Original Article

Iraqi National Journal of Medicine Jan 2021, vol. 3, Issue 1

Antisocial Behavior Etiologies

Sundus Fadhil Hantoosh[1], Rasha Sadeq Ameen[1], Seenaa S. Amin[2]
[1] Training and Development Department, Forensic DNA Center for Research and Training/ University of AL-Nahrain, Baghdad, Iraq.
[2] Department of Clinical Laboratory Science, College of Pharmacy, University of Baghdad, Baghdad, Iraq.

ABSTRACT

Antisocial behavior is a broad term that encompasses many facets of destructive behavior, most of which bring harm to another person or involve the violation of rights of others. Main antisocial behavior victims are young individuals, women and children. Numerous factors interact together for the development of aggression and antisocial behavior; these factors are social, environmental, physiological, neurological, and genetic. Consequently, this paper addresses the principal etiologic factors that participate in the development of antisocial behaviors for children, adolescents, and adult individuals**.**

Keywords: antisocial behavior, predatory, aggression, violent.

Corresponding author: Sundus Fadhil Hantoosh Email: sundus.alnahi@gmail.com
Disclaimer: The authors has no conflict of interest.

DOI: https://doi.org/10.37319/iqnjm.3.1.6
Received: 25 Aug 2020 Accepted: 13 Nov 2020 Published online: 15 Jan 2021

Introduction

Crime proves to be a major burden on modern society. The heterogeneous nature of criminal behavior makes it difficult to unravel the causes behind such actions. A lot of researches suggest that many factors (genetic and environment) play a role in antisocial behavior of a person. The World Health Organization (WHO) refers to violence as being a main public health issue (1) and defines it to be "the intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in or has a high likelihood of injury, death, psychological harm, maldevelopment, or deprivation" (2). In other words, aggression means a behavior intentionally causing physical and/or psychological harm to individuals or property, and it is a serious issue of social and clinical importance (3). Antisocial, violent, or aggressive persons act on impulse, which indicates that they do not think before acting; possibly, there are deficiencies in their thinking ability because of interpersonal cognitive issues, and they have an extremely egoistic behavior. As a result, such persons reveal little sympathy towards others and face difficulty in understanding what other individuals are thinking or agreeing that others may have different perspectives (4). Such cognitive deficiencies lead to a prejudice situation, making people more vulnerable to respond violently and aggressively, and even commit crimes (5).

Children with depressed mothers reveal growing emotional and behavioral issues, which involve antisocial behavior (6); this leads to families having poor interactions, stressful family contexts, and unsuitable parenting (7). In human beings, two principal subtypes of aggression are known: impulsive-reactive-hostile-affective (impulsive) and a controlled-proactive-instrumental-predatory (controlled); these subtypes vary from each other in quality with respect to their phenomenology and neurobiological characteristics (8). Impulsive aggression is uncontrolled, excitable, joined by fear or irritation, identified by high arousal levels, and even self-destructive (9). In the case of non-impulsive and controlled (predatory) aggression subtype, people are known to be unstable, with instrumental aggression that is ordinarily used to achieve a target beyond hurting a victim. The arousal level in these cases is low, as shown by their low baseline heart rate and skin conductance levels (3). It is proposed that child psychiatric cases with aggressive behavioral issues represent impulsive aggression more than controlled aggression (8). However, limited research has been conducted on the factors influencing criminal behavior; understanding these behaviors could ultimately inform and improve current treatment strategies of antisocial behavior. To fill this gap somewhat, this paper will talk about the main etiologies of antisocial behavior.

Risk Factors for Antisocial Behavior:

Malnutrition

Malnutrition is increasingly recognized as a risk factor for children's externalizing behaviors, which include aggression, hyperactivity, delinquency, conduct disorder, and antisocial personality disorder. It is hypothesized that malnutrition can interfere with brain functionality by diminishing neuronal

growth and development of the brain, altering neurotransmitter functions, increasing neurotoxicity, and impairing cognitive functions. Adequate nutrition is crucial for normal brain growth (10). Nutrition is crucial during pregnancy and infancy, which are considerable periods for the formation of the brain and development of essential skills (i.e., cognitive, motor, and socio-emotions) during childhood and adulthood. Adequate proportions of various essential nutrients is crucial for ideal brain functioning, while poor nutrition exhibits decreased neuropsychological work and adversely impacts synaptic flexibility. Deficiency in neuropsychological functioning can decrease self-control significantly and elevates the risk of misconduct (11).

Extreme physical offense and externalizing attitude levels in early infancy considerably elevate the likelihood of perpetrator actions in the adolescence stage (12). Fergusson et al. (13) demonstrated that bullying and externalizing actions in the infancy were related to severe assault during adulthood. Moreover, it is important to understand that the quality of food in the diet, or differences in the frequency of certain groups of foods eaten, could result in the development of antisocial behaviors during childhood (11).

It was found that reactive hypoglycemia was common in criminals and delinquents (14). A study measured oral glucose tolerance test (OGTT) on a number of fierce culprits and controlled subjects. Violent culprits who were diagnosed as suffering from antisocial personality or intermittent explosive disorders had significantly lower blood glucose than those that were controlled or those fierce culprits diagnosed without antisocial personality or intermittent explosive disorders. This explains that enhanced insulin secretion is a causative for violent behavior, especially with subjects considered as alcoholic, as alcohol potentiates insulin release (15). However, the study performed by Oh et al. (16) demonstrated that high ingestion of sweets during childhood considerably elevates incidence of antisocial behavior disorder.

It was noted that taking polyunsaturated fatty acids is crucial for brain functions (17). A double-blind, placebo-controlled, and randomized experimental trial documented that supplementation of adult prisoners' food with essential fatty acids considerably decreased antisocial and violent behavior (18). Animal studies presented the view that deprivation of omega-3 fatty acids at stringent growth times does not only decrease synaptic differentiation and formation, but also elevates aggression by adversely changing concentrations of serotonin (19). Woo et al. (20) mentioned that low-fat diet and diet with elevated concentrations of fatty acids and minerals decreased the chances of promoting attention deficit hyperactivity disorder (ADHD), while snacks enriched with candy and sowbread had the opposite effect.

It was recognized that protein deficits impairs brain growth and makes individuals vulnerable to aggressive behavior (21). Additionally, other studies showed that antisocial behavior is correlated with protein deficiency, which contributes to

brain impairment and, consequently, predisposes antisocial behavior disorder, and as proteins are synthesized principally from amino acids, it is important to acknowledge that diet poor in amino acid tryptophan may lead to high rates of aggressive and antisocial behavior (22,23,24).

Iodine is crucial for thyroid hormones productions, which are essential for central nervous system (CNS) growth. Severe iodine deficiency in pregnant females results in the underproduction of thyroid hormones, thus resulting in cretinism in the children. Cretinism disorder is characterized by mental retardation, facial deformities, and huge stunted development (10). It was proposed by Bath et al. (25) that even mild iodine deficiency in the first trimester of pregnancy negatively affected children's cognition eight years later (25).

Additionally, iron is a crucial component of hemoglobin; it transports oxygen to all body organs, including the brain. Iron deficiency anemia (IDA) is a disease characterized by iron deficiency individuals; it leads to hemoglobin underproduction, which is a risk factor for developing cognitive impairment (10). Lozoff et al. (26) revealed that anemic children, who suffered the disease during the first two years of their life, continued to underperform in schools and suffered cognitive disorders from the age of four to nineteen. There is evidence in animal studies that iron deficiency was responsible for development of aggressive attributes (21). It was also noted that iron deficiency contributed to brain impairments present in adult offenders, which then resulted in antisocial behavior disorder (23).

The ion, zinc, is plenteous in the brain; it is involved in the brain structure and function via its contribution in deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) synthesis and its efficacy in carbohydrate, fat, and protein metabolism (27). It was observed that deficiency of zinc resulted in aggressive antisocial behavior (21).

Genetic Factors

Genetic factors also contribute to individual differences in antisocial behavior. Behavioral genetic research relies on the different levels of genetic relatedness between family members in order to estimate the contribution of heritable and environmental factors to individual differences in antisocial behavior. Several candidate genes have been identified to be associated with antisocial behavior or their known risk factors. Many of these candidate genes' findings have also been replicated in both human and animal studies. A majority of these candidate genes were identified through examination of (i) the dopamine system, which is involved in mood, motivation and reward, arousal, and other behaviors; (ii) the serotonin system, which is involved in impulse control, affect regulation, sleep, and appetite; or (iii) the epinephrine/norepinephrine system, which facilitates fight-or-flight reactions and autonomic nervous system activity (28). All three of these systems are affected by monoamine oxidase A (*MAO-A*) function (29, 30). The low-activity alleles of *MAO-A* interacts with maladaptive childhood environment (31) and has been associated with aggression, violent delinquency, externalizing behavior, and lower inhibitory control (32, 33).

Gene aberrations cause constitutional brain deformities that lead to sentimental, behavioral, or cognitive anomalies; they, in turn, result in antisocial behavior (34).

Genetic studies found that polymorphisms at the promoter region of the gene was included in monoamine oxidase A (MAOA) genotype (5). Four recurrences of alleles are correlated with the high action gene's level i.e. to higher genetic expression, while three repetitions are related to a decreased action level (35). Widom et al. (36) performed a prospective study with 802 cases of persons who suffered from violence and negligence, which continued till adulthood. Those who suffered from violence and negligence were compared to a controlled group, which had not been subjected to assault during infancy. The MAOA genotypes of these persons were compared. The population was divided according to ethnicity: Caucasians and non-Caucasians. They concluded that in the presence of an adverse environment at infancy, Caucasians with low activity genotypes had higher risk of developing behavioral problems throughout their life. For children subjected to abuse at infancy, a higher expression level of MAOA was associated to lower violence and antisocial behavior frequency at adulthood. This association was not found among non-Caucasian persons, in whom the genetic polymorphism looks less related to the gene's expression levels. The low MAOA genotype activity was not a proof of antisocial behavior and aggression in the absence of abuse at infancy. Thus, the authors documented that the MAOA genotype had mild influence on the impact of negligence and assault which occurred at childhood, on the growth of aggression and antisocial behavior in adults (36,37).

MAOA is the enzyme that responsible for break down the neurotransmitter serotonin which is low in individuals with antisocial behavior disorder. Men with a common polymorphism in the MAOA gene have about an 8% decrease in the volume of the amygdala, anterior cingulate, and orbitofrontal (ventral prefrontal) cortex. These brain structures are responsible for emotions and are compromised in antisocial behavior persons (38). Serotonin is the hormone responsible to regulate sympathy and behavior; it also plays a part to block aggressive behavior (39,40). Aberration in serotonin function is correlated with aggression (41,42). Low levels of serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) are bound with a lifetime of aggressive behavior, aggression in patients with mental disorders, committing suicide, impulsive killing, and recidivism of killers (41,43–47). A meta-analysis study of twenty different researches conducted by Moore et al. (48) found that low concentrations of serotonin are linked to aggression. It was proposed that serotonin hypofunction is correlated with impulsive aggression types (49). A research demonstrated that impulsive violent criminals had less cerebrospinal fluid 5-HIAA concentrations than non-impulsive violent criminals (50). A literature documented that impulsive alcoholic offenders had lower cerebrospinal fluid 5-HIAA concentrations than non-impulsive alcoholic offenders and persons from the controlled group (44).

Genetic effects on impulsive aggressive behavior were recognized in research studies from the study conducted by Berggard et al. (51); they acknowledged combination between serotonergic dysfunction, specifically low concentrations of serotonin genotype 5-HT2A-1438GG and offending behavior. Beitchman et al. (52) assessed the impact of polymorphisms in the transporter gene of serotonin and aggressive antisocial behavior during both childhood and puberty. The study involved eighty-two individuals, their ages ranged from five to fifteen years, and they were genotyped for 5HTTLPR and 5HTT variable-number-tandem-repeat polymorphisms. The alleles with a low genic expression in the transcription control site, in the serotonin transporter gene 5-HTTLPR (S/S, LG/S, Lg/Lg) was potentially correlated with a doubled hazard of assault at childhood in comparison with individuals with elevated expression alleles. This led to the finding that low expression alleles in adults is really correlated with the most violent behavior.

Spoont (53) showed that serotonin stabilizes information processing in neural systems, causing controlled behavioral, affective, and cognitive product, whereas aberrations in serotonin activity cause altered information processing tendencies (53). High levels of serotonin were thought to result in excessive restraint, cognitive inflexibility, and anxiety, while low levels were proposed to cause behavioral disinhibition and distractibility (3). It was found that dysregulation of CNS serotonergic activity participated in behavioral states and psychological traits, correlated with violence and aggression (54). Diminished serotonergic function disinhibits aggression directed against the self and others, maybe by whetting sensitivity to stimuli that elicit aggression and irritation, and blunting sensitivity to cues that signal penalization (53). There is a positive correlation between the degree of serotonin system impairment and the severity of the aggression displayed by the person.

In human beings, the dopaminergic system is associated with recognition and experience of aggressive behavior (55). Impulsive behavior has been found to be enhanced by elevation in dopaminergic activity (56–58). In cases with borderline personality disorders, dopaminergic hyperfunction was found to be associated with impulsivity and sympathy dysregulation in these patients (59,60). A study was conducted by Guo et al. (91) on the effect of dopamine transporter gene, which regulates synthesis and release of dopamine transporter protein responsible for utilizing dopamine in the synaptic cleft and for the expression of the receptor gene (61). Two thousand and five hundred adolescents and young adults were included in this study; it showed that ten repetitions of forty pairs of nitrogenous bases at region 3 multiplies the hazard of juveniles being comprised in drastic and misdemeanant behaviors, while heterozygosis of dopamine receptor D2 (DRD2) elevates the danger by 20%, and homozygosis multiplies the danger of the diversity (61).

A study conducted by Kotler et al. (62) revealed the presence of polymorphism in the catechol-O-methyl transferase (COMT) enzyme transcriptor gene; the study included 353 persons, of which 180 were with schizophrenia and 173 were controlled. The results revealed that patients homozygous for the

polymorphism, which specifies the efficiency level of COMT gene, displayed significantly higher aggressiveness scores in comparison to the heterozygous ones. It is believed that the gene's decreased efficiency is correlated with deficiencies in the prefrontal cortex, thus resulting in the reduction of aggressive motives.

Entanglements in the Prenatal Period and at Birth

A study was carried out on 177 male children, whose mothers continuously smoked during the third trimester and were known for committing violent and non-violent offenses (63). The authors discovered that intrauterine exposure to tobacco represents a well-known dangerous factor for the enhancement of aggression and antisocial behavior disorders. Orlebeke et al. (64) recognized that smoking during pregnancy was strongly combined to opposition, hyperactivity, and aggression. Animal literature had bound the intrauterine exposure to tobacco, to damages in the noradrenergic system, decreased levels of serotonin and dopamine, decreased brain glucose concentrations, and damage to the basal ganglions, cerebral, and cerebellar cortexes (65).

Fetal exposure to alcohol can destruct corpus callosum, where aggressive behavior was related to damage in the corpus callosum (5). A study conducted by Roebuck et al. (66) was carried out on two children groups: the first heavily exposed to alcohol during the intrauterine period, and the second not exposed. Those group exposed to alcohol showed cognition and psychological impairments and were involved in delinquency.

A study was carried out, comprising 201 children, who had been exposed to cocaine during intrauterine period, and 270 children, who had not exposed. The authors concluded that the first group was with delinquent behaviors, with boys being more vulnerable to aggressive behavior. Cocaine influences monoaminergic systems, and intrauterine exposure to cocaine intervenes with growth of these systems (67).

Neurological Changes

The burgeoning field of social neuroscience is beginning to provide important insights into the neural mechanisms, which underlie the cognitive and affective processes that guide social behavior in everyday life. One particularly important sub-field within this area that has significant societal implications concerns the neural basis to antisocial behavior. The perspective that will be developed here is that there are some similarities between the neural system, underlying moral decision-making in normal individuals, and brain mechanisms thought to be impaired in delinquent, criminal, violent, and psychopathic populations. Despite the increasing evidence for neurological impairment in antisocial individuals, very few structural and functional brain-imaging studies have been conducted specifically on the recognized medical disorder for antisocial behavior. Key areas found to be functionally or structurally impaired in antisocial populations include dorsal and ventral regions of the prefrontal cortex (PFC), amygdala, hippocampus, angular gyrus, anterior cingulate, and temporal cortex. Regions most commonly activated in moral judgment tasks consist of the

polar/medial and ventral PFC, amygdala, angular gyrus, and posterior cingulate. It is hypothesized that the rule-breaking behavior common to antisocial, violent, and psychopathic individuals is in part due to impairments in some of the structures (dorsal and ventral PFC, amygdala and angular gyrus) subserving moral cognition and emotion (68).

The frontal lobe is combined with functions which include planning, making decisions, monitoring, making estimations, regulating behavior according to internal and external drivers, sensations, and controlling one's attitude (5). The prefrontal region is responsible on organizing of sentiments, responses, and motives created by the limbic system. Damage or harm to the prefrontal region compromises the control of subcortical areas, thus producing negative sentimental responses and aggressive antisocial behavior (69). Ventral and orbital prefrontal lesions are linked to greater predisposition, to be engaged in aggressive behavior. In both children and adolescents, traumatic head injuries, especially to frontal regions are correlated with excess violence (65). Damages to the medial temporal lobe, where the limbic system bodies are located, are correlated with impulse control disorders, aggression, and antisocial behavior (5).

Hormonal Factors

The reasons for regarding androgens to be included in antisocial and aggressive behavior is that males have higher levels of androgens, which results in higher levels of violence and antisocial behavior than females (3). Women involved in offenses tend to be extra violent during their menstrual cycle (70). This is explained by the low concentrations of estrogen that represent the menstrual cycle (70). Moreover, high testosterone concentrations were found to be associated with aggressive behavior disorders (71–74). In adult individuals, high testosterone levels in cerebrospinal fluid, plasma, and saliva are combined with both antisocial behavior disorders, aggression, and violent crimes. A literature found that alcoholic men diagnosed with antisocial personality disorder (ASP) were found to be irritable, impulsive and aggressive, avoiding monotonous action, and with higher free cerebrospinal fluid testosterone concentration as compared to the controlled group (49). Studies of adults with antisocial behavior observed a negative relationship between cortisol concentrations and the level of behavioral derivation (76). Lower concentrations of cortisol indicate that these persons are physiologically under-aroused and that the negative feedback mechanisms acting on their hypothalamic-pituitary-adrenal axes are hypersensitive, or that they have elevated threshold for stress (77).

The serious forms of aggressive behavior are more occurring in males than in females. Some suppose the reasons is that females have larger corpus callosum, good interhemispheric communication, elevated verbal capability, and faster maturement of the frontal areas, thus stimulating the growth of cognitive and social potencies, which make females cooperative with interpersonal issues (77).

Farrington et al. (77) revealed the following social factors as the predictors of aggression and violence: indigence, felony family history, problematic breeding, school flopping, attentiveness deficiency, hyperactivity, and antisocial behavior

during infancy. Children who are inconsistently disciplined or gratified, who have family struggles, or whose father and/or mother is/are involved in offenses are not able to develop the needed skills to address social conflicts. Abuse at infancy including maternal rejection, inter-parental violence, negligent parenting, repeated loss of the primary caregiver, severe discipline, and sexual and physical abuse constitute dangerous factors for producing violence and aggressive behavior; this leads to offensive, aggressive, and antisocial behavior when children become adults (78). Teenagers less attached to their mothers, whose parents are absent, who are poorly engaged, or who are sentimentally and sympathetically cut off get easily involved with delinquency, become alcoholics and drug abusers, and socialize with delinquent peers (5).

Conclusions

Inadequate nutrition, genetic factors, environmental complications before and at birth, neurological changes, hormonal alterations, intoxicants, gender differences, and social factors significantly contribute in the development of aggressive antisocial behavior in children, adolescents, and adults.

References

1- World Health Organization. (1996). WHA 49.25 Prevention of violence: a public health priority. Forty-ninth Assembly May 20-25. Geneva: World Health Association.

2- World Health Organization. (2002).World report on violence and health. Geneva, Switzerland: World Health Organization.

3- Goozen S.; Fairchild G.; Snoek H.; Harold G. (2007). The evidence for a neurobiological model of childhood antisocial behavior. Psychological Bulletin, 133(1):149-182.

4- Bennett S.; Farrington DP.; Huesmann R. (2005). Explaining gender differences in crime and violence: the importance of social cognitive skills. Aggression Violent Behavior, 10:263-88.

5- Mendes D.; Mari J.; Singer M.; Barros G.; Mello A. (2009). Study review of the biological, social and environmental factors associated with aggressive behavior. Rev Bras Psiquiatr, 31(Suppl11):(S77-85).

6- Warner V.; Weissman MM.; Mufson L.; Wickramaratne P. (1999). Grandparents, parents and grandchildren at high risk for depression: a three-generation study. J AmAcad Child Adolesc Psychiatry, 38:289-296.

7- Kim-Cohen J.; Miffitt T.; Taylor A.; Pawlby S.; Caspi A. (2005). Maternal depression and children's antisocial behavior. Arch Gen Psychiatry, 62:173-181.

8- Vitiello B.; Stoff DM. (1997). Subtypes of aggression and their relevance to child psychiatry. Journal of the American Academy of Child and Adolescent Psychiatry, 36: 307–315.

9- Stoff DM.; Vitiello B. (1996). Role of serotonin in aggression of children and adolescents: Biochemical and pharmacological studies. In DM. Stoff and RB. Cairns (Eds.), Aggression and violence: Genetic, neurobiological and biological perspectives (pp. 67–85). Mahwah, NJ: Erlbaum.

10- Prado E.; Dewey K. (2014). Nutrition and brain development in early life. Nutrition Reviews, 72(4):267-284.

11- Jackson D. (2016). The link between poor quality nutrition and childhood antisocial behavior: a genetically informative analysis. Journal of Criminal Justice, 44:13-20.

12- Thompson R.; Tabone J. K.; Litrownik A. J.; Briggs E. C.; Hussey J. M.; English D. J.; Dubowitz H. (2010). Early adolescent risk behavior outcomes of childhood externalizing behavioral trajectories. Journal of Early Adolescence, 31(2): 234–257.

13- Fergusson D. M.; Boden J.M.; Horwood L. J. (2014). Bullying in childhood, externalizing behaviors, and adult offending: Evidence from a 30-year study. Journal of School Violence, 13(1): 146–164.

14- Gray G. (1986). Diet, crime and delinquency: a critique. Nutrition Reviews, Supplement/May 1986:89-94.

15- Virkkunen M. (1983). Insulin secretion during the glucose tolerance test in antisocial personality. Br J Psychiatry, 142:598-604.

16- Oh S. Y.; Ahn H.; Chang N.; Kang M. H.; V Oh J. (2013). Dietary patterns and weight status associated with behavioural problems in young children. Public Health Nutrition: 1-7.

17- Gómez-Pinilla F. (2008). Brain foods: The effects of nutrients on brain function. Nature Reviews Neuroscience, 9(7): 568–578.

18- Gesch CB.; Hammond SM.; Hampson SE.; Eves A.; Crowder MJ. (2002). Influence of supplementary vitamins, minerals and essential fatty acids on the antisocial behaviour of young adult prisoners: randomised, placebo-controlled trial. Br J Psychiatry, 181:22–28.

19- Hibbeln J. R.; Ferguson T. A.; Blasbalg T. L. (2006). Omega-3 fatty acid deficiencies in neurodevelopment, aggression and autonomic dysregulation: Opportunities for intervention. International Review of Psychiatry, 18(2): 107–118.

20- Woo H. D.; Kim D.W.; Hong Y. S.; Kim Y. M.; Seo J. H.; Choe B. M.; Kim J. (2014). Dietary patterns in children with attention deficit/hyperactivity disorder (ADHD). Nutrients, 6(4): 1539–1553.

21- Halas ES.; Reynolds GM.; Sandstead HH. (1977). Intra-uterine nutrition and its effects on aggression. Physiol Behav. ,19:653–661.

22- Neugebauer R.; Hoek HW.; Susser E. (1999). Prenatal exposure to wartime famine and development of antisocial personality disorder in early adulthood. JAMA., 4:479–481.

23- Raine A. (1993). The Psychopathology of Crime: Criminal Behavior as a Clinical Disorder. Academic Press; San Diego: 1993.

24- Werbach M. (1995). Nutritional influences on aggressive behavior. J Orthomolecular Med, 7:45-51.

25- Bath SC.; Steer CD.; Golding J.; Emmett P.; Rayman MP. (2013). Effect of inadequate iodine status in UK pregnant women on cognitive outcomes in their children: results from the Avon Longitudinal Study of Parents and Children (ALSPAC). Lancet, 382:331–337.

26- Lozoff B.; Beard J.; Connor J.; Barbara F.; Georgieff M.; Schallert T. (2006). Long-lasting neural and behavioral effects of iron deficiency in infancy. Nutr Rev., 64(Suppl):S34–S43.

27- Sandstead HH.; Frederickson CJ.; Penland JG. (2000). History of zinc as related to brain function. J Nutr., 130(Suppl 2):496S–502S.

28- Bartels M, van de Aa N, van Beijsterveldt CEM, Middeldorp CM, Boomsma DI. Adolescent self-report of emotional and behavioral problems: interactions of genetic factors with sex and age. Journal of the Canadian Academy of Child and Adolescent Psychiatry. 2011; 20(1):35–52. [PMC free article] [PubMed] [Google Scholar.

29- Niv S, Baker LA. Genetic marker for antisocial behavior. In: Thomas C, Pope K, editors. The origins of antisocial behavior: A developmental perspective. Oxford University Press; New York: 2010. [Google Scholar].

30- Tuvblad C, Baker LA. Huber R, Brennan P, Bannasch D, editors. Human Aggression across the Lifespan: Genetic Propensities and Environmental Moderators. Advances in Genetics (Aggression) 2011;75:171–214. [PMC free article] [PubMed] [Google Scholar].

31- Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, et al. Role of geno-type in the cycle of violence in maltreated children. Science. 2002; 297(5582):851–854. [PubMed] [Google Scholar].

32- Brunner HG, Nelen M, Breakefield XO, Ropers HH, van Oost BA. Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. Science. 1993;262(5133):578–580. [PubMed] [Google Scholar].

33- Guo G, Ou X, Roettger M, Shih JC. The VNTR repeat in MAO-A and delinquent behavior in adolescence and young adulthood: associations and MAO-A promoter activity. European Journal of Human Genetics. 2008;16:626–634. [PMC free article] [PubMed] [Google Scholar].

34- Raine A. (2011). From genes to brain to antisocial behavior. Current Directions in Psychological Science, 17(5):323-328.

35- Denney RM.; Koch H.; Craig IW. (1999). Association between monoamine oxidase A activity in human male skin fibroblasts and genotype of the MAOA promoter-associated variable number tandem repeat. Hum Genet,105(6):542-51.

36- Widom CS.; Brzustowicz LM. (2006). MAOA and the "cycle of violence:" chidhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. Biol Psychiatry, 60(7):684-9.

37- Cases O.; Seif I.; Grimsby J.; Gaspar P.; Chen K.; Pournin S.; Muller U.; Aguet M.; Babinet C.; Shin JC. (1995). Aggressive behavior and altered amounts of brain serotonin and norepinephrine in mice lacking MAOA. Science, 268: 1763–1766.

38- Meyer-Lindenberg A.; Buckholtz J.W.; Kolachana B.; Hariri A.R.; Pezawas L.; Blasi G.; Wabnitz A.; Honea R.; Verchinski B.; Callicott JH.; Egan M.; Mattay V.; Weinberger DR. (2006). Neural mechanisms of genetic risk for impulsivity and violence in humans. Proceedings of theNational Academy of Sciences, U.S.A., 103, 6269–6274.

39- Davidson RJ.; Putnam KM.; Larson CL. (2000). Dysfunction in the neural circuitry of emotion regulation-a possible prelude to violence. Science, 289:591–594.

40- Volavka J. (1999). The neurobiology of violence: an update. Journal of Neuropsychiatry and Clinical Neurosciences, 11:307–314.

41- Coccaro EF. (1989). Central serotonin and impulsive aggression. British Journal of Psychiatry Supplements, 8:52–62.

42- Raleigh MJ.; McGuire MT.; Brammer GL.; Pollack DB.; Yuwiler A. (1991). Serotonergic mechanisms promote dominance acquisition in adult male vervet monkeys. Brain Research, 559:181–190.

43- Brown GL.; Goodwin FK.; Ballenger JC.; Gover PF. (1979). Aggression in humans correlates with cerebrospinal fluid amine metabolites. Psychiatry Research, 1:131–139.

44- Virkkunen M.; Rawlings R.; Tokola R.; Poland RE.; Guidotti A.; Nemeroff CB.; Bissette G.; Kalogeras K.; Karonen SL.; Linnoila M. (1994). CSF biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholic, violent offenders, fire setters, and healthy volunteers. Archives of General Psychiatry, 51:20–27.

45- Traskman-Bendz L.; Asberg M.; Schalling D. (1986). Serotonergic function and suicidal behavior in personality disorders. Abdominal Aortic Aneurysm: Genetics, Pathophysiology, and Molecular, 487:168–74.

46- Lidberg L.; Tuck JR.; Asberg M.; Scalia-Tomba GP.; Bertilsson L. (1985). Homicide, suicide and CSF 5-HIAA. Acta Psychiatrica Scandinavica, 71:230–236.

47- Virkkunen M.; De Jong J.; Bartko JJ.; Goodwin FK.; Linnoila M. (1989). Relationship of psychobiological variables to recidivism in violent offenders and impulsive fire setters: a follow-up study. Archives of General Psychiatry, 46:600–603.

48- Moore T.;Scarpa A.; Raine A. (2002). A Meta-Analysis of Serotonin Metabolite 5-HIAA and Antisocial Behavior. Aggressive Behavior, 28:299–316.

49- Virkkunen M.; Linnoila M. (1993). Brain serotonin, type II alcoholism and impulsive violence. Journal of Studies on Alcohol, 11:163–169.

50- Linnoila M.; Virkkunen M.; Scheinin M.; Nuutila A.; Rimon R.; Goodwin FK. (1983). Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. Life Sciences, 33:2609–2614.

51- Berggard C.; Damberg M.; Longato-Stadler E.; Hallman J.; Oreland L.; Garpenstrand H. (2003). The serotonin 2A-1438 G/A receptor polymorphism in a group of Swedish male criminals. Neuroscience Letters, 347:196–198.

52- Beitchman JH.; Baldassarra L.; Mik H.; De Luca V.; King N.; Bender D.; Ehtesham S.; Kennedy JL. (2006). Serotonin transporter polymorphisms and persistent, pervasive childhood aggression. Am J Psychiatry, 163(6):1103-5.

53- Spoont M. R. (1992). Modulatory role of serotonin in neural information processing: Implications for human psychopathology. Psychological Bulletin, 112: 330–350.

54- Zageer D.; Hantoosh S.; AL-Rubai H. (2019). Amino Acids Deficits in Brain. Book Publisher International. India-United Kingdom. ISBN: 978-93-89246-36-0(print). DOI: 10.9734/bpi/mono/978-93-89246-36-0.

55- Lawrence AD.; Calder AJ.; McGowan SW.; Grasby PM. (2002). Selective disruption of the recognition of facial expressions of anger. Neuroreport, 13:881–884.

56- Bergh C.; Eklund T.; Sodersten P.; Nordin C. (1993). Altered dopamine function in pathological gambling. Neuropsychobiology, 28:30–6.

57- Brunner D.; Hen R. (1997). Insights into the neurobiology of impulsive behavior from serotonin receptor knockout mice. Abdominal Aortic Aneurysm: Genetics, Pathophysiology, and Molecular, 836:81–105.

58- Seo D.; Patrick C. (2008). Role of serotonin and dopamine system interactions in the neurobiology of impulsive aggression and its comorbidity with other clinical disorders. Aggress Violent Behav, 13(5):383-395.

59- Chotai J.; Kullgren G.; Asberg M. (1998). CSF monoamine metabolites in relation to the diagnostic interview for borderline patients (DIB). Neuropsychobiology, 38:207–212.

60- Friedel RO. (2004). Dopamine dysfunction in borderline personality disorder: a hypothesis. Neuropsychopharmacology, 29:1029–1039.

61- Guo G.; Roettger ME.; Shih JC. (2007). Contributions of the DAT1 and DRD2 genes to serious and violence delinquency among adolescents and young adults. Hum Genet, 121(1):125-36.

62- Kotler M.; Barak P.; Cohen H.; Averbuch IE.; Grinshpoon A.; Gritsenko I. (1999). Homicidal behavior in schizophrenia associated with a genetic polymorphism determining low COMT activity. Am J Med Genet, 88(6):628-33.

63- Brennan PA.; Grekin ER.; Mednick SA. (1999). Maternal smoking during pregnancy and adult male criminal outcomes. Arch Gen Psychiatry, 56(3):215-9.

64- Orlebeke JF.; Knol DL.; Verhulst FC. (1997). Increase in child behavior problems resulting from maternal smoking during pregnancy. Arch EnvironHealth, 52(4):317-21.

65- Liu JH. (2004). Prenatal and perinatal complications as predispositions to externalizing behavior. J Prenatal Perinatal Psychol Health, 18(4):301-11.

66- Roebuck TM.; Mattson SN.;Riley EP. (1999). Behavioral and psychosocial profiles of alcohol-exposed children. Alcohol Clin Exp Res,23(6):1070-6.

67- Delaney-Black V.; Covington C.; Templin T.; Ager J.; Nordstrom-Klee lB.; Martier S.; Leddick L.; Czerwinski RH.; Sokol RJ. (2000). Teacher-assessed behavior of children prenatally exposed to cocaine. Pediatrics, 106(4):782-91.

68- Raine, A., & Yang, Y. (2006). Neural foundations to moral reasoning and antisocial behavior. Social cognitive and affective neuroscience, 1(3), 203-213.

69- Jones H. (1984). Neuropsychology of violence. Forensic Reports, 5:221-33.

70- Liu J.; Wuerker A. (2005). Biosocial bases of aggressive and violent behavior implicationsfor nursing studies. Int J Nurs Stud, 42(2): 229-41.

71- Archer J. (1991). The influence of testosterone on human aggression. Br J Psychol, 82:1–28.

72- Archer J. (1994). Testosterone and aggression. J OffendRehabil, 21:3–25.

73- Olweus D.; Mattson Å.; Schalling D.; Löw H. (1988). Circulating testosterone levels and aggression in adolescent males: A causal analysis. Psychosom Med, 50:261–272.

74- Aromaki A.; Lindman R.; Erikkson C. (1999). Testosterone, aggressiveness, and antisocial personality. Aggressive Behavior, 25:113-123.

75- Bergman B.; Brismar B. (1994). Hormone levels and personality traits in abusive and suicidal male alcoholics. Alcoholism, Clinical and Experimental Research, 18: 311–316.

76- Kruesi M. J.; Schmidt M. E.; Donnelly M.; Hibbs E. D.; Hamburger S. D. (1989). Urinary free cortisol output and disruptive behavior in children. Journal of the American Academy of Child and Adolescent Psychiatry, 28: 441–443.

77- Farrington DP. (1998). Predictors, causes and correlates of male youth violence. In: Tonry M, Moore M, editors. Youth violence. Chicago: University of Chicago Press, p.421-75.

78- Reif A.; Rösler M.; Freitag CM.; Schneider M.; Eujen A.; Kissling C.; Wenzier D.; Jacob CP.; Retz-Junginger P.; Thome J.; Lesch KP.; Retz W. (2007). Nature and nurture predispose to violent behavior: serotonergic genes and adverse childhood environment. Neuropsychopharmacology, 32(11):2375-83.