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Nature versus Nurture: Are Criminals Born or Raised? - A Comprehensive Analysis

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ABSTRACT

The genes comprised of deoxyribonucleic acid (DNA) has fascinated man ever since it was discovered in 1869. The connection of human gene with physical, intellectual and emotional attributes has generated a lot of interest in the recent times. Aggressive behaviour seen in some, might be triggered by genes and environmental conditions. Could this be the driving force that transforms aggression into criminal tendencies? This paper explores the association between genetic factors such as monoamine oxidase A (MAOA), serotonin, Klinefelter's syndrome, Jacob's syndrome, cadherin-13 (CDH-13), dopamine transporter 1 (DAT-1), dopamine receptor D2 (DRD2), catechol-O-methyltransferase (COMT), environmental factors and aggressive behaviour. The mechanism by which these genes work is like a domino effect, creating a chain of events that is highly complex to understand. In this paper we review the factors contributing to aggression with the help of real-life examples and comprehensive data analysis. Understanding the roots of a crime is pivotal to develop a modus operandi to curb crime rate.

Keywords-Aggression, Gene, MAOA, Serotonin, Klinefelter's syndrome, Jacob's syndrome, Cadherin-13(CDH-13), DRD2, DAT1, COMT

INTRODUCTION

Human behaviour has intrigued scientists for decades. Why do certain people behave in an antisocial manner under particular conditions? Is it due to their genes (nature), or the way they were brought up (nurture), or due to trauma, or is such behaviour because of an unknown reason? Historically, certain societies have always been accused of being more violent than others. Can it be due to genetic factors? Researchers have found that certain ethnic groups such as the Maoris, Jarawas, North Sentinelese, Onge and Shompen have heightened levels of aggression. This was found to be linked to the lower activity of monoamine oxidase A (*MAOA*) in their blood. A dysfunction in the cadherin activity has also been linked to alcohol dependence, violent behaviour, attention deficit hyperactivity disorder (ADHD) and many other psychiatric conditions. A normal karyotype 46, XX corresponds to female and 46, XY to male. Any deviation from the normal karyotype is called aneuploidy. Another study by Stochholm showed that certain aneuploidies like XXY and XYY have ended up in jail due to theiraggressive behaviour(Stochholm *et al.* 2012). The convicts also had lower IQ and didnot show remorse for the crimes they committed. Lower IQ was also found to be linked with Klinefelter syndrome, as the addition of each X-chromosome results in a drop in Intelligence quotient (IQ). The emotional and the genetic basis of a crime are very much interlinked for us to look at them individually. Once an individual faces a hazardous circumstance, he/she hasto make instant decisions, which might not allow them to assess the ramifications of their actions and respond appropriately. This might lead to detrimentalconsequences for the person, even resulting in their incarceration. Various data about genetic abnormalities, biochemical mechanisms, real-world implications and statistical data on the number of such individuals, behavioural genetics, case studies, and their association with criminality are discussed here.

LINK BETWEEN MONOAMINE OXIDASE A AND HUMAN BEHAVIOUR

Monoamine oxidase A, abbreviated as*MAOA* is a crucial gene that is responsible for the oxidative deamination of dopamine, serotonin (5-hydroxytryptamine), and norepinephrine. This gene, located on the X chromosome, is required to regulate hormones essential for maintaining an individuals' emotions, moods, and behavioural tendencies. The monoamines, 5-hydroxytryptamine and noradrenaline are converted into their corresponding carboxylic acid through an aldehyde intermediate. It controls the intraneural levels of 5-hydroxytryptamine (5-HT) and noradrenaline (Rang *et al.* 2015). The levels of serotonin are irreversibly linked to the levels of monoamine oxidase A. Happiness and well-being is linked to oxytocin and other hormonal secretions in one's body. The feeling of sadness or happiness is determined by the hormone that is secreted at a particular instance of time. A study found that lower expression of *MAOA* led to elevated happiness levels in women. Such results were not correlated in men (Chen *et al.* 2013). Males are three times more likely to have an *MAOA* mutation (Eme 2007). So *MAOA* cannot be the only determinant of happiness in humans. The expression of multitudes of genes and hormones are responsible for human emotions. Behavioural tendencies are difficult to grasp, but *MAOA* gives us a chance at understanding them. For decades, many were stereotyped to be 'born violent', but once scientists knew that genes were contributing to their violent behaviour, only then they understood that the problem runs deep. We have only scratched the surface in studying the link between genetics and criminology.

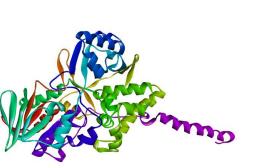


Fig. 1. The crystal structure of human monoamine oxidase A (Son et al. 2008)

In a study conducted by Cases, O., Seif, et al., a strong connection between serotonin and *MAOA* was found. *Maoa*deficient male mice with a C3H/HeJ genetic constitution have heightened aggression levels, which was caused due to the knockout of 5-HT receptors and lower activity of monoamine oxidase A (Cases *et al.* 1995). This study suggests that human males with lower activity of *MAOA* could probably demonstrate aggressive behvaiour. Furthermore, lower activity of *MAOA* also has been linked with alcoholism (Saito *et al.* 2002). These studies lend further evidence in reinforcing theconnection of genetic predispositions to criminal activities.

For example, several males in a specific Dutch kindred showed symptoms of borderline mental retardation, recursive and disturbed impulsive behaviour (Morell 1993; Appelbaum 2005). In anotherstudy done by Brunner in 1978, a woman explained how men in her family had violent sexual tendencies. The occurrence of this behaviour across several generations raised suspicionabout a medical reason behind it. This behaviour could also be explained by a theory called Operant Conditioning. When a person is made to perform a specific reversible activity by reinforcement techniques, they tend to repeat those activities throughout their lifetime, and it never skips their consciousness (Staddon and Cerutti 2003). The posterity of the violent Dutch family may or may not have a mutation in their MAOA gene. However, they could have violent tendencies as they have seen their father/grandparents/other male members in their family behave violently. This reinforcement of criminal tendencies is through operant conditioning. It can always be reversed by bringing up the child in a conduciveenvironment to the individual's growth. Identical twins with a mutant MAOAseparated at birth and raised in different environments tend to have different behaviours because defective MAOA is not the only reason for individuals to act in a particular way. The environment, situations and the family or whosoever raises the children have a prominent impact on their behaviour. Many criminals have put forth their genetic shortcomings as the sole reason behind their heinous crimes, but in most of these cases it has been found that the perpetrator is fully aware of the crime. About 10% of the criminals have found to have lower activity of the MAOA gene (Tiihonen et al. 2015). Many states in the USA have questioned whether genes play a role in criminal behaviour of an individual. The tendency of a criminal to become a recidivist also increases when the individual becomes aware that pleading guilty by insanity due to genetic abnormalities gets them a reduced sentence. A nonculpability verdict gives the offender another chance to execute another crime, which is like a vicious cycle. MAOAalso called the warrior gene holds the potential to clarify doubts on questionable human behaviour. Still, the implications are so vast that a singular conclusion is difficult to arrive at.

JACOB'S SYNDROME AND ANTISOCIAL BEHAVIOUR

47, XYY is a sex chromosomal aneuploidy affecting males, characterized by an extra Y chromosome. This disorder is also called Jacob's syndrome or YY syndrome. Males with this karyotype tend to be taller than average with a reduction in IQ. This condition is often associated with other behavioural disorders such as attention deficit hyperactivity disorder (ADHD), learning disorders and developmental disorders such as autism spectrum disorder (ASD). There is evidence of delayed milestones such as delayed speech, language problems, increased incidence of dyslexia and developmental delay due to this aneuploidy.

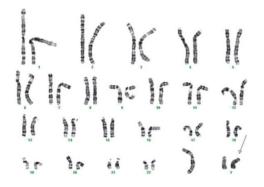


Fig. 2.karyotype of an individual with Jacob's syndrome (47, XYY)(Abdel-Razic et al. 2012)

This condition occurs in approximately 1 out of 1000 live births in men due to nondisjunction at meiosis II, resulting in a 47, XYY karyotype. In some cases, the additional Y-chromosome results from nondisjunction during cell division at the post-zygotic mitosis producing<u>mosaics</u>[46, XY/47, XYY]. In spite of the sex chromosomal aneuploidy, many XYY men are fertile. But the production of a hyper-haploid sperm increases the risk of transmitting the abnormality to the subsequent generations. Therefore, most of the men with XYY karyotype are diagnosed late or never diagnosed at all. Approximately only 15% of XYY individuals were found to be analysed in the Danish population (Stochholm *et al.* 2012). Lower IQ was found to be a significant factor contributing to criminal behaviour in XYY individuals. Though the crime rate was found to decrease by improving the socio-economic conditions of the XYY individuals, an authentic relationship between crime and socio-economic factors could not be correlated(Stochholm *et al.* 2012). Electroencephalography (EEG) helps in analysing the electrical voltages commonly known as brain waves that propagate along the pre-neural system. Oscillation in the brain wave activity is observed among antisocial individuals. It is also detected during the transition from wake to sleep state involving a change from fast wave activity to slow wave activity. Hakola & Iivanainen used pneumoencephalography in 1978 to demonstrate brain atrophy in five XYY men. EEG results indicate an elevation in the slow alpha frequency in XYY men, that specifies developmental defects in the sensitive portions of the central nervous system(MEDNICK *et al.* 1981).

Males exhibit a heightened fight-flight response compared to females. This is perhaps due to the difference between the XX and XY karyotype, the level of testosterone and also because of the man's nature to hunt alone or in small groups in the hunter gatherer cultureduring the Stone Age. There is a Sex Determining Region (SRY) on the Y Chromosome due to the presence of SRY gene that directs the embryonic gonads to develop into testes (Fechner 1996). The presence of SRY gene directs the development of the foetus into a male. However, the absence of SRY gene directs the development of the foetus into a female. Any mutation or deletion in part or whole of the Y-linked SRY gene has been observed in many 46,XY females who are infertile(Mutlu et al. 2015). Therefore, during embryonic development, the gender of the foetus is determined by the presence or absence of the SRY gene irrespective of the karyotype. SRY gene is not merely responsible for sex determination, but is also a regulatory gene expressed in the brain, kidney, heart, lungs, and the adrenal glands. According to the study of Daniel Ely, activation of SRY gene increases tyrosine hydroxylase (TH) promoter activity resulting in increased formation of dopamine and norepinephrine (Ely et al. 2007). Hence, the SRY gene is involved in catecholamine metabolism that drives stress responses.Positive emission tomography(PET) studies helps in assessing the functional activity of organs using radioactive markers. PET studies by Lee and Harley indicate that males show a higher dopamine release due to significantly increased number of neurons in the substantia nigra pars compared to females. SRY gene regulates tyrosine hydroxylase(TH) by binding to the TH promoter. SRY binds to the MAOA promoter, activating transcription and increasing the catalytic activity of MAOA. The biosynthesis of MAOA helps in regulating catecholamine metabolism.SRY gene mediates regulatory actions in the peripheral tissues, which results in an increased catecholamine secretion. Increased catecholamine secretion results in violent behaviour. The SRY gene also causes an increased dopamine and tyrosine hydroxylase release in the substantia nigra pars resulting in heightened aggression(Lee and Harley 2012). The gene expression of SRY is elevated in males with an abnormal karyotype 47, XYY, owing to the extra Y chromosome. Figure 3 represents how certain mutations hinder the MAOA mediated degradation or serotonin mediated inhibition of dopamine resulting in aggressive behaviour. Other Y-linked genes may also contribute to the aggressive behaviour encountered in 47, XYY karyotype.

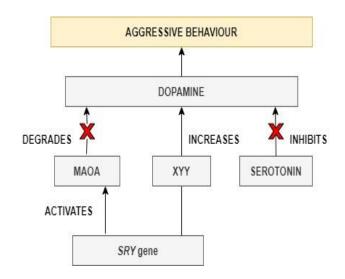


Fig.3.The association between dopamine and aggressive behaviour (Ely et al. 2007; Lee and Harley 2012)

ROLE OF CADHERIN-13 IN ABNORMAL BEHAVIOUR

Unlike the members of the cadherin superfamily, which have a transmembrane and cytoplasmic domain, cadherin-13 also referred to as T-cadherin or H-cadherin is a glycosylphosphatidylinositol anchor. Since cadherin-13 is detached from the cell membrane, it is considered a regulatory molecule and does not aid in adhesion. Cadherin dysfunction has been identified as a risk factor for attention deficit hyperactivity disorder (ADHD), schizophrenia, alcohol dependence, violent behaviour, and other psychiatric conditions(Rivero *et al.* 2015).

Fig. 4. Structure of cadherin-13 obtained through NMR spectroscopy (Dames et al. 2008)

Cadherin - 13 is localized to the parvalbumin and somatostatin-expressing gamma amino butyric acid (GABA) neurons in the stratum oriens of the hippocampus. GABAergic neurons regulating the inhibitory synapse in the brain are regulated by the interaction between cadherin-13, integrin B1 and B3. A mutation in the cadherin-13 (CDH-13) gene increases the basal inhibitory synaptic transmission to the hippocampal subfield, Cornu Ammonis-1 (CA-1) pyramidal neurons. It disrupts the E/I (Excitation / Inhibition) ratio leading to neuro-developmental disorders, increased aggression, learning disability and violent behaviour (sangadah 2020).

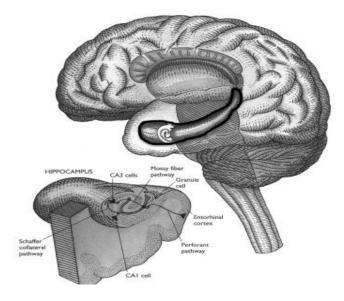


Fig.5.Hippocampus (Spencer and Bland 2019)

ANTISOCIAL BEHAVIOUR AMONG KLINEFELTER INDIVIDUALS

Klinefelter syndrome or 47, XXY karyotype is a sex chromosomal aneuploidy characterized by an extra X chromosome in males. Although the incidence of a higher grade abnormality such as 48 XXXY, 49 XXXXY is high, they are often aborted. The probability of having a Klinefelter child increases with maternal age. Males with this karyotype tend to have poor motor coordination, less body hair, breast growth, mild cognitive impairment, language impairment, emotional immaturity, low IQ and a higher risk of breast cancer.

This common chromosomal abnormality affects 1 out of 1000 live males, where the extra X chromosome is obtained from either of the parents due to nondisjunction during embryonic meiosis. A mosaic variant (46 XY/47 XXY) is produced in case of a nondisjunction event during mitosis, which expresses a less severe form of the abnormality depending on the ratio of abnormal to normal cells(Jonathan Posner and Bradley S. Peterson 2008). Approximately 90 - 95% of the Klinefelter men are infertile owing to their congenital condition of hypogonadism. The Klinefelter syndrome is severely underdiagnosed as significant symptoms are evident only after the onset of puberty. It was found that only 10% of the Klinefelter individuals were diagnosed prenatally and 25% were diagnosed during the later stages of infancy in the North Thames region (Richard-Devantoy *et al.* 2014). Another primary reason for the underdiagnosis of this condition is the high frequency of mild phenotypes (Bonomi *et al.* 2017). The Klinefelter males are presented with long legs and tall stature due to the X-linked short-stature homeobox gene (*SHOX*)(Descartes *et al.* 2014). SlowerElectroencephalography (EEG) frequencies predominate during childhood and gradually increase as the brain develops. Criminals are expected to have a similar EEG frequency to normal children. In XXY males a rise in slow alpha wave frequency, represents a probable developmental defect in the central nervous system. Since alpha waves decrease with relaxation and increase with a state of tension, the elevation in alpha wave frequency among XXY individuals could be the basis for their antisocial behaviour (MEDNICK *et al.* 1981). The crucial question is whether the EEG slowing precedes antisocial behaviour or is it the aftermath of the antisocial behaviour? A study done by Sarnoff A. Mednickestablished a relation between antisocial behaviour and theft in 10–13-year-

old Danish boys. If Klinefelter syndrome is coupled with an X-linked MAOA disorder, the risk of developing Type-2 alcoholism increases(Saito *et al.* 2002).

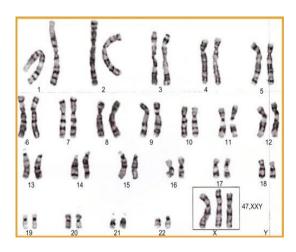
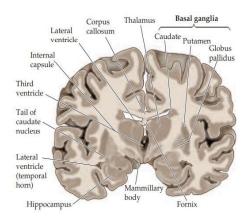


Fig. 6. Klinefelter 47, XXY karyotype (Hutaff-Lee et al. 2013)

Characteristics	Type 1 Alcoholism	Type 2 Alcoholism		
Onset age (years)	After 25	Before 25		
Loss of control	Frequent	Infrequent		
Guilt and fear	Frequent	Infrequent		
Fighting and arrest	Infrequent	Frequent		
Table 1 Difference between Type-1 and Type-2 alcoholism (Gupta and Basu 1999)				

ROLE OF SEROTONIN IN AGGRESSION

Serotonin or 5-hydroxytryptamine (5-HT) is a neurotransmitter involved in reward prediction, learning, and memory. Besides regulating physiological processes like body temperature, sleep, appetite, pain and motor activity, serotonin also regulates advanced brain functions like cognition and behaviour. The high frequency of serotoninergic projections in the hippocampus and prefrontal cortex indicates the association of serotonin with learning and memory. Furthermore, the serotoninergic neurons were found to play a significant role in decision making and social relationships by the hippocampus (Dale *et al.* 2016). Mutations affecting normal functioning of the serotonin metabolism results in antisocial behaviour.



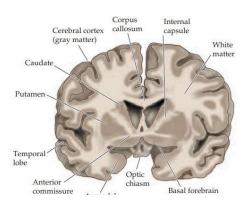


Fig. 7.Coronal view of the brain (Halterman 2005)

Brain-imaging is a novel discipline in neuroscience that explores the function and structure of the brain. Magnetic resonance imaging (MRI), functional magnetic resonance imaging (FMRI), positron emission tomography (PET) and electroencephalography (EEG) are some of the brain imaging technologies that help in diagnosis, prognosis, and treatment of psychological and neurological impairments. Brain-imaging studies show that widespread serotonergic abnormalities in the brain are associated with depression and suicidal tendencies. In contrast, serotoninergic deficits localized in the ventral prefrontal cortex are related to impulsive aggressive behaviour. A mutation in the serotonin transporter also called as 5-hydroxytryptamine transporter (5-HTT) is associated with violent and suicidal behaviour. Aggressive behaviour from childhood has been associated with a low concentration of 5-hydroxyindoleacetic acid (5-HIAA), the primary metabolite of serotonin. Research has also demonstrated that alcoholic and violent offenders have a lower concentration of 5-HIAA compared to the normal population (Seo et al. 2008).

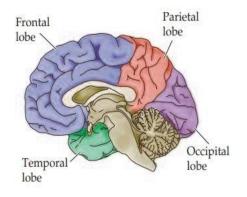


Fig.8. Four lobes of the brain (Halterman 2005)

Serotonin regulates emotion, behaviour, and aggression in the brain. Thus, serotonergic dysfunction is associated with antisocial behaviour. Appetitive aggression is described as the fascination towards violent criminal activites. While dopamine was found to trigger appetitive aggression, serotonin was found to inhibit appetitive aggression. The cell bodies and terminals of the dopaminergic neurons receive rich projections from the serotonergic neurons. Research has demonstrated that 5-hydroxytryptamine 2 (5-HT2) receptors were found to inhibit the activity of the dopaminergic neuron. A serotonin deficiency hinders the serotonergic function therefore resulting in hyperactivity of the dopaminergic system. Dopamine hyperactivity might secondarily trigger violent impulsive behaviour. This theory indicates an association between antisocial behaviour and the homeostasis between dopamine and serotonin (Seo *et al.* 2008).



Fig. 9.Crystal structure of serotonin bound to serotonin 1A (5-HT1A) receptor(Xu et al. 2021)

Table 2 A tabular representation of disabilities caused by the damage to a specific lobe of the brain(Halterman 2005)

DAMAGED LOCATION	DISABILITY	
Frontal lobe	Broca's aphasia (decreased motor ability to speak), apathy(lack of emotion and interest), delayed response, inattentive, reduced fluency of speech	
Temporal lobe	Impaired hearing ability, impaired memory, wernick's aphasia (inability to comprehend language)	
Occipital lobe	Loss of vision, visual hallucination	
Parietal lobe	Impaired sensation, right-left disorientation(inability to distinguish right from the left side), apraxia (inability to perform skilled task)	

GENETIC FACTORS CONTRIBUTING TO VIOLENT BEHAVIOUR

Modifications in the androgen receptor (AR) gene, dopamine transporter gene (DATI), and dopamine receptor D2 (DRD2) gene contribute significantly to aggression and antisocial behaviour. The building block of all nucleic acids is a nucleotide that consists of a sugar moiety, a phosphate group and a nitrogenous base. The primary nucleotides are adenine (A), guanine (G), cytosine (C), thymine (T) and uracil (U). Trinucleotide repeat expansion causes many genetic disorders such as fragile X syndrome, myotonic dystrophy and juvenile myoclonic epilepsy. CAG repeat expansion causes Huntington's chorea and spino-cerebellar ataxias. The X-linked androgen receptor (AR) gene inherited by men from their mothers was found to establish a negative relationship between the number of CAG repeats and aggressive behaviour. The androgen sensitivity was lower with an increase in the number of CAG repeats in the AR gene. The frequency of identifying a fewer number of CAG repeats in male perpetrators and rapists was found to be very high (Rajender *et al.* 2008). The catecholamine-O-methyltransferase (COMT) gene plays a significant role in dopamine catabolism and was found to interact with *DAT1* and increase the probability of aggressive behaviour(Butovskaya *et al.* 2013).

Reward signals produced by specific neurons help guide neurological activities controlling an individual's actions, decision making and choices. Dopaminergic neurons in the *substantia nigra* (SN) play a major role in reward prediction. A reward prediction error is the difference between predicted and actual rewards. Genetic predispositions that decrease the dopaminergic functions generate a negative reward prediction error that might trigger individuals to depend on risky experiences like alcoholism, substance abuse and violent behaviour(El 2016). The dopamine transporter 1 (*DAT1*), located in the short arm of the 5th chromosome, helps activate the dopamine receptor by regulating the intracellular dopamine level. It also aids in the reabsorption of dopamine into the presynaptic neuron. A variation in the number of tandem repeats in the *DAT1* gene is associated with aggression. A variation in a single position of a DNA sequence is known as single nucleotide polymorphism (SNP). The dopamine receptor D2 (*DRD2*) is often associated with aggressive behaviour due to single nucleotide polymorphism (SNP) of the *Thermus aquaticus* 1 (Taq1) involving a transition from thymine (T) to cytosine (C). This polymorphism of *DRD2* was observed in individuals exposed to substance abuse and alcoholism. Multiple gene interactions between *DAT1* and *DRD2* genes were found to contribute to aggressive behaviour significantly. An association between incarcerated parents and the subsequent arrest of their children for aggressive behaviour has been found due to *DAT1-DRD2* interaction(Butovskaya *et al.* 2013).

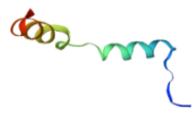


Fig. 10. Crystal structure of sodium-dependent dopamine transporter (DAT1) (Source: Swissmodel)

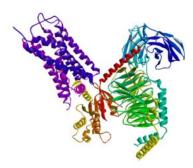


Fig. 11.Crystal structure of D2 dopamine receptor (DRD2)(Yin et al. 2020)

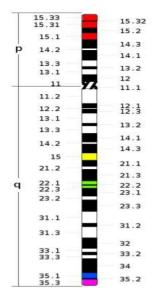


Fig. 12. Ideogram of chromosome 5Source: University of Washington Department of Pathology: ©1994

ROLE OF THE ENVIRONMENT IN CRIMINAL BEHAVIOUR

The University of Southern California's study of Risk Factors for Antisocial Behaviour (RFAB) on identical twins separated at birth, which is a classic example of nature versus nurture, revealed that the development of antisocial behaviour is contributed by the interplay between genetics, environment, biological and social factors. According to the study done by Chakraborty apart from genetic factors the environment in which the individual is raised influences aggressive behaviour (Chakraborty *et al.* 2012). An association between risk of child abuse, socio-economic and demographic factors in slum areas was found byRahayuand Hamsia. This study indicated that women and children in these densely populated areas were subjected to physical, emotional and verbal abuse (Rahayu and Hamsia 2020). Adolescence is a critical period of neural development specified by increased interaction between the different areas of the brain. This is the most vulnerable period of the development phase due to significant changes in the prefrontal lobe accompanied by cognitive and behavioural maturation (Arain *et al.* 2013). Growing up in an environment of familial violence including physical, verbal, psychological abuse and inadequate social bonding increases risk of antisocial behaviour. In addition, living in a locality with a high level of violent exposure encourages children to have violent role models.

An association between carbon monoxide, ozone, particulate matter and crime was established by Thomson and Youunan. It was found that an increase in pollution coupled with increase in temperature is associated with assault and theft crimes. The adrenal glands, located on top of both the kidneys, consist of the adrenal cortex and adrenal medulla. The adrenal medulla secretes catecholamines such as epinephrine and norepinephrine, which are responsible for driving stress responses. The adrenal cortex secretes mineralocorticoids like aldosterone that help maintain sodium(Na) and water balance. It also secrets glucocorticoids like cortisol that help regulate metabolic activities of the body. Particulate matter is found to trigger a stress response that directs the pituitary to release adrenocorticotropic hormone (ACTH), which signals the adrenal glands to increase the secretion of glucocorticoids. The binding of the glucocorticoid – cortisol to the glucocorticoid receptor (GR) leads to hippocampal atrophy and interference in the regulation of neurotransmitter factors like serotonin. The elevated secretion of glucocorticoids due to environmental pollutants might differentially affect the proliferation of neural progenitors, demyelination, and microstructural changes, leading to the loss of communication between different parts of the brain (Younan *et al.* 2016; Thomson 2019).

Violent behaviour is usually a learned behavioural response to real-life experiences. According to Sutherland (1947), criminal behaviour is learned within intimate personal groups through interaction with family and society. This learning involves imbibing motives, antisocial attitude and techniques

of committing a crime. Such motives are classified in the legal language as favourable and unfavourable. Unfavourable behaviours are mostly associated with the environment in which the individual is raised. Criminal behaviour was found to be the expression of the deprived needs and values of the individual(Sutherland 1947).

Conditioning is a concept of learning, where the behavioural response is associated with a stimulus from the environment. Operant conditioning is produced by a stimulus and maintained by the consequences. When such a stimulus is reinforced, it strengthens the response of the individual. Aggression could be directly influenced from operant conditioning through positive reinforcements where the presence of the stimulus increases the response. For example, it is a general perception that if a child watches a violent television show, he will behave violently. It is assumed that the stimuli triggering the behavioural response are the violent acts in the show, whereas the stimuli controlling the violent behaviour are the child's environment. Thus, these violent shows, games are associated with a negative behavioural response from the child, which therefore results in violent behaviour(Jeffery 1965).

The case study of Mary Bell(Downing 2016), a 11-year-old convicted murderer, revealed a strong association between environmental factors and aggressive behaviour. Mary Bell's father, Billy Bell, was arrested numerous times for petty crimes like burglaries. The fact that her mother was a prostitute made Mary a victim of moral disengagement and lack of social cognition. It was found that Mary's mother exploited her sexually during her childhood, therefore Mary acquired behavioural and mental patterns subconsciously that lead to the crime she had committed. Since she was attached to her mother as a protective factor, she targeted males and disfigured their genitals. Due to a deranged childhood, sexual abuse, poor role modelling from both her parents, Mary wrongfully perceived violence as a remedy against abuse and victimized men to protect herself and her mother.

Allele is an alternative form of a gene. The variation of allelic frequencies across different racial and ethnic groups due to migration from one environment to another hinders the reproducibility of genetic studies on aggression. In refugees, behavioural and cognitive problems may arise from the physical and psychological trauma experienced during their childhood in war-torn countries and refugee camps. Through operant conditioning, individuals exposed to sexual assault, substance abuse, child trafficking and war zone refugees behave aggressively in response to a stimulus produced from the violent environment they were living in. Recent studies indicate that genetic predispositions, environmental factors and their interaction might be responsible for antisocial behaviour that could later progress into violent criminal acts.

A HYPOTHETICAL PEDIGREE ANALYSIS

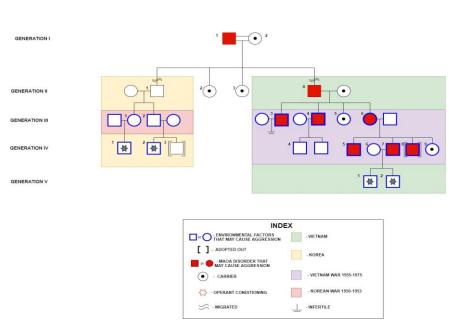


Fig. 13.A hypothetical pedigree representing the inheritance of an X-linked MAOA disorder and aggressive behaviour

We have constructed a hypothetical pedigree (fig 12) to explain the association between genetic, environmental factors and crime. The above pedigree represents the inheritance of an X-linked *MAOA* disorder and the representation of aggressive behaviour by individuals of a family across five generations. *MAOA* defect being an X-linked recessive disorder, the consanguineous marriage between the affected father I -1 and mother I -2 who is a carrier increases the risk of inheritance of by 31% (Shawky *et al.* 2013). The couple had four children, one unaffected male II -1, two carrier females II -2, II -3, and one affected male II -4. The two carrier females- II -2, II -3 have a 25% probability of passing on the defective *MAOA* gene to the next generation The unaffected II -1 male migrated to Korea, married an unaffected female, and gave birth to one unaffected femaleIII-1 and one unaffected male III -2. Having grown up during the Korean War (1950-1953), the unaffected female III-1 and unaffected male III -2 behaved aggressively owing to the environmental factors. However, they did not have the genetic disorder. II -4 migrated to Vietnam, married a carrier immigrant female, and gave birth to one affected XXY male III-3, one affected male III -4, one carrier female III-5, and an affected female III -6. Having grown up during the Vietnam War (1955-1975), the affected XXY male III-3, affected male III-4 and affected female III-6 behaved aggressively due to interaction of both the environmental factors and MAOA disorder. III-5 carrier female behaved aggressively mostly due to environmental factors, since she was a carrier, the genetic factor might have a very low probability of contributing to her aggressive behaviour. III-1 married an unaffected male and gave birth to one

unaffected male IV-1, though the Korean War ended before the IV generation due to the aggressive nature of the unaffected female parent III-1, through operant conditioning, IV-1 showed aggressive behaviour. The unaffected male III-2 married an unaffected female and gave birth to two unaffected males – IV-2, IV-3. As in the case of unaffected male IV-1, due to the aggressive nature of the unaffected parent III-2, the unaffected male IV-2 showed aggressive behaviour due to operant conditioning. Since IV-3 was adopted by another family, he did not experience operant conditioning. As a result he did not behave aggressively and grew up to be a normal individual. III-3 being an XXY male, was infertile and could not pass on the defect to the next generation. The affected male III-4 married an unaffected female and gave birth to two unaffected males – IV-4, IV-5. The carrier female III-5 had a 25% probability of passing the defect to the next generation. The affected XY males IV-7, IV-8 and a carrier female III-6 married an unaffected III-6 female were highly aggressive owing to both environmental and genetic factors. Being adopted out of Since the affected IV-6 XXY male is infertile, he could not pass on his genes to the next generation. The affected IV-7 male married an unaffected female and gave birth to two unaffected males V-1, V-2, though they lack the genetic disorder, they behaved violently due to the aggressive nature of both parents.

The environmental and genetic factors individually contribute to the aggressive behaviour of the individuals. The interaction between the two factors may steeply elevate the risk of aggressive behaviour in such cases. In this hypothetical pedigree, the Vietnamese and Korean War triggered individuals with and without genetic predispositions to behave aggressively. Even after the Vietnamese and Korean War, aggressive behaviour is likely to be found in the next generation. Though genetic factors and the War condition were absent, operant conditioning played a significant role in the aggressive behaviour of generation V in Vietnam and generation IV in Korea. Thus, there is no specific factor controlling violent behaviour but the interaction of more than one factor contributes to aggression.

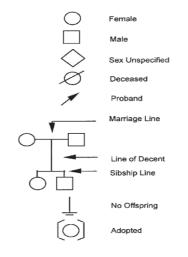


Fig.14. Common symbols for pedigree(Middelton et al. 2002)

A PICTORIAL REPRESENTATION OF THE BIOSYNTHESIS OF CATECHOLAMINES

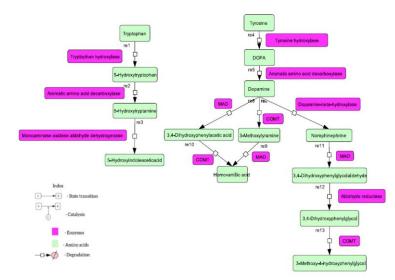


Fig. 15.Biochemical pathway linking dopamine, serotonin, MAO and Catechol-O-methyltransferase (COMT) (Andreou *et al.* 2014)(Andreou *<i>et al.* 2014)(Andreou *<i>et al.* 2014)[46][46](Andreou *<i>et al.* 2014)[46](Andreou *et al.*, 2014)(Andreou *et al.*, 2014)(Andre

The majority of the genetic disorders contributing to antisocial behaviour are due to the defective metabolic regulation of catecholamines. We have constructed the biochemical pathway of catecholamines (Fig 15) using the modelling tool - cell designer(Funahashi *et al.* 2008). The above-mentioned biochemical pathway represents tyrosine and tryptophan as the amino acids responsible for producing neurotransmitters such as dopamine, serotonin, and norepinephrine. Inhibition of the enzymes Catechol-O-methyltransferase (COMT) and monoamine oxidase (MAO) prevents the degradation of dopamine, resulting in the hyperactive dopamine system.5-Hydroxyindole acetic acid (5- HIAA), the primary metabolite of serotonin, helps the serotoninergic system inhibit dopamine. But the presence of a defective MAO enzyme will hinder the degradation of 5-Hydroxytrptamine (5-HT) to 5-Hydroxyindole acetic acid (5-HIAA), resulting in serotonergic dysfunction, which results in the hyperactivity of the dopaminergic system. Dopamine hyperactivity was found to contribute to aggressive behaviour secondarily. Hence, an inherited defect of the MAO, COMT or the other enzymes catalysing the biochemical pathway will hinder an individual's metabolic regulation, resulting in abnormal behavioural patterns.

DISCUSSION

The human race has always been obsessed with the genetic association to the crimes happening around the world. But, is there really a warrior gene present that drives humanity to orchestrate violent crimes? Contrary to the public opinion that situations drive a man to aggression, evidence states otherwise. Over the years XYY Males have been found to exhibit notorious behaviour. Many physical and mental characteristics have been linked to this phenotype, such as large deciduous teeth,tall stature, aggression, and low intelligence(Schröder *et al.* 1981). However, the high incidence of criminality in this particular phenotype is not found to be related to high aggression levels but instead found to be linked to their lower IQ(Witkin *et al.* 2016). During fight-or-flight situations, certain individuals might choose the violent path, and this may be due to their genetic abnormality.

Klinefelter syndrome is the most common genetic abnormality found in males, which occurs in about 1 in 1000 males, which is a lot more when compared to other genetic disorders. Klinefelter is distinguished by hypogonadism or relatively small testes. This is not due to the lack of testosterone but actually due to the lack of a second testicular hormone which facilitates the feedback inhibition and alters the level of pituitary gonadotropins. (Ferguson-Smith 2013). The extra X chromosome has been related to mental retardation, and in some instances, the X chromosome also gets inactivated, leading to several implications (Sands 1980). A particular case investigated by Pashayanshowed that a Klinefelter syndrome affected individual had severe mental retardation, bilateral epicanthic folds, and aniridia (malformed iris) which arethe consequences of the additional X chromosome present. This is why the human body inactivates the extra X chromosome, the process being named Lyonisation. Mammalian females have developed this unique process of dosage compensation to overcome this hurdle(LYON 1961). Klinefelter syndrome affected individuals also have a high rate of the criminality of about 64% (Schröder et al. 1981), which proves the direct link between mental retardation and criminality as seen in XYY before. MAOA or monoamine oxidase A is an enzyme whose activity results in the catabolism of serotonin and adrenergic activity. It has also been proved that children with a lower activity of the MAOA gene subjected to abuse and/or maltreatment are more prone to resort to violence (McDermott et al. 2013). MAOA is also termed the warrior gene, as it may give certain violent tendencies to those containing it. Certain violent offenders and Maori males possessing this particular gene have given rise to the question of whether the warrior gene has been identified (Gillett and Tamatea 2012). A large Dutch kindred were also found to have abnormalities in the MAOA gene. All men in this particular family showed impulsive aggressive behaviour, abnormal sexual behaviour, and arson (Brunner 1996). The general characteristics of these individuals were to have a withdrawn nature with less or no social life whatsoever, along with occasional violent outbursts. This evidence points out that a large population possessing the MAOA gene abnormality is a risk for future criminal behaviour. Conducive environments for such individuals will essentially reduce ill-fated outcomes.Lower levels of serotonin might lead to hormonal imbalances, thereby resulting in outbursts. This also has specific psychopathological applications in bulimia, aggression, and suicidal tendencies. The average rate of synthesis of serotonin in males was discovered to be 52% higher than in females, which might be due to the lower efficiency of the system transporting 5-Hydroxyindoleaceticacid out of thecerebrospinal fluid (CSF) in females than in males (Nishizawa et al. 1997). The marked difference between serotonin levels in males and females might be the leading factor why females are more prone to depression and suicidal tendencies (MELTZER 1990). The leading thought into all of this is not knowing what they are jumping into without thinking of the consequences due to the depressed state of mind. The individual as such may or may not have the ability to discern a particular situation and act aggressively. In an attempt to solve this hormonal imbalance, SSRIs (Selective Serotonin Reuptake Inhibitors) were prescribed. Paroxetine, also called as Aropax, Paxil, Pexeva, Seroxat was introduced in the US in 1992, and it gained publicity quickly as it proved to be the solution for people with anxiety and depression. Later it was discovered that Paroxetine lead to a 62% increase of breast cancer in women(Nevels et al. 2016). Paroxetine was found to cause the highest incidence of withdrawal syndrome among other drugs of its class. It was also found played a significant role in children attempting suicide (Wild 2005). Though it was the major SSRI prescribed for a long time, it was later discontinued due to the unfavourable side effects such as low sodium levels in the blood, teeth grinding and bleeding. So the real reason behind these individuals having depression or suicidal thoughts is not apparent. However, now it is very clear that there is a strong association between serotonin and human behaviour.

Table 3A table describing the physical and mental implications of genetic abnormalities (Morell 1993; Cases et al. 1995; Saito et al. 2002; Geerts et al.
2003; Appelbaum 2005; Seo et al. 2008; Chen et al. 2013; Skuse et al. 2018)

Karyotype	Genetic abnormalities	No. of individuals	Physical aberration	Behavioural aberration
46, XY 46, XX	No genetic abnormalities	7,412,410,064	No physical aberration	No behavioural aberration
46, XY 46, XX	MAOA deficient	449,239,693	No physical aberration	Alteration in happiness levels, heightened aggression, borderline mental retardation, regressive recursive, and disturbed impulsive behaviour
46, XY 46, XX	Serotonin deficient	26,400,000	No physical aberration	Depression, Violence, alcoholism, aggression and antisocial behaviour
47, XXY	Additional X chromosome	7,966,829	Muscle weakness, less body hair, breast growth, poor coordination and infertility	Low IQ, increased state of tension, Type-2 alcoholism, antisocial behaviour
47, XYY	Additional Y chromosome	3983414	Macrodontia (abnormal size of a tooth or a group of teeth), macroencephaly(large head), pes planus, clinodactyly(flat feet), ocular hypertelorism(increases distance between eyes) and abnormal side-to-side curvature	Low IQ, aggression, Hyperactivity, Attention Deficit hyperactivity Disorder, Autism, Learning Disorder, criminality

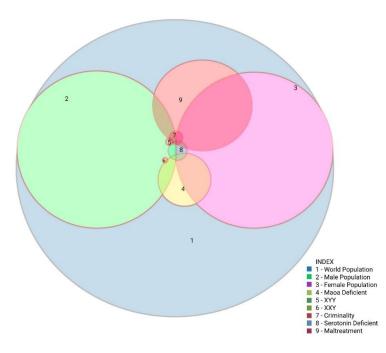


Fig. 16. A Venn diagram representing the link between genetics, environment and criminality(Schröder *et al.* 1981; Nishizawa *et al.* 1997; Saito *et al.* 2002; Appelbaum 2005; Kim-Cohen *et al.* 2006; Eme 2007; Wijlaars *et al.* 2012; Tiihonen *et al.* 2015)(rarediseases.org)

Figure 16is a Venn diagram explaining the association between genetics, environment, and criminality. The genetic abnormalities taken up here are MAOA deficiency, XYY(Jacob's syndrome), XXY (Klinefelter Syndrome), and serotonin deficiency. It can be seen that there is a strong association between maltreatment, criminality, and genetic disorders. The above mentioned data is sourced from studies conducted in various countries all over the world within a time span of 1992-2020. It has been noted that 1 in every 1000 males have XYY, whereas 1 in every 500 -1000 has XXY, making XXY the most common genetic disorder in males. MAOA deficiency was found in a greater subgroup of the population, but MAOA deficiency need not necessarily point towards an aggressive individual. Many of us will be having low activity of MAOA within ourselves, but we would have learned to live by coping up with it. The MAOA gene variant is estimated to be found in approximately 5.6% of the total population(Eme 2007). The majority of the numbers show that males are more prone to having a low activity of MAOA because women have two X chromosomes and thus face a lesser risk of having an active MAOA mutation. Males have a three times higherrisk of having an MAOA mutation than females(Eme 2007). The case becomes more interesting as we find anassociation between XXY and mutant MAOA gene. A study reveals that 3% of XXY individuals have a mutantMAOAgene(Saito et al. 2002), which links the two phenotypes, and individuals falling in this region have a great chance of exhibiting violent behaviour. Another study showed that 85% of males who have been subjected to maltreatment and had a lower activity of MAOA showed antisocial behaviour (Appelbaum 2005). This gives us proof that when people with genetic abnormalities get subjected to non-conducive environments, they can easily be provoked to unleash their violent phase. These incidents may be impetuous or may be meticulously planned, depending upon the situation the person faces. A child who does not have any genetic abnormality may also resort to violence, as environmental factors significantly impact. The situations and parents with whom a child is being brought up have a massive influence on the child's thoughts and actions. A child prone to high negativity and less affection through violent experiences have a considerable risk of becoming a criminal (Labella and Masten 2018). That is precisely why maltreatment stands as one of the significant reasons for criminality. Oppressed people have no way other than to oppose and come out of the arduous situation aggressively, or they might become victims forever. This is also why more African Americans tend to be recidivists (Jung et al. 2010). Certain societies have long been suppressed, and violence might be their only way out. As mentioned by Young I.M, the five faces of oppression continue to have an enormous impact on the human minds driving them to desperate measures to confront their perilous circumstances(Young 2011). So as long as maltreatment exists, innocent people would reach for the knife to defend themselves.

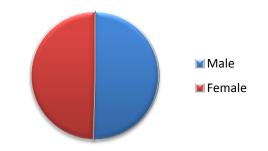


Fig. 17. The Population Sex Ratio of male vs female (Source: data.worldbank.org)

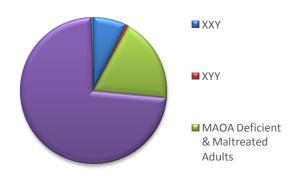


Fig.18. The subset of criminals having MAOA Deficient, maltreatment, XXY, XYY, XY and XX (Saito *et al.* 2002; Appelbaum 2005; Kim-Cohen *et al.* 2006; Eme 2007)

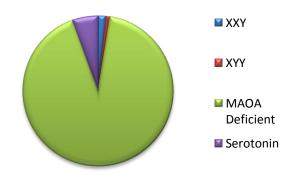


Fig. 19. The subset of population with genetic abnormalities XXY, XYY, MAOA deficiency and serotonin mutation (Nishizawa *et al.* 1997; Eme 2007; Wijlaars *et al.* 2012) (Source: rarediseases.org)

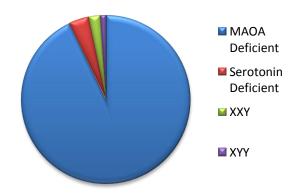


Fig. 20.Genetic abnormalities MAOA deficiency, serotonin deficiency, XXY and XYY present in the subset of male population (Nishizawa *et al.* 1997; Eme 2007; Wijlaars *et al.* 2012) (Source: rarediseases.org)

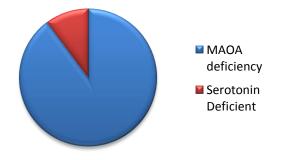


Fig. 21.Genetic abnormalities MAOA deficiency, serotonin deficiency present in the subset of female population (Nishizawa *et al.* 1997; Wijlaars *et al.* 2012)

Figures 17 – 21 represent data that segregate the world population based on Genetics, Criminality, Sex, and Environment. Most of the data has been sourced from studies conducted in the United States, as they encourage a lot of researches in this particular stream. Males have always been associated with aggressive behaviour, proved by the fact that almost 93.2% of the criminals as male, while females constitute a meagre 0.6%. (Ghiglieri 1999). But it is not always the case that males cannot control their aggression. About 5-10% of criminals have lower *MAOA* activity or *CDH13* gene defect(Tiihonen *et al.* 2015). Those with a mutant *MAOA* gene, when subjected to maltreatment, have a substantial risk of becoming criminal, as the genetic and environmental factors come together. Only when such situations happen, usually the genetic basis expresses itself in the behavioural genetics of an individual. A mere 15% of XYY are diagnosed with the condition, which in itself is a danger as those who are left undiagnosed may or may not grow up in conducive environments. They also have criminal tendencies in common, leading to a higher number of them being incarcerated(Schröder *et al.* 1981). It has been found that 64% of XXY have criminal tendencies (Schröder *et al.* 1981). Many have pleaded non-culpability after being charged with a crime by highlighting their genetic abnormality. Lawrence E. Hannell, a tall male with an XYY chromosome syndrome, was acquitted of a murder of a seventy-seven-year-old widow on the grounds of insanity (D.B. 1971). Several other cases reported worldwide have raised an important question: Whether genes drive a human to commit murder, or he does it with consciousness and then blame the gene for it? But XYY males are conscious of their actions (D.B. 1971), thereby not making them totally up for non-culpability. All of this adds up to only one solution; genetic counselling should be given to the predisposed individuals at the conducive age as an attempt to nurture them against their natural inst

CONCLUSION

A massive hike in crime rate across all cohorts in the past decade has urged researchers to investigate the correlation between genes and criminal activity. The presence of a genetic disorder does not necessarily mean the individual adopts a criminal lifestyle. Hence, genetic predispositions are not the only factors pertaining to crime and aggression. Aggressive behaviour is due to the interaction between a multitude of neurological, genetic disorders, and environmental factors. Based on the intensity of the biological disorders and frequency of exposure to environmental factors, the severity of the violent behaviour will vary. It was proposed that treatment, medication and therapy could provide promising results in controlling crime to a certain extent. Identifying these genetically vulnerable individuals is the most crucial step that precedes the implementation of behavioural control strategies. Early childhood diagnosis and intervention is likely to control the behavioural patterns of the genetically predisposed individual. This is because of the flexibility to change their behavioural pattern as children compared to adults. Raising a genetically vulnerable individual in a healthy and understanding environment will help tone down their aggressive nature. Unfortunately, most genetically predisposed individuals are not diagnosed during the conducive period or never diagnosed at all due to the lack of perceptible symptoms during their early childhood. After a detailed study on all the factors affecting crime and aggression, we propose implementing genetic testing for every individual born and genetic counseling for all the predisposed individuals and their parents. Diagnosis of genetically abnormal individuals is scarce in developing countries like India and several other underdeveloped nations. Spreading awareness on the benefits of genetic testing and counselling will help in diagnosing and nurturing the genetically abnormal individuals. Genetic testing provides information about the genetic constitution of the individual through techniques like new born testing, diagnostic testing, prenatal testing and preimplantation testing. Whereas genetic counselling will help the predisposed individuals understand their genetic vulnerabilities and adapt to a lifestyle that helps counterbalance their aggressive nature. Though there are other factors contributing to crime apart from the above mentioned aspects, genetic counselling will immensely help in curbing crime related to genetic and environmental predispositions. This strategy will help enhance the well- being of the predisposed individuals providing them an opportunity to have a better life.

Author Contribution

Poorvaja J and Rahul J conceptualized the paper and its structure. Poorvaja J and Rahul J wrote the paper, created the figures and did the editing. Radha Ramachandran added substantial additions to the paper.

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Competing interests

The authors declare no competing financial interests.

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