Genetic basis of aggression: Overview and implications for legal proceedings

Smilja Teodorović1*, Bogdan Uzelac1

Abstract: History and patterns of aggressive behavior represent an integral part of the offender's profile during forensic investigations. Traditionally, the majority of evidence on determinants of criminal behavior is contributed by environmental data. In the past two decades, a growing body of research in the fields of neurobiology and genetics has immensely enriched our understanding of the topic. This paper provides a systematic overview of genetic factors believed to govern the development of aggressive and criminal behavior in humans. A particular emphasis is given to the polymorphisms of genes involved in serotonergic and dopaminergic metabolisms in relationship to varying aggressive behavioral outcomes. In addition to approaches focused on individual genes, whole genome analyses, interplay between genetic factors, as well as gene-environment interactions, are also discussed with respect to this complex behavior. Finally, severity of incorporating these findings into the justice system, as well as the importance of considering them in contemporary criminalistics, are contemplated.

Key Words: aggression, violence, genetics, behavioral, genes, monoamine oxidase A.

Aggressive behavior, characterized by a conscious tendency to harm others against their will, can be exhibited through illegal, violent and antisocial behavior and is often present in criminal offenses. It is a complex behavior, regulated by multiple factors, including environmental, cognitive, neurobiological and genetic [1]. Twin and adoption studies played a significant role in behavioral genetics [2]. For instance, Rushton et al. assessed heritability and individual differences regarding five traits (aggression, altruism, empathy, assertiveness and nurturance) on 573 pairs of adult twins, suggesting that a genetic component is crucial in the development of aggression [3]. The progress in cytogenetics and molecular biology in the second part of the 20th century allowed examining relationships between genetic factors and aggressive and criminal behaviour, first at the chromosomal level and later at the level of single nucleotides. The intent of this review is to present key findings in the field and point out their applicability in the legal system.

Chromosomal aneuploidies

Jacob’s syndrome (XYY) affects one in 1000 males, most of which are educated normally and lead healthy lives, despite lower IQ. Given that society traditionally tends to view aggression as a male behavioral pattern, in the 1960s Patricia Jacobs and colleagues hypothesized that “supermales” with an extra Y chromosome will be overrepresented in a population of 197 psychiatric institution inmates, previously qualified as “mentally retarded” with dangerous, violent or criminal tendencies [4, 5]. The results revealed XYY incidence of 3.5% in examined population, compared to 0%-0.2% in the general population [6]. However, this study failed to take into account reasons for being in a mental institution (mental illness vs. aggression), history

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of abnormal behavior, type of criminal acts that the subjects are prone to, etc. Other studies confirmed the overrepresentation of XYY genotype in penal or mental institutions, but also suffered from sampling bias, due to preselecting affected males [7, 8]. By screening 34380 infants, Gotz and colleagues assessed 16 XYY males for antisocial personality disorder (APD) and 17 XYY males for criminal conviction frequencies and suggested that these measures were higher in affected males compared to healthy controls due to lower intelligence [9]. Additional longitudinal studies examining affected males from general population revealed that XYY men are not at greater risk of being incarcerated compared to healthy men, as early works suggested [10, 11]. In fact, several lines of evidence point out that the most common criminal acts among XYY males are proprietary offenses, thus not characterized by more violence [9, 12]. A recent longitudinal study from Denmark compared conviction rates for eight crime types between 161 XYY men and 15356 controls, ages 15-70 [13]. Results indicated a significant increase in violent criminal offenses (sexual abuse, homicide, burglary, violence and arson) in XYY men. However, when socioeconomic variables such as poverty, social exclusion and inadequate upbringing, as well as intellectual functioning were also taken into account, criminal patterns decreased to levels comparable to controls.

Klinefelter syndrome (KS) (XXY) is present in 1/581 to 1/917 individuals and is characterized by a specific phenotype [14]. In 1988, Miller proposed higher frequency of arson by KS individuals compared to general public [15]. Gotz et al. failed to confirm association between KS men and APD incidence, as well as criminal conviction rate, although small sample size must be noted [9]. The abovementioned Danish study also compared conviction rates between 934 KS men and 88979 healthy controls and reported an increase in sexual abuse, burglary and arson in KS individuals, although this could also be due to possible testosterone therapy [13]. Again, socioeconomic parameters reduced the observed risk to levels seen in controls.

Other chromosomal aneuploidies have also been considered in terms of violent behavior. For instance, 48, XXYY men were indicated to be more likely to exhibit aggressive and delinquent behavior in comparison to 48, XXXY and 49, XXXXY individuals [14]. Yet, it is clear that these rare chromosomal disorders could not account for the widespread violence, shifting the focus to putative candidate genes.

**Candidate genes**

Neurotransmitters monoamines (serotonin, dopamine, adrenaline, noradrenaline, histamine) can affect mood and behavior, as well as awareness, memory and sexual drive [16] and several lines of evidence suggested that serotonin deficiency is connected to impulsivity, violence and aggression [17]. Thus, numerous candidate genes have been researched in association with aggressive traits (Table 1), including genes involved in serotonin metabolism (5-HTT, TPH), dopamine metabolism (DRD2, DRD4, DAT1) and enzymatic degradation (MAOA, COMT).

**The infamous MAOA gene**

Monoamino oxidase A (MAOA) is a mitochondrial enzyme involved in deamination of excess monoamines. As impulsivity, mood swings, aggression, sleep disorders, depression and sexually deviant behavior have been noted in MAOA deficient individuals, MAOA gene variants have been hypothesized in development of violent criminal behavioral patterns [18]. Brunner et al. (1993) examined males in a large Dutch family with a history of borderline mental retardation and abnormal

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**Table 1. Key candidate genes discussed as risk factors for aggressive behavior**

<table>
<thead>
<tr>
<th>Gene name</th>
<th>Risk polymorphism/allele</th>
<th>SNP identification number</th>
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<tbody>
<tr>
<td>MAOA</td>
<td>C936T (exon 8)</td>
<td>rs72554632</td>
</tr>
<tr>
<td></td>
<td>30bp VNTR (promoter)</td>
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<tr>
<td></td>
<td>CA (intron 2)</td>
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<tr>
<td>S-HTT</td>
<td>44bp insertion/deletion</td>
<td>rs1800532</td>
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<tr>
<td></td>
<td>(promoter)</td>
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<tr>
<td></td>
<td>17bp VNTR (intron 2)</td>
<td>rs1799913</td>
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<tr>
<td>TPH1</td>
<td>A218C (intron 7)</td>
<td>rs6582071</td>
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<td></td>
<td>A779C (intron 7)</td>
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<tr>
<td>TPH2</td>
<td>haplotype (intron 5-8)</td>
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<td></td>
<td>rs1473473</td>
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<tr>
<td>S-HT-2A</td>
<td>C (promoter)</td>
<td>rs6311</td>
</tr>
<tr>
<td>COMT</td>
<td>Val158Met</td>
<td>rs4680</td>
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<tr>
<td>DRD2</td>
<td>TaqIA RFLP (downstream)</td>
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<tr>
<td>DRD4</td>
<td>C</td>
<td>rs3758653</td>
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<tr>
<td>DAT1</td>
<td>40bp VNTR (3’ UTR)</td>
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</tr>
<tr>
<td>BDNF</td>
<td>C</td>
<td>rs7103411</td>
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<tr>
<td></td>
<td>T</td>
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<tr>
<td>HTR1E</td>
<td>A</td>
<td>rs1406946</td>
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<tr>
<td>PNMT</td>
<td>T</td>
<td>rs2934966</td>
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*rs – reference SNP*
behavior (impulsive aggression, rape attempts, arson, and exhibitionism) [18]. Five subjects exhibiting the behavior possessed MAOA deficiency in vitro, due to a missense C936T mutation in exon 8. Although the genetic basis for the inability to control impulsive anger has been proposed, this rare single nucleotide polymorphism (SNP) has not been reported since. Yet, rodent studies report higher aggression rates, as well as maladaptive defensive reactivity and enduring responses, in MAOA deficient mice [19, 20].

Six variants (2, 3, 3.5, 4, 5 and 6 repeats (R)) of a 30bp MAOA upstream variable number tandem repeat (uVNTR) have been described in the promoter region[21, 22]. It has been suggested that 3.5R and 4R are optimal transcription activator elements, given that they result in significantly higher MAOA expression (MAOA-H) in vitro, compared to 3R and 5R (MAOA-L) [22], although contrasting findings have been reported for 5R [23]. In an attempt to link MAOA alleles with aggressive behavior, Manuck and colleagues genotyped 110 Caucasian males, suggesting that 3R and 5R carriers score significantly lower on aggressiveness and impulsivity, but not lifetime aggression or hostility, in comparison to 3.5R and 4R men [24]. Others indicated the protective/low risk nature of 4R allele [25], which persist across cultural settings, as 3R variant was associated with impulsive/antisocial behavior in 125 Brazilian alcoholics of European origin [26].

Beaver and colleagues examined 2196 individuals from the National Longitudinal Study of Adolescent Health and found that 2R and 3R male carriers are 1.94 times more likely to belong to a gang and 1.82 times more likely to use weapons for fighting, compared to 3.5R, 4R and 5R carriers [27]. In addition, 2R and 3R allele gang members were 4.37 times more likely to use weapons in comparison to gang members with other genotypes, which may explain the variability in levels of violence among gang members. A recent study on 49 violent and 40 nonviolent male prisoners showed that 2R and 3R alleles are significantly more present in incarcerated Caucasians who performed violent, as opposed to nonviolent crimes [28]. Importantly, this association was only marginally true for African American population, although it is unclear whether this is due to racial differences or sampling.

In an attempt to elucidate individual contribution by 2R and 3R alleles to aggressive traits, Guo and colleagues analyzed 2524 participants from the same dataset and reported that 2R carriers report at least twice as much delinquency in adolescence, young adulthood and adulthood compared to men with other alleles [29]. Increased violence, arrest and incarceration rate during lifetime [30], as well as increased risk of stabbing and shooting multiple victims during adolescence and childhood [31] have been reported for 2R carriers, as opposed to other genotypes, in African American males.

A comprehensive meta-analysis considering 31 independent studies on MAOA-uVNTR examined low activity/high risk (2R, 3R) and high activity/low risk (3.5R, 4R) genotypes, while data for 5R allele was treated as per original research [32]. The results show only a modest positive association between low activity alleles and antisocial behavior, the heterogeneity from different studies attributed to context-dependent role of low activity genotypes on antisocial behavior.

Another MAOA polymorphism of a dinucleotide, CA, repeat in intron 2 (MAOA-CAn), has also previously been described [33]. After eight MAOA-CAn alleles have been dichotomized into short (<114bp) and long (>114bp), Vanyukov and colleagues reported a lack of association between the repeat length and measures of aggressiveness or conduct disorder in adolescents [34]. Manuck et al. observed slightly lower aggression/impulsivity scores in men with long MAOA-CAn alleles, although this difference only approached statistical significance [24]. Interestingly, these results have been reported despite the linkage disequilibrium between MAOA-CAn and MAOA-uVNTR.

Finally, given that MAOA is X-linked, genetic variance in aggressive behavior has typically been pursued for males and not females.

Despite many associative findings, a study based on an in vivo imagining of MAOA concentration alone, disputed the association with uVNTR [35]. Similarly, Widom and Brzostowitz emphasized that MAOA genotype alone typically does not play a role in forming aggression in individuals [36]. Such results must also be considered, together with the fact that not all individuals with “risky” alleles/genotypes (if documented) will exhibit aggressiveness. Additionally, it is well documented in the literature that adverse environmental factors, such as childhood abuse, increase the chance of developing antisocial personalities or performing violent criminal offenses [37]. Taken together, these arguments emphasize the significance of accounting for adverse environmental conditions and genetics (GxE) when studying the basis of aggressive behavioral patterns.

Caspie and colleagues typed 540 Caucasian males from the New Zealand Dunedin Multidisciplinary Health and Developmental Study and assessed four antisocial behavior criteria (deviant behavior, conviction for a violent criminal offence, predisposition to violence and antisocial personality symptoms), reporting that 12% of examinees have low MAOA genotype/activity (3R and 5R) together with an abusive childhood and that 44% of all men sentenced for violent crimes belong to this category [38]. High MAOA genotype/activity males (3.5R and 4R), on the other hand, appear to be able to moderate their behavior, despite the childhood abuse [21, 39]. Many researchers have argued that MAOA genotype will predispose to aggression only in the particular environmental context, such as early traumatic
experiences, maltreatment, strict parenting style, etc. [21, 25, 26, 36, 37, 39-44], although conflicting reports exist [45]. Thus, a specific GxE model of behavioral outcome has emerged from the presented research, which appears to be restricted to male Caucasians, either due to varying childhood environmental factors or varying impact of MAOA-regulated polymorphisms on MAOA metabolism between races [46]. Importantly, McDermott and colleagues conducted a controlled experiment in which they observed a higher incidence of demonstration of aggressive behavior in low, compared to high, MAOA genotype/activity males, as response to financial loss [47]. These results were more pronounced when the extent of provocation was higher.

5-HTT Gene

Serotonin transporter recycles serotonin in the synaptic space, hence serotonin transporter gene (5-HTT) has been implicated in behavioral research. A 5-HTT gene–linked polymorphic region (5-HTTLPR) in the transcriptional control region of the gene exists in long (L), 528bp, and short (S), 484bp, forms due to a 44bp insertion/deletion [48]. As opposed to SS homozygotes and heterozygotes, LaLa homozygotes (~32% frequency) exhibit high transcriptional activity in vitro and two fold higher serotonin uptake [39, 48-50].

Beitchman and colleagues found that the incidence of genotypes contributing to low expression levels is significantly higher in a group of 82 healthy girls and boys, who have shown aggressive behavior for at least 2 years [51]. Retz et al. detected significant overrepresentation of SS genotype in Caucasian violent offenders, when analyzed 132 criminal and 21 civil offenders [52] and similar results have been reported for Chinese males [53]. Yet, as it has been proposed that only 5% of violent behavior can be attributed to the 5-HTTLPR [52], researchers have also analyzed behavioral outcomes of low expression genotypes in the presence of environmental stressors. In an analysis of 847 young Caucasians from Dunedin Multidisciplinary Health and Development Study, Caspi and colleagues reported greater suicidal tendencies (autoagression) (14%) in S allele carriers, when faced with repeated stressful life situations [39]. Conway and colleagues genotyped 381 mostly Caucasians, but also Asian Pacific Islanders and Aboriginals, and suggested that 5-HTTLPR genotype may contribute to aggressive response to chronic, rather than acute stress [54]. Others have corroborated these findings [42, 55]. To test for GxE, Cicchetti et al. investigated 627 children, using self-, peer- and adult behavioral reports of maltreated, low income subjects [56]. Maltreated children with LaLa genotype exhibited decreased risk for development of delinquent behavior, regardless of the abuse onset and timeframe. These results were confined to maltreated children, indicating that genotype alone does not predict antisocial behavior.

A meta-analysis which included 18 studies reported that there exists only a moderate positive association between 5-HTTLPR S allele and ASB and that studies which are based on small sample sizes tend to overrepresent the role of 5-HTTLPR [32]. On the other hand, some studies, while based on modest number of subjects, bring value in terms of the experimental approach. For instance, Beitchman and colleagues specifically typed for a third, rare allele, Lg, which results from a substitution of a single base in La leading to low transcription, suggesting that previous grouping of Lg with La could have contributed to curbing the S/La differences [51]. On the other hand, Ni et al. did not find an association between 5-HTTLPR and borderline personality disorder (BPD), even when considering Lg and S alleles together [57].

Another 17bp VNTR in 5-HTT gene intron 2 has been described and implicated in transcriptional regulation, such that 12R form acts as a significantly stronger transcriptional enhancer compared to 10R allele [58]. Analyzing subjects from familial schizophrenia, approximately 30% lower 5-HTT mRNA levels were observed in “low expressing” genotypes (10/10 and 10/12), compared to a “high expressing” (12/12), although the difference was not statistically significant. Higher frequency of a 10R allele and a lower frequency of a 12R allele, in comparison to controls, have been linked with BPD [57] and autoagression [59], although this individual effect is likely weak. Rather than making conclusions from allelic differences, when Hranilovic and colleagues analyzed haplotypes (high expressing at both loci, low expressing at only one locus and low expressing at both loci), potential combined effect of the two regions on 5-HTT gene expression was observed [50]. Ni and colleagues also argue in favor of the combined effect in increasing serotonin concentration in the synaptic cleft [57].

TPH1 and TPH2 Genes

Concentration of 5 hydroxy indol acetic acid (5-HIAA), one of the products of serotonin degradation by tryptophan hydroxylase 1 (TPH1), is considered a reliable indicator of serotonin turnover in the brain [18]. Manuck et al. genotyped 251 males and females and proposed that the presence of TPH1 SNP A218C corresponds to higher aggression levels, as well as perception and manifestation of unprovoked rage [24]. Another study addressed an A779C transversion which contributes to an “upper” (U) and “lower” (L) allele, and is in strong linkage disequilibrium with A218C, in 154 healthy subjects and 86 suicide attempters of German origin [60]. Differences in allele/genotype frequencies were not reported in two examined groups. Yet, U allele carriers were associated with higher state and trait anger, as well as angry temperament, indicating the contribution of TPH1 gene in manifestation of aggression. A recent meta-
analysis based on 37 studies and both polymorphisms of interest yielded a positive significant association with autoaggressive behavior [61]. Yet, there also exist studies which observed a lack of this association [62, 63]. It should be noted that when Cicchetti and colleagues typed two SNPs in maltreated children, rs1800532 and rs1799913 resulting in G and T alleles, genotype alone did not show association with antisocial behavior [56]. Yet, when GxE interaction was considered, T allele carriers who have been maltreated were more likely to exhibit delinquent behavior, particularly if they fit the early onset/recent maltreatment profile.

A rate limiting enzyme in serotonin synthesis in the brain is triptophan hydrolase 2 (TPH2). Zhou and colleagues genotyped 1798 individuals from 4 populations (African American, US Caucasians, Finish Caucasians and American Indians), who were either healthy or characterized by previous suicide attempts, major depression and/or anxiety disorder [64]. The researchers identified a “protective” and a “risk” haplotype between TPH2 introns 5 and 8 (~52kb) for anxiety, depression and autoaggression in examined Caucasians and African Americans. This concurs with results seen by others, including association with BPD, cognitive impulsiveness in ADHD patients and aggression affect lability [65-67]. Conflicting findings have also been reported [61, 68], although direct comparisons are challenging, given that some studies utilized distinct set of markers.

Other candidate genes

A study on 203 suicide attempters and 363 healthy German subjects indicated that the C allele of the functional SNP rs6311 in the promoter of the serotonin receptor 2A (5-HT-2A) gene is associated with aggressiverelated behavior and less inhibition of aggression, while a CC genotype correlates to increased anger and aggressive-related behavioral patterns [69]. A more comprehensive study assessed 582 SNPs in 14 genes related to serotonin and ~60000 subjects, argues lack of association between nNOS disorders could significantly impact serotonin metabolism [75]. Others have shown that A2A2 DRD2 genotype is overrepresented in subjects who do not get involved in criminal behavior [78]. Analyzing 40bp VNTR of dopamine active transporter 1 (DAT1) gene, resulting in 9R and 10R alleles, in over 2500 adolescents and young adults, Guo et al. published that 10R carriers are twice as likely to get involved in serious and violent delinquent behavior [75].

Nitrogen oxide synthetase (nNOS) gene is involved in metabolism of nitrogen oxide (NO), neurotransmitter found in parts of the brain involved in emotional regulation [8]. nNOS knockout mice (nNOS-/-) exhibit notable NO deficiency in the brain and an increase in aggressive and inappropriate sexual behavior [79, 80], particularly in the context of social exclusion ("single-housed males"). The authors also illustrated NO and serotonin interaction in the brain, thus nNOS disorders could significantly impact serotonin metabolism.

Yet, a recent comprehensive meta-analysis, which synthesized 185 studies on 12 polymorphisms and ~60000 subjects, argues lack of association between analyzed candidate genes and violent and aggressive outcome [81]. These findings concur with an idea that complex behaviors are likely governed by hundreds of thousands of genes and point out that a quest for genetic determinants of aggression/violence requires different approaches.

Gene-gene interactions

Ten serotoninergic genes were tested in BPD patients and significant interactions between 5-HT2C and TPH2, as well as among 5-HT2C, 5-HTT, MAOA and TPH2 have been reported (Table 2) [63]. Another study investigated the role of GxGxE in development of criminality in 1819 Swedish adolescents [42]. 5-HTT gene showed interaction with MAOA and BDNF genes, with or without family maltreatment, while MAOA and BDNF genes revealed interaction only in the presence of
family maltreatment or sexual abuse. Additionally, the interaction between all three genes and family maltreatment or sexual abuse was found, with the most extreme haplotypes (MAOA LL, 5-HTT LL, BDNF Val-Val and MAOA SS/LS, 5-HTT SS/LS, BDNF Val-Met/Met-Met) scoring the highest for criminality (Table 2).

Researchers also addressed the interaction between DRD2 and DRD4 (48bp VNTR which can be repeated between 2 and 11 times) on the development of criminal behavior in 1725 young Americans and found association with major theft and burglary, gang fighting, but not physical assaults [82, 83]. DRD2 A1 and DRD4 alleles with 7R repeats or more are proposed as risk conferring and each of the two alleles appears to strengthen the effect of the other gene (Table 2). Similarly, Oades and colleagues considered joint gene influences and found that DRD4 C allele was overexpressed and HTR1E and TPH2 A alleles underexpressed in overt impulsivity [65].

Whole genome analyses

When whole genome analysis of 3963 individuals was performed, four polymorphisms displayed significant genome-wide association with childhood conduct disorder, two in intergenic regions on chromosomes 11 and 13, and two in C1q and tumor necrosis factor-related protein 7 (C1QTNF7) gene, but none in traditional candidate genes [84]. Tielbeck and colleagues focused on 1649 adults and found the strongest, albeit not significant genome-wide, association between antisocial behavior and chromosomes 5, 14, 15 and 21 [85]. Similarly, no SNPs or genes were identified to associate positively on a genome-wide scale. A gene for dual specificity tyrosine-phosphorylation-regulated kinase 1A (DYRK1A), one of the candidate genes for mental retardation, and its three SNPs (rs12106331, rs2835702 and rs2835771) showed the most association. Specific search for a link between antisocial behavior and previously reported candidate genes, including MAOA, did not yield any findings. A study on behavioral disinhibition (including risky and impulsive behavior) in 1901 adolescents reported lack of individual locus or SNP association with genome-wide significance [86]. Yet, seven new loci (MAGI2, NAV2, CACNA1C, PCDH9, MYO16, IQCH and DLGAP1) emerged in gene-based association tests.

It must be noted that although genome-wide association of individual genes/SNPs has not been detected, genetic contribution of behavioral variance is typically around or above 50% [85, 86]. Therefore, further whole genome analyses involving drastically larger sample size and the effects of adverse environmental factors will be of great benefit.

Consideration of genetic basis of aggressive behavior in criminal trials

Only 81 court cases in which genetic make-up impacted the imposed sentence for violent crimes occurred worldwide from 1994-2011 [87]. In several court cases in the US, such as People v. Tanner [88], defense attorneys attempted to prove lack of rational and moral judgment at the time of the crime due to XYY genotype [89]. In the case of Stephen Mobley, accused of murdering a restaurant manager during a robbery in 1991, defense appealed a death sentence requesting Mobley be tested for a MAOA mutation [90]. The court rejected the appeal, stating insufficient scientific validation. Two decades later, on the contrary, Davis Bradley Waldroup’s jail sentence was reduced from 1st degree murder to voluntary manslaughter, due to a MAOA deficiency in the brain [91, 92]. In 2007, Abdelmalek Bayout was on trial in Italy for stabbing to death Walter Felipe Perez, who allegedly provoked him on religious basis [93]. After Bayout was found to have “risky” forms of five genes (including MAOA) previously implicated in aggressive behavior in stressful situations, the judge shortened the jail sentence. Similarly, Stefania Albertani’s sentence for murdering her sister in 2009 was reduced from life in prison to 20 years, due to low MAOA activity and changes in gray matter of frontal cingulate gyrus (involved in controlling behavioral inhibitions) and insula (which has been linked to aggression) [93].
Previous examples illustrate a more liberal approach employed by Italian courts. Yet, as seen from genome-wide and interaction studies, single gene investigations can be quite misleading and expensive for the judicial system, given that complexity of elucidating determinants of aggressive behavior lies in the number of involved variables and their interplay (Genetic x Environmental x Neurobiological x Cognitive). In order to gain better scientific validation, it will be essential to increase the number of longitudinal studies and number of subjects, consider the function of hundreds of thousands of genes/the entire genome, possible protective effects of other genes, sustainability of results in all ethnic groups, differentiate between types of violence, impulsive vs. premeditated aggression, employ more objective criteria for evaluating aggression, rather than self-assessments, etc. Therefore, despite impressive progress in the field in the past two decades, it is essential that courts exercise extreme caution when considering the idea of amending criminal responsibility on the basis of genetic predisposition, such as to avoid overestimating the power of the results still premature for use in the legal system, due to lack of causation.

CONCLUSION

While genetics has gained increasing importance in criminalistics and forensics, the public stands divided between providing legal protection for individuals suffering from genetic abnormalities and preventing these aggressive individuals to repeatedly harm others. Even though the research in the field has not given a “one fits all” solution, data coming from whole genome and meta-analyses clearly argues that significant portion of variance in violent/criminal behavior stems from genetic predisposition [85, 86]. Therefore, it is essential to incorporate this topic into contemporary criminalistics, in an attempt to develop treatment and intervention plans, as well as preventive support, aimed at decreasing likelihood of exhibiting violent behavior and reducing the extent of violence.

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