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Review Article

Behavioral genetics and criminal responsibility at the courtroom

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ABSTRACT

Several questions arise from the recent use of behavioral genetic research data in the courtroom. Ethical issues concerning the influence of biological factors on human free will, must be considered when specific gene patterns are advocated to constrain court's judgment, especially regarding violent crimes. Aggression genetics studies are both difficult to interpret and inconsistent, hence, in the absence of a psychiatric diagnosis, genetic data are currently difficult to prioritize in the courtroom. The judge's probabilistic considerations in formulating a sentence must take into account causality, and the latter cannot be currently ensured by genetic data.

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1. Introduction

Worldwide sentence guidelines for judges permit punishment mitigations for defendants with demonstrated reduced mental capacity due to a psychiatric illness, whose intentionality and free will were reduced at the time of the criminal act. As a consequence the court may establish compulsory internment and treatment in a psychiatric institution, sentence reduction, or both.

Recent cases show that molecular behavioral genetics are currently becoming influential on courts. While lawyers during the 1990s advanced guilt limitation due to putative brain serotonergic deficiency of their clients, the courts mostly rejected their claims and the sentences were little affected by expert testimony of genetically-determined reduced ability to control one's own impulses [1]. However, in 2009, a judge of an Italian appeals court reduced by one year the prison sentence of an adult immigrant with schizophrenia who had killed another immigrant

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by mistake, believing that the victim was the one who was mocking him. The Court of Assizes of Appeal of Trieste supported that in accordance with the results of tests matching "numerous international studies", certain elements in the murderer's genetic code "significantly increased the risk that he would develop impulsive aggressive behavior". [2]. Since the murderer was found to be a carrier of a few genetic variants that existing literature data associated with a predisposition to aggressiveness, the court reduced the final sentence from nine years and two months to eight years [2–6].

This sentence took into account biological data to further back the clinical diagnosis of schizophrenia. The scientific basis for backing the judgment was wide, as it was derived from studies focusing on specific populations of patients, who all had a diagnosis of schizophrenia (for example, [7–16]), not from population studies. In the same year, a Tennessee court, accepting a Monoamine Oxidase A (MAOA) gene variant per environment interaction (MAOA-L × childhood abuse), reduced the charge of a defendant from first degree murder to voluntary manslaughter [17]. In fact, the jury felt not like giving a death penalty after a forensic psychiatrist produced evidence that the defendant had a "warrior gene" conferring him vulnerability in some conditions, and condemned the defendant to a 32-year imprisonment [18]. The defendant appealed twice to further reduce his penalty, but the judge rejected most of his arguments [19,20], sticking to the facts and not taking into account further genetic considerations.

Currently, several defendants attempt to blame their own genetic constitution for their crimes and to ask for penalty mitigation during court sentencing. This raises issues of both ethical and moral nature mainly concerning the influence of biological factors on human free will, the risk of incurring in a deterministic eugenic science, the distinction between scientific knowledge *sensu strictu* (epistemological) and knowledge in a broader sense (gnoseological), the problem of the interpretation of scientific research data, and the problem of the sentence and its reduction.

The Italian psychiatrist Lombroso was first to propose in 1876 an empirical biological theory of criminal behavior [21]. According to this theory, some people are born rather than become criminals, and manifest different characteristic "atavistic" physical traits, such as their cranial structure, nose size, jaw jutting, jug ears, skin wrinkles, tattoos, that result from a regression to a more primitive state of evolution, which may facilitate their identification. For Lombroso, only one third of the criminal population belongs to this group of people, while other "criminaloids", with just some of the atavistic traits of criminality, may be influenced by an adverse environment [21].

The debate focused lately on genetic and aggressive behaviors, with two different standpoints emerging. On one hand, some consider the baby as entering the world as a "*blank slate*" and that environment, not heredity, determines all behavior [22,23]. On the other hand, others go back to Lombroso, supporting that harboring a gene alteration is basic and that the environment just facilitates the expression of aggressive–impulsive behavior. The more deterministic theories, according to which the antisocial, violent, and aggressive behaviors of some criminals are completely genetically-triggered, are now almost completely been put aside, but the current revision of the Lombrosian concepts is not much of a theoretical advance, as they only take into account the recently emerged epigenetics, but this is only pushing the issue forward.

In this paper we will discuss a number of questions that arise from the use of behavioral genetic research data in the courtroom. A major problem concerns the relationships between specific genomic structures of defendants and their intentionality at the time of the criminal act (criminal responsibility).

2. Genetic determinants of aggression and impulsivity in humans

Several geneticists estimated that the risk of aggressive behavior, both reactive and proactive, is influenced by genetic factors by about 40–50%; different twin studies showed them to play a greater role in adulthood and in men, compared to women [24–27].

The risk of inducing antisocial behavior may grow exponentially when harmful genetic and environmental factors synergistically interact in the same (pathological) direction [28]. The enzyme monoamine oxidase A (MAOA), which degrades amine neurotransmitters, such as norepinephrine, epinephrine, serotonin, and dopamine, has been shown to play a key role in the regulation of aggressive behavior. In fact, its gene has been called "warrior gene"; inactivating mutations in its coding region significantly correlated with aggressive and impulsive behaviors [29]. Evidence of gene/environment interaction has been provided that carriers of the low-activity MAOA variant who were exposed to physical or psychological abuse in childhood have a significantly higher risk of impulsive, aggressive, and violent behaviors in late adolescence and early adulthood [30].

The polymorphism that was found to be mostly involved in aggression and violence is the variable number of tandem repeats (VNTR), which is located 1.2 kb upstream of the coding region in the MAOA promoter, and has an average of four alleles, with 3, 3.5, 4, and 5 30-bp tandem repeats.

Alleles with two repeats (low enzyme expression) showed correlations with increased levels of delinquency and violent behaviors, as compared to the other MAOA-VNTR variants, while the allele with 4-repeat (high enzyme activity) was correlated with lower levels of impulsive aggression in boys [31,32].

Serotonin transporter (5-HTT) is a transmembrane protein that allows the reverse transport of serotonin from the synaptic cleft to the presynaptic neuron. In the promoter region of the serotonin transporter SLC6A4 encoding gene, which is located on chromosome 17q11.2 [33], a functional insertion–deletion polymorphism 5-HTTLPR has been found. Differently from the small (S) allele of 5-HTTLPR, the long (L) allele contains a 44-bp insertion. The S variant correlates in mice with low expression of the transporter and a consequent reduction of the transport of serotonin [34].

Pavlov and colleagues (2012) [35] reported that the presence of the genotype SS can explain 5% of the inter-individual variance in aggressive behavior in humans. The S allele was significantly associated with increased aggression and impulsivity in children [36,37], adopted children [38], adolescents [39], girls and young women [40], cocaine-dependent individuals [41], and patients with personality disorders [42,43].

Other genes, including genes for the estrogen, androgen, and serotonin receptors, for tryptophan hydroxylase, for the dopamine transporter, beta-hydroxylase, and receptors, and catechol-*O*-methyl transferase have also been involved in the genetics of impulsive and aggressive behaviors (for a review, see [35]).

Taken together, these data suggest that human aggression levels may correlate with multiple genetic factors.

3. Genetic constitution, violent impulsive behavior, and criminal responsibility: the problem of the causal link

So far we have discussed the role of different specific genes and alleles involved in impulsive/aggressive behaviors. We will now expose the factors that do not permit to clearly establish a causal link between genetic constitution, impulsive/aggressive behaviors, and criminal responsibility in determining intentionality at the time of the criminal act. For example, for genuinely genetic