism: Clinical and Experimental Research*, 34(5), 853–860.

Tiihonen, J., Rautiainen, M. R., Ollila, H. M., Repo-Tiihonen, E., Virkkunen, M., Palotie, A., ···& Saarela, J. (2015). Genetic background of extreme violent behavior. *Molecular Psychiatry*, 20(6), 786–792.

Tuvblad, C., Narusyte, J., Comasco, E., Andershed, H., Andershed, A. K., Colins, O. F., ···& Nilsson, K. W. (2016). Physical and verbal aggressive behavior

and COMT genotype: Sensitivity to the environment. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 171(5), 708–718.

Vassos, E., Collier, D. A., & Fazel, S. (2014). Systematic meta-analyses and field synopsis of genetic association studies of violence and aggression. *Molecular Psychiatry*, 19(4), 471–477.

Van Wert, M., Mishna, F., Trocme, N., & Fallon, B. (2017). Which maltreated children are at greatest risk of aggressive and criminal behavior? An examination of maltreatment dimensions and cumulative risk. *Child Abuse & Neglect*, 69, 49–60.

Veroude, K., Zhang-James, Y., Fernàndez-Castillo, N., Bakker, M. J., Cormand, B., & Faraone, S. V. (2016). Genetics of aggressive behavior: An overview. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 171(1), 3–43.

Vitaro, F., Brendgen, M., & Barker, E. D. (2006). Subtypes of aggressive behaviors: A developmental perspective. *International Journal of Behavioral Development*, 30(1), 12–19.

Wagner, S., Baskaya, Ö., Anicker, N. J., Dahmen, N., Lieb, K., & Tadić, A. (2010). The catechol o-methyltransferase(COMT) val(158)met polymorphism modulates the association of serious life events (SLE) and impulsive aggression in female patients with borderline personality disorder (BPD). *Acta Psychiatrica Scandinavica*, 122(2), 110–117.

Weeland, J., Overbeek, G., de Castro, B. O., & Matthys, W. (2015). Underlying mechanisms of gene–environment interactions in externalizing behavior: A systematic review and search for theoretical mechanisms. *Clinical Child and Family Psychology Review*, 18(4), 413–442.

Wrangham, R. W. (2018). Two types of aggression in human evolution. *Proceedings of the National Academy of Sciences of the United States of America*, 115(2), 245–253.

Xu, Y., & Zhang, Z. (2008). Distinguishing proactive and reactive aggression in Chinese children. *Journal of Abnormal Child Psychology*, 36(4), 539–552.

Zhang, J., & Zheng, Y. (2018). Childhood maltreatment profiles among incarcerated Chinese males and their associations with personality disorder symptoms and criminal behaviors. *Psychiatry Research*, 268, 272–278.

Zhang, W., Cao, C., Wang, M., Ji, L., & Cao, Y. (2016). Monoamine oxidase a (MAOA) and catechol-o-methyltransferase (COMT) gene polymorphisms interact with maternal parenting in association with adolescent reactive aggression but not proactive aggression: Evidence of differential susceptibility. *Journal of Youth and Adolescence*, 45(4), 812–829.

Note: Figure translations are in progress. See original paper for figures.