Available online at www.ujpronline.com
Universal Journal of Pharmaceutical Research
An International Peer Reviewed Journal
ISSN: 2831-5235 (Print); 2456-8058 (Electronic)

REVIEW ARTICLE

INTEGRATING GENOMIC DISCOVERIES INTO FORENSICS: A MINI REVIEW

Nwawuba Stanley Udogadi*[1], Ogbemudia Divine[2], Ehikhamenor Edeaghe[1]
[1]Centre for Forensic Programmes and DNA Studies, University of Benin, Benin City, Nigeria.
[2]Department of Biochemistry, Faculty of Life Science, University of Benin, Benin City, Nigeria.

Article Info:

Article History:
Received: 6 February 2022
Reviewed: 12 March 2022
Accepted: 14 April 2022
Published: 15 May 2022

Cite this article:
Nwawuba SU, Divine O, Edeaghe E. Integrating genomic discoveries into forensics: A mini review. Universal Journal of Pharmaceutical Research 2022; 7(2):81-85.
https://doi.org/10.22270/ujpr.v7i2.750

***Address for Correspondence:**
Nwawuba Stanley Udogadi, Centre for Forensic Programmes and DNA Studies, University of Benin, P.M.B. 1154, Benin City, Nigeria. Tel- +234-8065699068;
E-mail: *nwawubastanley@gmail.com*

Abstract

The justification that behavioral genetic evidence provides in criminality remains a wonder. Scientific evidence suggests that a decreased activity of the enzyme monoamine oxidase (MAOA-L) dubbed the warrior gene surges the possibility for aggressive, antisocial behavior and increased criminality. Ever since the human genome project was completed, breakthrough in genetics and biotechnology are now advancing at a speedy rate. Advancement in genetics and biotechnology has opened a frontier to carry out investigation on the influence of genetics on human behavior at the molecular-genetic level. The interplay between genes and susceptibility to diseases has extensively been accepted, but, the similar link between genes and predisposition to criminal behavior is yet to gain a wide acceptance. Notwithstanding this cynicism, biological studies have revealed that relatives of convicted criminals have higher chances of criminality, suggesting a genetic association to certain criminal behavioral tendencies. Emerging data and evidence indicates that without recourse to genetics, there would be a limited opportunity in elucidating the mechanism and reason for criminal, aggressive and antisocial behavior of some individuals. In this light, reports from some scientific finding revealed that monoamine oxidase A (MAOA) provides the strongest association between genetic variation and aggressive behavior particularly low gene variant monoamine oxidase A (MAOA-L) which has been introduced by experts as evidence for criminal behavior. Beside a satisfaction of scientific curiosity, genetic research portends the potential to contribute to preventive measures and investigation into the genetic etiological correlation of criminal behavior may result to the opening of a new frontier for treatment and intervention.
Keywords: Genetics and Crime, Genes and criminal Behavior, Monoamine Oxidase, and Warrior Genes.

INTRODUCTION

Genetic factors, an imperative basis of influence implicated in several pathological as well as psychological conditions has recently been demonstrated to also influence the predisposition of certain individuals to criminal behavior[1,2]. Since the completion of the human genome project (HGP), breakthrough in genetics and biotechnology are now advancing at a speedy rate[3,4]. Advancement in genetics and biotechnology has opened a frontier to carry out investigation on the influence of genetics on human behavior at the molecular-genetic level[5,6]. The interplay between genes and susceptibility to diseases has extensively been accepted, but, the similar link between genes and predisposition to criminal behavior is yet to gain a wide acceptance[7]. Notwithstanding this cynicism, a body of extensive studies have revealed that behavioral models suggest that neurotransmitter pathways in the brain region linked to cognitive function and the encoding of emotions maybe influenced by genetic variations[8,9,10]. A biological study conducted by Taylor[11] revealed that relatives of convicted criminals have higher chances of criminality, suggesting a genetic association to certain criminal behavioral tendencies. MAOA has widely been reported to be a key gene associated to criminal behavior and it is found on the X chromosome functioning as a regulator for the metabolism of some neurotransmitters including serotonin, dopamine and norepinephrine associated with aggression and rash behavior[11,12,13,14]. As a result of the regulatory function of the MAOA gene in the metabolism of neurotransmitters, it has extensively been suggested that a dysfunctional MAOA gene continue to be an excellent genetic predictor of an aggressive behavior,

and wide range of scientific findings have associated an increased risk of aggressive behavior to MAOA gene deficiency[10,15,16]. Conversely, it is rare for MAOA gene to be absent but a low activity variant of the gene (MAOA-L) exist and have established to be linked to aggressive, criminal and violent behavior[1,17,18].

Experimentally, it was reported that males of a large Dutch kindred whom exhibited anomalous aggressive behavior were observed to have low level of MAOA (MAOA-L) activity associated with a deleterious point mutation in the 8th exon of the gene[18,19]. Similarly, it was observed that the unaffected males of the Dutch kindred were not affected this mutation[18]. Overall, MAOA-L individuals are found to be very aggressive relative to MAOA-H (high MAOA activity) individuals[20,21,22]. To end with, reducing a jail term of a convicted murderer owing to the MAOA gene seems implausible, but it has been reported. The first attempt to use evidence of MAOA levels in defense of a convicted murderer was in the United State of America in the year 1994[17]. Since then, it has become a contestable discus among lawyers and scientist alike. In Italy and the USA alike, it was observed that a common genetic variation, present in around a third of Caucasian men, has successfully been used in the defense for violent criminals[11]. This review provides updates on the current knowledge and position of genes attributed to criminal behavior. The paper would also highlight the discovery of Aggression-Causing genes, the linkage between MAOA gene and aggressive and antisocial behavior, and the forensic use of behavior with recourse to genetics in criminal proceedings.

Discovery of Aggression-Causing Genes

In the last couple of years, geneticist and molecular biologist have been searching for a link between genetic root and violent criminal behavior and this led to the discovery of some aggression-causing genes[18]. This aggression-causing gene have received growing interest from the legal and scientific milieu as they consider the influence of such genes on the criminal justice system and personal responsibility[11,18,23]. A growing body of methodical evidence have shown that aggression related genes holds two interest and opposite preposition: first, the potential of prediction and mitigating imminent crime of affected individuals via gene knock-out and second reducing affected individual jail terms based on predisposition to aggressor gene[1,18,24]. While recourse to genetic make-up provides a scientific basis to clear up legal complexity, there remains a huge concern that there might be a focus to genetic makeup as a way out in dealing with criminality by neglecting other potential environmental based causal factors[24].

In an attempt to discover the aggression causing genes, experimental studies implicated monoamine oxidase dubbed warrior gene as the aggressor gene in both mice and human studies and this generic abnormality have maintained a great deal of attention for both scientist and legal practitioners[1,18]. Although experimental studies of aggressive behavior in mice commenced around fifty years now, a breakthrough by geneticist and molecular biologist in identifying specific genes implicated in aggressive behavior have only recently been recorded and accepted[5,18]. Experimental study using mouse offers key insight on elucidation of aggressive and antisocial behavior in human owing in part to the fact that there is neurological similarity between mice and humans[18,24,25,26].

Using genetic "knockout" technology, a family of mice was genetically altered to be monoamine oxidase A (MAOA) deficient and experimentation revealed that the catalytic activity of MAOA was silenced resulting to the production of increased levels of neurotransmitters (serotonin and norepinephrine) causing an impulsive aggressive behavior in the mice[5]. The study also observed that they male mice frequently attacked one another by biting each other's genitals and rump and the study concluded that the absence of MAOA in the genetically altered mice provides a proof of genetic link to the aggressive behavior[5,6,18,24]. In validation to the mice study, an experimental study conducted for a reported four generation involving a males of a Dutch kindred showed that a total of fourteen males exhibited aggressive, criminal and antisocial behavior including arson, aggravated attacks, sexual assault, and exhibitionism and the study concluded with the observation that the affected males were found to be deficient of the MAOA gene[5,6,18,24]. Correspondingly, the neurogeneticist and collaborator of the Dutch kindred experimental study, Xandra Breakefield reported that an abnormal accumulation of neurotransmitters due to the deficiency of the MAOA genes results to a challenge in handling stressful predicaments eliciting an impulsive, violent and aggressive manner of behavior among affected individuals[5,18,24,27].

Epidemiological design in validation of genetic influence on criminal behavior

Epidemiological designs which involves the use of family, twin and adoption as parameters to determine the influence of genetics on aggressive behavior suggest that criminal behavior may strongly be associated to genetic makeup[1]. In respect to twin as an indices of epidemiological design, a twin studies involving well over ten twins conducted in different countries comparing the rate of criminal behavior between monozygotic twins (MZ) and dizygotic twins (DZ) revealed a genetic link to criminal behavior. Generally, these studies corroborate the existing findings that criminal behavior is linked to genetic predisposition and specifically, a greater concordance rate for aggressive behavior and criminality was noticed for MZ twins than for DZ twins[1,5,6].

As per adoption, adoption studies provide a natural experiment to test the reality and degree of inherited predispositions[1]. In this light, existing adoption studies reported in The United States, Sweden and Denmark revealed that criminal behavior may have important genetic influences[5,28,29]. In Sweden a study conducted by Bohman *et al.*,[28] which assessed the extent of criminality and alcoholism in 2324 study population of Swedish adoptees and their biological and adoptive parents, revealed that a biological background positive for criminality contributed to an increased risk of criminality in the adopted-away children. In Denmark, a study conducted by Mednick *et al.*,[29] on the

relationship of genetics to criminal behavior using an extensive data set consisting of 14,427 Danish adoptees (ranging in age from 29 to 52 years) and both sets of biological and adoptive parents. The study found out that adopted-away sons had a higher risk of having a court conviction if their biological parent, rather than their adoptive parent, had one or more court convictions. Finally, in USA the first adoption study conducted by Crowe in 1974 revealed a genetic relationship to criminal behavior and this finding was corroborated by another independent conducted by Cadoret and colleagues therefore validating the concept that criminal behavior may have important genetic influences[1,6].

Overview of Monoamine oxidase (MAOA)

Monoamine oxidase A gene (MAOA) regarded as ''warrior gene'', is an enzyme that in humans is encoded by the MAOA gene[19,22]. This gene is one of two neighboring gene family members that encodes the mitochondrial enzymes catalyzing oxidative deamination of neurotransmitters including dopamine, norepinephrine, and serotonin[5,6,18,30,31]. It has been reported that mutation in the MAOA genes leads to Brunner syndrome, and has particularly been linked to aggressive and antisocial behavior[32,33,34]. The promoter region of the MAOA carries a conserved binding sites for specificity protein 1 (Sp1); a human protein encoded by the SP1 gene (Sp1), GATA-binding factor 2 (GATA2) which is a nuclear protein regulating gene expression, and the TATA-binding protein (TBP) considered as a general transcription factor that binds specifically to a DNA sequence called the TATA box[35,36,37]. Going forward, the human promoter region of the MAOA gene contains a 30-base repeat sequence repeated over a varying number of times[20,38,39]. There are 2R (two repeats), 3R, 3.5R, 4R, and 5R variants of the repeat sequence, with the 3R and 4R variants been the predominant in all populations, however, the 3.5R and 4R variant has been observed to be very active as compared to the other variants[38,39]. Additionally, variations in the MAOA promoter repeat has been reported; 52-59% of African American men, 48-62% of Chinese men, 62% of Maori men, 57% of Japanese men and 33-37% of European men carried the 3R allele, while 5.2% of Black men, 0.1% of European men, and 1.3% of Asian men carried the 2R allele[5,27].

Observational, survey-based and experimental studies as well as the recent advances in an emerging field of study, the neurocriminology have well demonstrated an impeccable scientific relationship between genetic makeup and criminal and antisocial behavior[17,30,31,40]. A body of extensive reports have implicated the MAOA gene as the strongest link between criminality, aggression, antisocial behavior and genetics[17,21,33,34].

Genetic Evidence in Criminal Proceedings: Case of the MAOA-L Gene

It has widely been reported that the past two decades have seen a global acceptance of neuroscientific evidence in criminal proceedings, and review of criminal cases revealed a growing trend in the use of neuroscientific evidence in the United State of America, United Kingdom, Canada and The Netherlands[27,32,38,41]. The methodology for obtaining neuroscientific evidence with a strict observation of chain of custody often involves the structural brain imaging, electroencephalography (EEG) and neuropsychological assessment[41]. MAOA-L gene in relation to neuroscientific evidence has been demonstrated to be introduced by expert witness as an evidence in defense of an accused based on predisposition to the MAOA-L gene[42,43]. Courts, scientist, critics and psychologist around the world have deliberated on the use of neuroscientific and genetic evidence in criminal cases as a "double-edged sword"[38,41,44]. On one side, neuroscientific/genetic evidence portends a vindicating potential owing to the fact that it contributes to the reduction of culpability of defendant, and on the other side, it may act as an aggravating factor because it supports the assumption of future dangerousness[42]. However, the United States legal theorist Denno[27], who has investigated several real criminal cases in which biological evidence was introduced, considers the double-edged sword theory a mythos. Undeniably, neuroscience and genetic evidence is progressively being introduced in criminal cases in the United States[27,45,46] in Canada[32], Western Europe[38,41], and Australia[44].

In determining culpability, admissibility of evidence, sentencing, and the lowering of charges to lesser offence owing to genetic predispositions, legal system particularly in the USA considers the guilt phase, sentencing phase and the appellate phase. The guilt phase largely embroils assessment of the accused to determine a committal the criminal act charged ('actus reus'), while having the requisite, culpable mental state ('mens rea')[47]. The mens rea is usually recognized for each crime and differs in lessening degrees of criminal responsibility with the objective to ensure of proportionality in punishment[47]. Legal decision-makers must be convinced that the accused was unable to form the mens rea required for a specific offence to be committed[48]. Negating the mens rea of an offence renders the accused not guilty[49]. In the guilt phase, MAOA-L gene evidence was reported to be ruled admissible and a lesser sentence was charged[22]. A case where MAOA-L gene was considered admissible in the guilt phase was the case of the State v. Waldroup (2011). The accused was charged with felony murder of Bradshaw and attempted first-degree murder of his wife, which carried the death penalty. But, after he tested for the MAOA-L gene, the defense counsel presented the evidence and contested that the accused's genetic predisposition to aggressive behavior was a causal factor for the crime[49]. The court admitted the evidence and charged the accused a lesser infractions of voluntary manslaughter and attempted second-degree murder, the least serious criminal charges available to them[22,49].

The sentencing phase, is where the penalty for the criminal offense is determined. Although the legal systems vary, but courts consider statutory and non-statutory mitigating factors and aggravating factors at sentencing[27,50]. As per the appellate phase, a reduction in sentence was also granted following a presentation of a genetic evidence for post-conviction appeals[51,52]. The first reported case where the MAOA-L evidence

resulted to lesser sentence was the case of Bayout v. Francesco (2009). The accused was convicted of assaulting another man to death owing to the fact that he misleadingly believed the victim had assaulted him previously in a wanton attack. He was afterward sentenced to nine years in prison. On appeal, new mitigating evidence was presented by a molecular neuroscientist who told the court the accused carried the MAOA-L genotype and the appeals court granted a one-year reduction of his sentence[53]. As the result of the complexity of genetic evidence, the jury in the Bradley Waldroup's case stated that "the more the genetic evidence is presented before a jury, the greater the possibility of diverting the attention away from the facts and focusing onto the other aspects of the case[52].

CONCLUSION

Genetic factors signify one source of impact on criminal behavior. Till in recent times, genetic link to criminal behavior was relegated to the background. Emerging findings and evidence indicates that without recourse to genetics, there would be a limited opportunity in elucidating the mechanism and reason for criminal, aggressive and antisocial behavior of some individuals. In this light, reports from extensive studies suggest a strongest link between genetic variation and aggressive behavior is implicated in monoamine oxidase A (MAOA). Therefore, we recommend that they should be an early molecular testing to determine individual who are at increased risk of certain negative outcomes, and the identified individuals who are at increased genetic risk for criminal offending should be placed on environmental buffers such as educational programs or gene editing to reduce the risk that this genetic predisposition will be expressed.

ACKNOWLEDGEMENTS

Authors are grateful to Centre for Forensic Programmes and DNA Studies, University of Benin, P.M.B. 1154, Benin City, Nigeria, for providing an excellent theoretical and practical exposition on Forensic Science.

AUTHOR'S CONTRIBUTION

Nwawuba SU: conceptualized and designed the article, drafted the article, critical revision. **Divine O:** Drafted the article, and critical revision. **Edeaghe E:** data curation, investigation. The final manuscript was read and approved by all authors.

DATA AVAILABILITY

Data will be made available on reasonable request.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this article.

REFERENCES

1. Jasmine AT, Sarnoff AM. Genetic Factors and Criminal Behavior. Federal Prob 2000; 64(2): 24-27.
2. Nwawuba SU, Akpata CBN. Awareness level on the role of forensic DNA database in criminal investigation in Nigeria: A case study of Benin city. J Forensic Sci Res 2020;4:007-014. *https://doi.org/10.29328/journal.jfsr.1001019*
3. Nwawuba SU, Mohammed KA, Adams TB, *et al*. Forensic DNA Profiling: Autosomal short tandem repeat as a prominent marker in crime investigation. Malaysian J Med Sci 2020; 27(4):22–35. *https://doi.org/10.21315/mjms2020.27.4.3*
4. Nwawuba SU, Momoh SM, Nwokolo CC, *et al*. Key DNA profiling markers for identification: A mini review. Pharm Pharmacol Int J 2021;8(6):337–343.
5. Wertz JA, Caspi1 DW, Belsky AL, *et al*. Genetics and crime: Integrating new genomic discoveries into psychological research about antisocial behavior. Psych Sci 2018; 29(5):791–803. *https://doi.org/10.1177/0956797617744542*
6. Visscher PM, Wray NR, Zhang Q, *et al*. 10 years of GWAS discovery: Biology, function, and translation. The American J Human Gen 2017;101:5–22. *https://doi.org/10.1016/j.ajhg.2017.06.005*
7. McGrath LM, Mustanski B, Metzger A, *et al*. A latent modeling approach to genotype–phenotype relationships: maternal problem behavior clusters, prenatal smoking, and MAOA genotype. Arch Women's Ment Health 2012; 15(4):269– 282. *https://doi.org/10.1007/s00737-012-0286-y*
8. Caspi A, McClay J, Moffitt TE, *et al*. Role of genotype in the cycle of violence in maltreated children. Sci 2002; 297:851–854. *https://doi.org/10.1126/science.1072290*
9. Foley DL, Eaves LJ, Wormley B, *et al*. Childhood adversity, monoamine oxidase a genotype, and risk for conduct disorder. Arch Gen Psych 2004; 1(7):738–744. *https://doi.org/10.1001/archpsyc.61.7.738*
10. Widom CS, Brzustowicz LM. MAOA and the 'cycle of violence': childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behaviour. Biol Psych 2006; 60(7):684–689. *https://doi.org/10.1016/j.biopsych.2006.03.039*
11. Taylor SE. Criminal minds: The influence of the monoamine oxidase A genotype and environmental stressors on aggressive behaviour. Burgmann J 2013; 2:143-145.
12. Choe DE, Shaw DS, Hyde LW, *et al*. Interactions between monoamine oxidase A and punitive discipline in African American and Caucasian men's antisocial behavior. Clin Psych Sci 2014;2(5):591–601. *https://doi.org/10.1177/2167702613518046*
13. Kieling C, Hutz MH, Genro JP, *et al*. Gene– environment interaction in externalizing problems among adolescents: Evidence from the Pelotas 1993 Birth Cohort Study. J Child Psych Psych 1993; 54(3):298–304. *https://doi.org/10.1111/jcpp.12022*
14. Garpenstrand H, Longato-Stadler E, Klinteberg B, et al. Low platelet monoamine oxidase activity in Swedish imprisoned criminal offenders. European Neuropsycho-pharmacol 2002;12(2):135–140. *https://doi.org/10.1016/s0924-977x(01)00148-1*
15. Kolla NJ, Attard S, Craig G, *et al*. Monoamine oxidase A alleles in violent offenders with antisocial personality disorder: High activity associated with proactive aggression. Criminal Behav Ment Health 2014; 24(5):368–372. *https://doi.org/10.1002/cbm.1917*
16. Beaver KM, Barnes JC, Boutwell BB. The 2-repeat allele of the MAOA gene confers an increased risk for shooting and stabbing behaviors. Psych Quart 2014; 85(3):257–265. *https://doi.org/10.1007/s11126-013-9287-x*
17. McDermotta R, Dustin T, Jonathan C, *et al*. Monoamine oxidase A gene (MAOA) predicts behavioral aggression

following provocation. Proceed National Acad Sci 2009; 106(7):2118–2123. *https://doi.org/10.1073/pnas.0808376106*
18. Sohrabi S. The criminal gene: the link between MAOA and aggression (Review). BMC Proceedings 2015; 9 (Suppl 1):A49-A62. *https://doi.org/10.1186/1753-6561-9-S1-A49*
19. Eme R. MAOA and male antisocial behavior: a review. Aggr Violent Behav 2013;18(3): 395–398.
20. Huang YY, Cate SP, Battistuzzi C, *et al*. An association between a functional polymorphism in the monoamine oxidase A gene promoter, impulsive traits and early abuse experiences. Neuropsychopharmacol 2004; 29(8):1498–1505. *https://doi.org/10.1038/sj.npp.1300455*
21. Enoch MA, Steer CD, Newman TK, *et al*. Early life stress, MAOA, and gene environment interactions predict behavioral disinhibition in children. Genes, Brain Behav 2010; 9(1):65–74. *https://doi.org/10.1111/j.1601-183X.2009.00535.x*
22. McSwiggan S, Elger B, Appelbaum PS. The forensic use of behavioral genetics in criminal proceedings: Case of the MAOA-L genotype. Int J Law Psychiat 2017; 50:17–23. *https://doi.org/10.1016/j.ijlp.2016.09.005*
23. Shih JC, Chen K, Ridd MJ. Monoamine oxidase: from genes to behaviour. Annual Rev Neurosci 1999; 22:197-217. *https://doi.org/10.1146/annurev.neuro.22.1.197*
24. Cecilee PH. Born to Kill - Aggression genes and their potential impact on sentencing and the criminal justice system. SMU Law Rev 1997; 2:603-611.
25. Barkats M. Infantile spinal muscular atrophy - From gene discovery to gene therapy [SMA: from gene discovery to gene therapy]. Méd Sci (Paris) 2020; 36(2):137-140. *https://doi.org/10.1051/medsci/2020010*
26. Kannan A, Jiang X, He L, *et al*. ZPR1 prevents R-loop accumulation, upregulates SMN2 expression and rescues spinal muscular atrophy. Brain 2020; 143(1):69-93. *https://doi.org/10.1093/brain/awz373*
27. Denno DW. The myth of the double-edged sword: an empirical study of neuroscience evidence in criminal cases. Boston Coll Law Rev 2015; 56:493–551.
28. Bohman M. Some genetic aspects of alcoholism and criminality. Arch Gen Psych 1978; 35:269-276. *https://doi.org/10.1001/archpsyc.1978.01770270019001*
29. Mednick SA, Gabrielli WF, Hutchins B. Genetic influences in criminal convictions: Evidence from an adoption cohort. Science 1984; 224:891-894. *https://doi.org/10.1126/science.6719119*
30. Jones DN, Raghanti MA. The role of monoamine oxidase enzymes in the pathophysiology of neurological disorders. J Chem Neuro 2021; 114:101957. *https://doi.org/10.1016/j.jchemneu.2021.101957*
31. Tripathi RKP, Ayyannan SR. Monoamine oxidase-B inhibitors as potential neurotherapeutic agents: An overview and update. Med Res Rev 2019; 39(5):1603-1706. *https://doi.org/10.1002/med.21561*
32. Chandler JA. The use of neuroscientific evidence in Canadian criminal proceedings. J Law Biosci 2015; 2(3):550–579. *https://doi.org/10.1093/jlb/lsv026*
33. Ramsay RR. Molecular aspects of monoamine oxidase B. Progress Neuro-Psychopharmacol Biol Psych 2016;69:81-9. *https://doi.org/10.1016/j.pnpbp.2016.02.005*
34. Fowler JS, Logan J, Shumay E, *et al*. Monoamine oxidase: radiotracer chemistry and human studies. J Labell Comp Radiopharm 2015; 58(3):51-64. *https://doi.org/10.1002/jlcr.3247*
35. Vizcaíno C, Mansilla S, Portugal J. Sp1 transcription factor: A long-standing target in cancer chemotherapy. Pharmacol Therap 2015;152:111-24. *https://doi.org/10.1016/j.pharmthera.2015.05.008*
36. Emery HB, Mabel MJ, Koichi RK. Human GATA2 mutations and hematologic disease: how many paths to pathogenesis? Blood Adv 2020; 4(18):4584–4592. *https://doi.org/10.1182/bloodadvances.2020002953*
37. Akhtar W, Veenstra GJC. TBP-related factors: a paradigm of diversity in transcription initiation. Cell Biosc Cell 2011; 1:1-23. *https://doi.org/10.1186/2045-3701-1-23*
38. Catley P, Claydon L. The use of neuroscientific evidence in the courtroom by those accused of criminal offenses in England and Wales. J Law Biosci 2015; 2(3):510–549. *https://doi.org/10.1093/jlb/lsv025*
39. Kiive E, Laas K, Akkermann K, *et al*. Mitigating aggressiveness through education? The monoamine oxidase A genotype and mental health in general population. Acta Neuropsychiatrica 2014; 26(01):19–28. *https://doi.org/10.1017/neu.2013.34*
40. Åslund C, Nordquist N, Comasco E, *et al*. Maltreatment, MAOA, and delinquency: sex differences in gene–environment interaction in a large population-based cohort of adolescents. Behav Genet 2011; 41(2):262–272. *https://doi.org/10.1007/s10519-010-9356-y*
41. DeKogel CH, Westgeest EJMC. Neuroscientific and behavioral genetic information in criminal cases in the Netherlands. J Law Biosci 2015; 2:580–605. *https://doi.org/10.1093/jlb/lsv024*
42. Gonzalez DG, Merlin B, Susanne E, *et al*. Neuroscientific and genetic evidence in criminal cases: A double-edged sword in germany but not in the United States? Front Psychol 2019; 10:12-23. *https://doi.org/10.3389/fpsyg.2019.02343*
43. Dorfman HM, Meyer-Lindenberg A, Buckholtz JW. Neurobiological mechanisms for impulsive aggression: The role of MAOA. Springer; Berlin Heidelberg 2014; 297-313.
44. Alimardani A. Neuroscience, criminal responsibility and sentencing in an Islamic country. Iran J Law Biosci 2018; 5:724–742. *https://doi.org/10.1093/jlb/lsy024*
45. Denno DW, McGivney AA. What real-world criminal cases tell us about genetic evidence. Hastings Law J 2013; 64:1591-1681.
46. Farahany NA. Neuroscience and behavioral genetics in US criminal law: an empirical analysis. J Law Biosci 2015; 2:485–509. *https://doi.org/10.1093/jlb/lsv059*
47. Edersheim JG, Weintraub W, Brendel R, *et al*. Neuroimaging, Diminished Capacity and Mitigation. In: Simpson, JR., editor. Neuroimaging in Forensic Psychiatry: From the Clinic to the Courtroom. John Wiley & Sons, Ltd; Chichester, UK: 2012; 163-193.
48. Rushing SE. Admissibility of brain scans in criminal trails: the case of positron emission tomography. Court Review 2014; 50:62–69.
49. Baum, ML. The monoamine oxidase A (MAOA) genetic predisposition to impulsive violence: Is it relevant to criminal trials? Neuroethics 2013; 6(2):287–306.
50. Morse S. Gene-environment interactions, criminal responsibility, and sentencing. In: Dodge, KA., Rutter, M., editors. Gene-Environment Interactions in Developmental Psychology. Guilford Press; 2011; 11-26
51. Turone F. Medical tests help reduce sentence of woman accused of murder. BMJ 2011;343:d5761
52. Bradley BH. Can Your Genes Make You Murder? Special Series Inside the Criminal Brain 2010. Accessed on 11/6/2021
53. Forzano F, Borry P, Cambon-Thomsen A, *et al*. Italian appeal court: a genetic predisposition to commit murder? European J Human Genet 2010; 18(5):519–521. *https://doi.org/10.1038/ejhg.2010.31*