·综 述·

# 童年期虐待与单胺氧化酶 A 基因多态性在青少年反社会行为中的交互作用

袁梦园, 苏普玉

安徽医科大学公共卫生学院儿少卫生与妇幼保健学系/出生人口健康教育部重点实验室/人口健康与优生安徽省重点实验室, 合肥, 230032

【摘要】 青少年反社会行为给个人健康、家庭和社会都带来沉重的负担, 引起了公众的广泛关注。目前, 大多数研究已证实童年期虐待、单胺氧化酶 A 基因与反社会行为之间存在关联。然而, 关于童年期虐待与单胺氧化酶 A 基因交互作用与反社会行为之间的关联性尚存在争议。本文旨在对童年期虐待与单胺氧化酶 A 基因交互作用与青少年反社会行为的最新研究成果及其可能的作用机制进行综述, 发现童年期虐待与单胺氧化酶 A 基因可能通过影响大脑中特定的神经环路、改变情绪调节功能等交互作用于反社会行为。此外, 本综述剖析了当前研究结果不一致的可能原因, 为今后研究指明方向, 也为青少年反社会行为的预防控制提供科学依据。

【关键词】 虐待儿童; 基因; 社会行为; 青少年; 精神卫生

【中图分类号】 R 179 G 444 B 844.2 【文献标识码】 A 【文章编号】 1000-9817(2021)05-0792-05

**Research progress for the interaction of child abuse and monoamine oxidase A gene on antisocial behavior among adolescents**

*YUAN Mengyuan, SU Puyu. Department of Maternal, Child and Adolescent Health, School of Public Health, Anhui Medical University/Key Laboratory of Population Health Across Life Cycle, Ministry of Education of the People's Republic of China/Anhui Provincial Key Laboratory of Population Health and Aristogenics, Hefei(230032), China*

**【Abstract】** Antisocial behavior among adolescents caused a severe burden on individuals, families and society, which has aroused a global concern. At present, the association of child abuse and monoamine oxidase A gene with antisocial behavior has been confirmed in most studies. However, there was a controversy about the interaction of child abuse and monoamine oxidase A gene on antisocial behavior. This study aimed to review latest findings and mechanisms of the interaction of child abuse and monoamine oxidase A gene on antisocial behavior among adolescents. The result showed that the child abuse interacted with monoamine oxidase A gene in antisocial behavior by influencing the specific neural circuits in the brain and changing the function of mood regulation, etc. Additionally, this study analyzed potential reasons for the inconsistency of current study findings, and identified the directions for future research. Moreover, this review was to provide a scientific basis for the prevention and control of adolescent antisocial behavior.

**【Keywords】** Child abuse; Genes; Social behavior; Adolescent; Mental health

反社会行为(antisocial behavior, ASB)是指社会活动过程中做出的致使自己、他人与社会遭受伤害或损失的、违反道德规范乃至触犯法律制度的行为[1]。近年来, 青少年反社会行为问题已成为一个重要的公共卫生问题, 引起人们的广泛关注。基于 Boricua Youth Study 队列研究结果显示, 反社会行为指数男性高于女性, 青少年高于儿童[2]。据世界卫生组织(WHO)报道, 全世界每年约有 20 万起凶杀事件发生在 10~29 岁青年人中, 占全球凶杀总数的 42%, 且男性多于女性[3]。国内数据显示, 2018 年未成年人罪犯总人数达 3.4 万人, 占同期犯罪人数的 2.41%, 其中青少年作案人员占全部作案人员的 17.2%[4]。品行障碍是儿童青少年最常见的反社会行为问题, 也是父母寻求儿童青少年精神和心理治疗的常见原因之一。一项研究对 2016 年美国 3~17 岁儿童健康调查(NSCH)数据进行分析发现, 7.4%的儿童青少年有品行问题[5]。品行障碍以严重的反社会性和攻击性为特征, 常与注意力缺陷/多动障碍(attention deficit hyperactivity disorder, ADHD)共存, 可发展为成年后的反社会人格障碍[6]。这些反社会行为均会对个体健康产生长期负面影响, 尤其是与物质滥用或情感症状共存时危害更为严重[7]。此外, 反社会行为还会给社会带来沉重的经济负担, 如仅在美国, 每年因青少年严重攻击行为造成

【基金项目】 国家自然科学基金面上项目(81874268)

【作者简介】 袁梦园(1997- ), 女, 安徽六安人, 在读硕士, 主要研究方向为儿童青少年发育与行为医学。

【通信作者】 苏普玉, E-mail: supuyu@ahmu.edu.cn

DOI: 10.16835/j.cnki.1000-9817.2021.05.036

的经济损失就超过 210 亿美元[8]。

本综述旨在总结国内外学者关于童年期虐待与单胺氧化酶 A(monoamine oxidase A, MAOA)基因多态性在青少年反社会行为中交互作用的最新研究进展,为后期研究提供依据,为青少年反社会行为的预防与控制提供线索。

## 1　童年期虐待与青少年反社会行为

生命早期积极或消极的经历均会对青少年期乃至成年后的行为产生广泛而深远的影响。儿童虐待即对 18 岁以下儿童的虐待与忽视行为,其行为主体通常是对儿童有抚养与监管义务、有操纵权的监护人,一般为父母等近亲属[9];WHO 认为儿童虐待包括躯体虐待、情感虐待、性虐待、忽视和经济上的剥削,威胁儿童的生存、发展、健康和尊严[10]。童年期虐待已成为全球性、普遍性的社会问题,损害早期发育阶段的大脑,增加成年后行为、躯体和精神卫生问题的发生风险[10]。从生物学角度来说,童年期虐待会促进大脑结构的改变,下丘脑-垂体-肾上腺轴的不典型发育[11-13]。研究发现,在情绪处理任务中,早期经历过虐待的个体皮质下边缘区域呈高反应性,同时额叶前部调节区的激活减少[14-16],目前这种失调的神经激活模式已在反社会型人格障碍患者中得到证实[17-18]。

## 2　*MAOA* 基因多态性与青少年反社会行为

*MAOA* 是一种存在于大多数哺乳动物中,由 X 染色体上基因编码的酶,可代谢中枢神经系统中与攻击性有关的单胺类神经递质(如 5-羟色胺和多巴胺等)[19-20]。人类 *MAOA* 基因中被研究最为广泛的区域是位于上游的可变数目串联重复序列(*MAOA-uVNTR*),由多倍重复的 30 个碱基对(30bp)组成的功能多态性区域,研究表明其可调节 *MAOA* 基因的转录效率[21]。通常有 2,3,3.5,4,5 倍重复,频率因种族而异,但 3~4 倍重复最常见[21-24]。一般来说,具有 3.5 和 4 倍重复序列的等位基因显示出高转录水平和高 *MAOA* 活性(*MAOA-H*),而 2 和 3 倍重复序列则显示低 mRNA 表达、低 *MAOA* 活性(*MAOA-L*),并且 *MAOA-L* 可能与单胺水平升高有关[21],关于 5 倍重复等位基因的转录效率研究结果仍存在争议[21,25]。首次将 *MAOA* 基因与攻击行为联系起来的是基于对荷兰一个特殊家族(男性 *MAOA* 基因发生无义突变)的观察,随后的动物实验(*MAOA* 基因敲除鼠)进一步佐证了该假说[26-27];但由于人类完全缺乏 *MAOA* 的情形很罕见,所以不能在人群中复制 *MAOA* 基因敲除鼠的动物实验研究结果。由于女性有一条 X 染色体永久不完全失活(随机)[28],*MAOA-uVNTR* 基因型的功能分类尚不清楚,因此大多数研究只开展了男性研究。

## 3　童年期虐待与 *MAOA* 基因多态性交互作用对青少年反社会行为的影响

基因-环境交互作用指个体应对刺激和环境的能力可能因其基因型组成不同而存在差异。2002 年,Caspi 等[29]首次发表了与人类攻击行为有关的基因-环境交互作用的研究,结果表明,经历过童年期虐待的男性中 *MAOA-L* 等位基因携带者发生反社会行为的风险增加。随后,恒河猴实验也进一步证实了 *MAOA* 基因多态性与不良养育环境交互作用可影响攻击性行为的产生[30-31]。Choe 等[32]在一个由高加索和非裔美国人组成的男性队列中,控制早期外化行为后,发现 1.5,2 和 5 岁时父母惩罚性管教与 *MAOA-L* 交互作用可增加 15~20 岁时反社会行为的发生风险。一项横断面研究证实了 *MAOA-uVNTR* 与家庭冲突或性虐待存在交互作用的同时,还发现了 3 个候选基因(*MAOA-uVNTR*、*5-HTTLPR* 和 *BDNF Val66Met*)之间的多基因-环境交互作用对犯罪行为的影响[33]。Zhang 等[34]在中国进行的一项队列研究表明,经历过童年期虐待(躯体虐待、情感虐待和性虐待)的男性 *MAOA-L* 携带者在青春期和成年早期更容易表现出攻击行为,其中早期虐待存在着主效应作用[33-34],*MAOA* 基因多态性不存在主效应作用[32-34]。然而,一项前瞻性研究表明,女性 *MAOA-uVNTR* 作用的方向与男性相反,风险等位基因为 *MAOA-H*,即女性 *MAOA-HH* 基因型与躯体虐待交互作用可预测品行障碍[35]。

## 4　童年期虐待与 *MAOA* 基因多态性交互作用对青少年反社会行为影响的可能机制

反社会行为具有特定的神经生物学、心理和行为学基础。研究表明,认知、情绪等与反社会行为之间存在关联,并且大脑中皮质-边缘环路在调节反社会行为中起到了关键作用[36]。越来越多的研究表明,早期虐待严重影响这一神经环路的发育和功能[37-38]。*MAOA* 在生物胺的代谢中起主要作用,包括 5-羟色胺、去甲肾上腺素和多巴胺等参与压力和情绪调节的神经递质[39]。近期在一项女性队列研究发现,*MAOA* 与早期虐待交互可通过增加青春期情绪反应性来作用于反社会人格障碍[40]。有证据表明,情绪反应性的增加与皮质边缘回路的潜在缺陷有关,特别是杏仁核、纹状体和前额叶皮质,情绪调节在很大程度上依赖于结构的动态相互作用[41-42]。脑成像研究表明,*MAOA-L* 等位基因(男性)与对情绪刺激的高反应性、前额叶调节区的参与减少以及调节情绪处理的皮质-边缘回路紊乱有关[43]。一项神经影像学研究发现,

*MAOA* 和虐待之间的相互作用与皮质边缘神经回路内的功能改变有关,而这些功能的异常与反社会行为风险的增加有关[44]。并且,*MAOA* 基因型在预测不良情绪调节能力上存在着性别差异,即经历过虐待的 *MAOA-H* 等位基因女性携带者和 *MAOA-L* 等位基因男性携带者在情绪处理过程中杏仁核激活程度较高[44]。

*MAOA* 易感基因型存在性别差异的可能机制,一方面 *MAOA* 基因位于 X 染色体上,尽管女性中有一条 X 染色体随机失活,但有研究表明 *MAOA* 基因可能逃避失活从而产生不同程度的表达谱[45];另一方面动物研究表明,*MAOA* 启动子中的 CpG 残基甲基化程度在性别之间差异很大[46]。此外,有证据表明 *MAOA* 基因多态性与外源性睾酮交互作用可影响男性的攻击性[47]。

上述结果表明,童年期虐待经历可能通过影响发育中大脑特定的神经环路及结构,导致情绪调节功能失调,从而进一步增加了反社会行为的发生风险。此外,大脑中神经元之间联系是由突触处的神经递质所介导的,因此单胺氧化酶 A 可能通过调节与压力和情绪调节相关神经递质的降解参与该过程。由于女性比男性多一条 X 染色体且性激素种类及水平存在差异,可能是导致 *MAOA* 风险基因型在性别之间不一致的原因。

## 5 小结

综上所述,目前基本达成一致的观点是,男性 *MAOA-L* 基因型与童年期虐待经历在预测青少年期反社会行为方面存在着交互作用,即经历过童年期虐待的 *MAOA-L* 携带者在青少年期发生反社会行为的风险更大。另外,关于女性 *MAOA* 风险等位基因的研究证据尚不足。此外,童年期虐待对青少年反社会行为的预测具有主效应作用,而 *MAOA* 基因型则无主效应作用。童年期虐待与 *MAOA* 基因型交互作用可以通过改变情绪调节相关神经环路的功能增加青少年期反社会行为发生的风险,揭示了其中部分生物学机制。因此,加强早期对童年期虐待的识别与控制对预防青少年反社会行为的发生具有积极意义。

但是,近年来也有研究否定了 *MAOA* 基因多态性与童年期虐待交互作用对青少年反社会行为的影响[48]。造成结果不一致的原因可能是:未考虑到多基因-环境模型[49];环境因素的测量及定义不同,如早期逆境的累积效应、暴露程度[50];反社会行为与其他行为障碍存在着共病性和表型交叉等。

## 6 展望

当前研究表明,单个基因对反社会行为的影响相对较小,需要进一步探讨多基因交互作用及多基因与环境交互作用在行为问题与精神障碍领域中的地位。早期生活环境无逆境并不等同于生活于积极的环境,因此同时对积极和消极的环境因素进行测量是十分必要的[51]。最近一项表观遗传学研究表明,*MAOA* ROI(跨越 *MAOA* 第一外显子和部分第一内含子的感兴趣区域)甲基化水平进一步促进了 *MAOA* 风险基因型和虐待对男性攻击行为的交互作用[52]。未来的机制研究尚需进一步关注表观遗传学方面,如不同环境中行为与精神障碍相关易感基因甲基化水平及转录效率的改变。

## 7 参考文献


[1] 吕鹏. 基于预测视角的反社会行为谱系类型与研究述评[J]. 晋阳学刊, 2020,41(2):104-114.
LYU P. The genealogy type and research review of antisocial behavior from the perspective of prediction[J]. Acad J Jinyang, 2020,41(2):104-114.

[2] DUARTE C S, KLOTZ J, ELKINGTON K, et al. Severity and frequency of antisocial behaviors:late adolescence/young adulthood antisocial behavior index[J]. J Child Fam Stud, 2020,29(12):1200-1211.

[3] World Health Organization. Youth violence[EB/OL].[2020-09-28]. http://www.who.int/news-room/fact-sheets/detail/youth-violence.

[4] 国家统计局. 2018 年《中国儿童发展纲要(2011—2020 年)》统计监测报告[EB/OL].[2020-07-01]. http://www.stats.gov.cn/tjsj/zxfb/201912/t20191206_1715751.html.
National Bureau of Statistics of the People's Republic of China.Statistical monitoring report on the program for the development of Chinese children (2011-2020) in 2018[EB/OL].[2020-07-01]. http://www.stats.gov.cn/tjsj/zxfb/201912/t20191206_1715751.html.

[5] GHANDOUR R M, SHERMAN L J, VLADUTIU C J, et al. Prevalence and treatment of depression, anxiety, and conduct problems in US children[J]. J Pediatr, 2019, 206: 256-267. DOI: 10.1016/j.jpeds.2018.09.021.

[6] FAIRCHILD G, HAWES D J, FRICK P J, et al. Conduct disorder[J]. Nat Rev Dis Prim, 2019,5(1):43.

[7] KOELCH M G, DÖPFNER M, FREITAG C M, et al. Conduct disorder and antisocial personality disorders:challenges for treatment in adolescence and young adulthood[J]. Fortschr Neurol Psychiatr, 2019, 87(11):634-637.

[8] CASTILLO-EITO L, ARMITAGE C J, NORMAN P, et al. How can adolescent aggression be reduced? A multi-level meta-analysis[J]. Clin Psychol Rev, 2020,78.DOI:10.1016/j.cpr.2020.101853.

[9] JIN J. Interventions to prevent child maltreatment[J]. JAMA, 2018, 320(20):2160.

[10] World Health Organization. Child maltreatment[EB/OL].[2020-09-28]. https://www.who.int/news-room/fact-sheets/detail/child-maltreatment.

[11] KUHLMAN K R, GEISS E G, VARGAS I, et al. HPA-axis activation as a key moderation of childhood trauma exposure and adolescent mental health[J]. J Abnorm Child Psychol, 2018,46(1):149-157.

[12] DAHMEN B, PUETZ V B, SCHARKE W, et al. Effects of early-life adversity on hippocampal structures and associated HPA axis functions [J]. Dev Neurosci, 2018, 40(1): 13-22.

[13] NEMEROFF C B. Paradise Lost: the neurobiological and clinical consequences of child abuse and neglect[J]. Neuron, 2016, 89(5): 892-909.

[14] MCCRORY E J, DE BRITO S A, KELLY P A, et al. Amygdala activation in maltreated children during pre-attentive emotional processing [J]. Br J Psychiatry, 2013, 202(4): 269-276.

[15] SUZUKI H, LUBY J L, BOTTERON K N, et al. Early life stress and trauma and enhanced limbic activation to emotionally valenced faces in depressed and healthy children[J]. J Am Acad Child Adolesc Psychiatry, 2014, 53(7): 800-813.

[16] VAN HARMELEN A L, VAN TOL M J, DALGLEISH T, et al. Hypoactive medial prefrontal cortex functioning in adults reporting childhood emotional maltreatment[J]. Soc Cogn Affect Neurosci, 2014, 9(12): 2026-2033.

[17] HYDE L W, BYRD A L, VOTRUBA-DRZAL E, et al. Amygdala reactivity and negative emotionality: divergent correlates of antisocial personality and psychopathy traits in a community sample[J]. J Abn Psychol, 2014, 123(1): 214-224.

[18] HYDE L W, SHAW D S, MURRAY L, et al. Dissecting the role of amygdala reactivity in antisocial behavior in a sample of young, low-income, urban men[J]. Clin Psychol Sci, 2016, 4(3): 527-544.

[19] BORTOLATO M, FLORIS G, SHIH J C. From aggression to autism: new perspectives on the behavioral sequelae of monoamine oxidase deficiency[J]. J Neural Transm (Vienna), 2018, 125(11): 1589-1599.

[20] GODAR S C, FITE P J, MCFARLIN K M, et al. The role of monoamine oxidase A in aggression: current translational developments and future challenges[J]. Prog Neuropsychopharmacol Biol Psychiatry, 2016, 69: 90-100. DOI: 10.1016/j.pnpbp.2016.01.001.

[21] SABOL S Z, HU S, HAMER D. A functional polymorphism in the monoamine oxidase A gene promoter[J]. Hum Genet, 1998, 103(3): 273-279.

[22] RODRIGUEZ-RAMOS A, MORIANA J A, GARCIA-TORRES F, et al. Emotional stability is associated with the MAOA promoter uVNTR polymorphism in women[J]. Brain Behav, 2019, 9(9): e1376.

[23] GUO G, OU X M, ROETTGER M, et al. The VNTR 2 repeat in MAOA and delinquent behavior in adolescence and young adulthood: associations and MAOA promoter activity[J]. Eur J Hum Genet, 2008, 16(5): 626-634.

[24] HUANG Y Y, CATE S P, BATTISTUZZI C, et al. An association between a functional polymorphism in the monoamine oxidase a gene promoter, impulsive traits and early abuse experiences[J]. Neuropsychopharmacology, 2004, 29(8): 1498-1505.

[25] BEACH S R H, BRODY G H, GUNTER T D, et al. Child maltreatment moderates the association of MAOA with symptoms of depression and antisocial personality disorder[J]. J Fam Psychol, 2010, 24(1): 12-20.

[26] GODAR S C, FITE P J, MCFARLIN K M, et al. The role of monoamine oxidase A in aggression: current translational developments and future challenges[J]. Pro Neuropsychopharmacol Biol Psychiatry, 2016, 69: 90-100. DOI: 10.1016/j.pnpbp.2016.01.001.

[27] BORTOLATO M, GODAR S C, TAMBARO S, et al. Early postnatal inhibition of serotonin synthesis results in long-term reductions of preservative behaviors, but not aggression, in MAO A-deficient mice [J]. Neuropharmacology, 2013, 75: 223-232. DOI: 10.1016/j.neuropharm.2013.07.003.

[28] GALUPA R, HEARD E. X-chromosome inactivation: a crossroads between chromosome architecture and gene regulation[J]. Annu Rev Genet, 2018, 52: 535-566. DOI: 10.1146/annurev-genet-120116-024611.

[29] CASPI A, MCCLAY J, MOFFITT T E, et al. Role of genotype in the cycle of violence in maltreated children[J]. Science, 2002, 297(5582): 851-854.

[30] NEWMAN T K, SYAGAILO Y V, BARR C S, et al. Monoamine oxidase a gene promoter variation and rearing experience influences aggressive behavior in rhesus monkeys[J]. Biol Psychiatry, 2005, 57(2): 167-172.

[31] KARERE G M, KINNALLY E L, SANCHEZ J N, et al. What is an "adverse" environment? Interactions of rearing experiences and MAOA genotype in rhesus monkeys[J]. Biol Psychiatry, 2009, 65(9): 770-777.

[32] CHOE D E, SHAW D S, HYDE L W, et al. Interactions between monoamine oxidase a and punitive discipline in African American and Caucasian men's antisocial behavior[J]. Clin Psychol Sci, 2014, 2(5): 591-601.

[33] NILSSON K W, COMASCO E, HODGINS S, et al. Genotypes do not confer risk for delinquency but rather alter susceptibility to positive and negative environmental factors: gene-environmentinteractions of BDNF Val66Met, 5-HTTLPR, and MAOA-uVNTR[J]. Int J Neuropsychopharmacol, 2014, 18(5): 107.

[34] ZHANG Y, MING Q, WANG X, et al. The interactive effect of the MAOA-VNTR genotype and childhood abuse on aggressive behaviors in Chinese male adolescents[J]. Psychiatr Genet, 2016, 26(3): 117-123.

[35] MCGRATH M L, MUSTANSKI B, METZGER A, et al. A latent modeling approach to genotype-phenotype relationships: maternal problem behavior clusters, prenatal smoking, and MAOA genotype [J]. Arch Womens Ment Health, 2012, 15(4): 269-282.

[36] EISNER P, KLASEN M, WOLF D, et al. Cortico-limbic connectivity in MAOA-L carriers is vulnerable to acute tryptophan depletion[J]. Hum Brain Mapp, 2017, 38(3): 1622-1635.

[37] TEICHER M H, ANDERSEN S L, POLCARI A, et al. The neurobiological consequences of early stress and childhood maltreatment[J]. Neurosci Biobehav Rev, 2003, 27(1/2): 33-44.

[38] TOTTENHAM N. The importance of early experiences for neuro-affective development[J]. Curr Top Behav Neurosci, 2014, 16: 109-129. DOI: 10.1007/7854_2013_254.

[39] WILLIAMS L M, GATT J M, KUAN S A, et al. A polymorphism of the MAOA gene is associated with emotional brain markers and personality traits on an antisocial index[J]. Neuropsychopharmacology, 2009, 34(7): 1797-1809.

[40] BYRD A L, MANUCK S B, HAWES S W, et al. The interaction between monoamine oxidase A (MAOA) and childhood maltreatment as a predictor of personality pathology in females: Emotional reactivity as a potential mediating mechanism[J]. Dev Psychopathol, 2018: 1-17. DOI: 10.1017/s0954579418000238.

(下转第 800 页)

[31] MCLOUGHLIN L T, LAGOPOULOS J, HERMENS D F. Cyberbullying and adolescent neurobiology [J]. Front Psychol, 2020, 11 (1511): 1-7.

[32] MACKEY W, CONEY N. The enigma of father presence in relationship to sons' violence and daughters' mating strategies: empiricism in search of a theory[J]. J Mens St, 2000, 8(3): 349-373.

[33] GALLUP A C, O'BRIEN D T, WHITE D D, et al. Peer victimization in adolescence has different effects on the sexual behavior of male and female college students[J]. Pers Ind Diff, 2009, 46(5-6): 611-615.

[34] TIMEO S, RIVA P, PALADINO M P. Learning to cope with everyday instances of social exclusion: a review of emotional and cognitive strategies for children and adolescents[J]. J Appl Behav Res, 2019, 24(4): 73-90.

[35] SHAALAN F, SIMON E. Narrative counselling for adolescents at risk of exclusion from school[J]. Br J Guid Couns, 2020. DOI: 10.1080/03069885.2020.1729342.

[36] MULVEY K L, BOSWELL C, ZHENG J. Causes and consequences of social exclusion and peer rejection among children and adolescents [J]. Emot Behav Dis Youth, 2017, 17(3): 71-75.

[37] FARRINGTON D P, TTOFI M M. School-based programs to reduce bullying and victimization[J]. Campb System Rev, 2009, 6: 13-47. DOI: 10.4073/csr.2009-6.

[38] YANG A, SALMIVALLI C. Effectiveness of the KiVa antibullying programme on bully-victims, bullies and victims [J]. Educ Res, 2015, 57(1): 80-90.

[39] VALDEBENITO S, EISNER M, FARRINGTON D P, et al. School-based interventions for reducing disciplinary school exclusion: a systematic review[J]. Campb Syst Rev, 2018, 14(1): 35-40.

[40] OBSUTH I, SUTHERLAND A, COPE A, et al. London Education and Inclusion Project (LEIP): results from a cluster-randomized controlled trial of an intervention to reduce school exclusion and antisocial behavior[J]. J Youth Adolesc, 2016, 46(3): 538-557.

[41] KILLEN M. Developing inclusive youth how to reduce social exclusion and foster equality and equity in childhood[J]. Am Educ, 2019, 43 (3): 8-12.

[42] DEMOL K, LEFLOT G, VERSCHUEREN K, et al. Revealing the transactional associations among teacher-child relationships, peer rejection and peer victimization in early adolescence[J]. J Youth Adolesc, 2020, 15. DOI: 10.1007/s10964-020-01269-z

[43] ALEX S, JO R. You kind of don't want them in the room: tensions in the discourse of inclusion and exclusion for students displaying challenging behaviour in an English secondary school [J]. Int J Incl Educ, 2018: 1-15. DOI: 10.1080/13603116.2018.1516821.

[44] 王玥. 心理学视域下校园欺凌的形成机理及对策[J]. 北京师范大学学报(社会科学版), 2019(4): 32-45.
WANG Y. Formation mechanism and intervention measures of school bullying from the psychological perspective[J]. J Beijing Normal U (Soc Sci), 2019(4): 32-45.

[45] 陈光辉, 杨晓霞, 张文新. 芬兰反校园欺凌项目 KiVa 及其实践启示[J]. 中国特殊教育, 2018(9): 80-85.
CHEN G H, YANG X X, ZHANG W X. The anti-bullying program KiVa in finland and its implications[J]. Chin J Sp Educ, 2018(9): 80-85.

[46] KARNA A, VOETEN M, LITTLE D, et al. Going to scale: a nonrandomized nationwide trial of the KiVa Antibullying Program for Grades 1-9[J]. J Cons Clin Psychol, 2012, 79(6): 796-805.

**收稿日期**: 2020-09-13 **修回日期**: 2020-10-07 **本文编辑**: 王苗苗

(上接第 795 页)

[41] CASEY B J, HELLER A S, GEE D G, et al. Development of the emotional brain[J]. Neurosci Lett, 2019, 693: 29-34. DOI: 10.1016/j.neulet.2017.11.055.

[42] LATHAM M D, COOK N, SIMMONS J G, et al. Physiological correlates of emotional reactivity and regulation in early adolescents[J]. Biol Psychol, 2017, 127: 229-238. DOI: 10.1016/j.biopsycho.2017.07.018.

[43] MEYER-LINDENBERG A, BUCKHOLTZ J W, KOLACHANA B, et al. Neural mechanisms of genetic risk for impulsivity and violence in humans[J]. Proc Natl Acad Sci USA, 2006, 103(16): 6269-6274.

[44] HOLZ N, BOECKER R, BUCHMANN A F, et al. Evidence for a sex-dependent MAOA× childhood stress interaction in the Neural circuitry of aggression[J]. Cereb Cortex, 2016, 26(3): 904-914.

[45] KOLLA N J, VINETTE S A. Monoamine oxidase A in antisocial personality disorder and borderline personality disorder[J]. Curr Behav Neurosci Rep, 2017, 4(1): 41-48.

[46] KONAR A, RASTOGI M, BHAMBRI A. Brain region specific methylation and Sirt1 binding changes in MAOA promoter is associated with sexual dimorphism in early life stress induced aggressive behavior[J]. Neurochem Int, 2019, 129: 104510. DOI: 10.1016/j.neuint.2019.104510.

[47] WAGELS L, VOTINOV M, KELLERMANN T, et al. Exogenous testosterone and the monoamine-oxidase A polymorphism influence anger, aggression and neural responses to provocation in males [J]. Neuropharmacology, 2019, 156: 107491. DOI: 10.1016/j.neuropharm.2019.01.006.

[48] RUISCH I H, DIETRICH A, GLENNON J C, et al. Interplay between genome-wide implicated genetic variants and environmental factors related to childhood antisocial behavior in the UK ALSPAC cohort [J]. Eur Arch Psychiatry Clin Neurosci, 2019, 269(6): 741-752.

[49] ZHANG Y, MING Q, YI J, et al. Gene-gene-environment interactions of serotonin transporter, monoamine oxidase a and childhood maltreatment predict aggressive behavior in Chinese adolescents[J]. Front Behav Neurosci, 2017, 11: 17. DOI: 10.3389/fnbeh.2017.00.017.

[50] OUELLET-MORIN I, COTE S M, VITARO F, et al. Effects of the MAOA gene and levels of exposure to violence on antisocial outcomes [J]. Br J Psychiatry, 2016, 208(1): 42-48.

[51] NILSSON K W, ÅSLUND C, COMASCO E, et al. Gene-environment interaction of monoamine oxidase A in relation to antisocial behaviour: current and future directions[J]. J Neural Transm, 2018, 125(11): 1601-1626.

[52] CHECKNITA D, BENDRE M, EKSTRÖM T J, et al. Monoamine oxidase A genotype and methylation moderate the association of maltreatment and aggressive behaviour[J]. Behav Brain Res, 2020, 382: 112476. DOI: 10.1016/j.bbr.2020.112476.

**收稿日期**: 2020-07-08 **修回日期**: 2020-09-20 **本文编辑**: 顾璇