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Behavioural Genetics in Criminal Cases: Past, Present, and Future

NITA FARAHANY & WILLIAM BERNET

Abstract

Researchers studying human behavioral genetics have made significant scientific progress in enhancing our understanding of the relative contributions of genetics and the environment in observed variations in human behavior. Quickly outpacing the advances in the science are its applications in the criminal justice system. Already, human behavioral genetics research has been introduced in the U.S. criminal justice system, and its use will only become more prevalent. This essay discusses the recent historical use of behavioral genetics in criminal cases, recent advances in two gene variants of particular interest in the criminal law, *MAOA* and *SLC6A4*, the recent expert testimony on behalf of criminal defendants with respect to these two gene variants, and the future direction of behavioral genetics evidence in criminal cases.

Use of Human Behavioural Genetics in Criminal Cases

With increasing frequency, practitioners in the U.S. criminal justice system have introduced expert testimony regarding the biological predispositions of criminal defendants, thus far with limited success. With the scientific research still in its infancy, criminal defendants have encountered significant hurdles when introducing expert testimony into U.S. criminal courtrooms regarding behavioural genetics. These defendants have primarily failed in their attempts because of the inadequacy of the science, the theoretical incompatibility of the evidence with the claim advanced, or because of procedural issues in U.S. criminal law barring the introduction of such evidence.

In several cases, criminal defendants have introduced biological predisposition testimony in an attempt to negate the presumption they acted voluntarily in the commission of the crime. The majority of defendants to advance such claims have done so in the context of drug or alcohol addiction.[1] In these cases, the defendant claims to have acted involuntarily as a consequence of his drug or alcohol addiction, for which he had a genetic predisposition.[2] This claim has largely failed primarily because it is at odds with the firmly rooted position in the U.S. criminal law that voluntary intoxication cannot serve to excuse criminal conduct. In contexts other than addiction, however, American courts have demonstrated some willingness to entertain the claim that a defendant's biological predisposition negates the voluntary act prerequisite for criminal liability. In the 2004 case of Herman Henry 'Bud' Von Dohlen, for example, the Supreme Court of South Carolina found persuasive the argument that the defendant's mental disease, severe depression arising from a genetic predisposition, rendered the homicide a product of disease, disassociated from the will, rather than a voluntary criminal act by the defendant.[3] Von Dohlen was convicted and sentenced to death for the armed robbery and murder of a dry-cleaning shop employee he fatally shot in the back of the head.[4] In support of his claim for post-conviction relief, a psychologist testified that as a result of 'his altered mental state '[the murder] was not a volitional thing but out of his conscious awareness or

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control.''[5] On appeal, the court reversed the earlier court's denial of post-conviction relief, finding instead that the psychological testimony created a 'reasonable probability the outcome of the trial might have been different had the jury heard the available information about [the defendant's] mental condition.'[6]

Defendants have advanced related arguments to rebut the mental state (mens rea) element of the crime, although, based on a review of appellate records in U.S. cases, only a few defendants have offered evidence of a behavioural predisposition for this purpose. In one of the few recorded instances of such a claim in the U.S., *State v. Davis*,[7] the defendant argued that his mental condition, to which he was genetically predisposed, prevented him from forming the requisite intent to commit first-degree murder, reckless endangerment, or possession of a weapon on school property.[8] In support of this defence, he presented psychiatric testimony that he had a 'genetic predisposition' for depression and mental illness, shown by the history of severe depression in his family.[9] The jury rejected his claim,[10] and the court affirmed on appeal, noting that the objective manifestations of Davis's behaviour prior to and during the commission of the alleged crime properly informed the jury's determination of his mental state.[11] Although genetic predisposition testimony has likewise been introduced to establish the defendant acted in accordance with a mental disease or defect in support of an insanity defence, courts generally conclude the defendant could still appreciate the wrongfulness of his conduct and conform to the law.[12] Nonetheless, when such testimony is introduced to bolster expert diagnosis of a mental condition, defendants have had more success.[13]

The majority of criminal defendants to have introduced expert testimony regarding their behavioural predisposition in U.S. criminal cases have done so in an attempt to mitigate their sentence, rather than to excuse or justify criminal conduct.[14] When used as mitigating evidence, defendants argue that their genetic predispositions make them less culpable offenders because their behaviour arose not as a result of 'bad character' but from 'bad genes.' Although such evidence could be used along with other mitigating circumstances, several criminal defendants have relied on behavioural genetics as the principal theory of mitigation during sentencing.[15] For example, in *Crook v. State*,[16] the defendant argued that his organic brain damage predisposed him to fits of violence. On appeal, the Supreme Court of Florida vacated the defendant's death sentence finding that the defendant's brain damage should clearly have been weighed to determine the appropriateness of a death sentence. As the science develops, particularly in elucidating the relationship between specific genetic factors and behavioural outcomes, mitigation theories like this one will likely become more prevalent in U.S. criminal cases. To date, however, only a few experts have managed to link the defendant's general behavioural predisposition and his specific criminal act in question;[17] establishing the link between a general genetic predisposition and the ultimate criminal act will be essential for this evidence to have significant future relevance.

Current Developments in Human Behavioral Genetics Research and Violence: *MAOA* and *SLC6A4*

The above cases illustrate the use of expert testimony in U.S. criminal cases regarding a defendant's general genetic predispositions with respect to his criminal conduct. It

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is possible that these earlier attempts to introduce behavioural genetics in the criminal law may have been too simplistic. Earlier claims in U.S. criminal cases rooted in behavioural genetics sought to establish that a single chromosomal abnormality (XXY), or a mutation at single gene (*MAOA* knockout) could explain, or even excuse, violent criminal behaviour. Recent scientific research, however, has illuminated a more compelling understanding of the interplay between specific gene variants, environmental stressors, and violence. These new scientific discoveries may provide a more meaningful understanding of behavioural differences between individuals, and have a greater potential impact on criminal proceedings.

In 2002, a research team based in New Zealand published a seminal paper that proposed a mechanism through which a person's genetic makeup and childhood experience might combine through a gene-environment interaction (G x E) to increase an individual's risk of becoming violent or expressing antisocial tendencies as an adult.[18] Essentially, this research team concluded that individuals with a particular allele of the *MAOA* gene, together with a history of serious childhood maltreatment were more likely to manifest violent and antisocial behaviour as adolescents and adults.[19] Previous research made evident that although many abused children become violent adults, most do not. The researchers postulated that a child's genetic makeup might modify his susceptibility to maltreatment. Specifically, the researchers tested whether a functional polymorphism in the promoter region of the *MAOA* gene would characterize genetic susceptibility to maltreatment. They selected the *MAOA* gene for study, in part, because an earlier study had identified a mutation of the *MAOA* gene in a Dutch family with a history of violence in the males.[20] This mutation, which eliminated MAOA enzymatic activity, was linked to male antisocial behaviour. While the *MAOA* mutation in the Dutch family has since been demonstrated to occur only rarely, the polymorphism of the promoter region of the *MAOA* gene causes common variants in gene expression.

The *MAOA* gene – located on the X-chromosome – encodes the *MAOA* enzyme, which metabolizes neurotransmitters such as serotonin, norepinephrine, and dopamine. The promoter region of the *MAOA* gene has either four repeats (causing high activity of the MAOA enzyme) or three repeats (causing low activity of the enzyme). In the DMHDS study population, 63% had four repeats and 37% had three repeats. The research team determined the gene variant possessed by each study participant, as well as the incidence of childhood abuse for each participant. They ascertained that endowment with the 3-repeat allele of the *MAOA* gene together with childhood maltreatment was significantly correlated with violent antisocial behaviour in adolescents and adults. Consequently, they concluded that '[f]or adult violent conviction, maltreated males with the low-MAO-A activity genotype were more likely than nonmaltreated males with this genotype to be convicted of a violent crime by a significant odds ration of 9.8.'[21] These study findings were replicated by Foley et al. (2004),[22] Huang et al. (2004),[23] Jaffee et al. (2005),[24] and Nilsson et al. (2005).[25] Although each of these later studies used varying definitions of child maltreatment, violent behaviour, and genetic risk, they all concluded that there was a gene x environment interaction consistent with the research reported by Caspi et al.(2002).

A year after publication of their paper on *MAOA* and child maltreatment, the research team of Caspi et al. (2003) published a second example of a gene x environmental

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interaction.[26] In the second paper, they reported a functional polymorphism in the promoter region of the serotonin transporter gene. The official term for the serotonin transporter gene is SLC6A4, although it is sometimes referred to as SERT and 5-HTT. The SLC64A gene – located on chromosome 17 – encodes a protein that facilitates activity of the serotonin transporter system. The serotonin transporter facilitates re-uptake of serotonin from the synapse back into the neuron. The promoter region of the SLC64A gene can have either a 'long allele' or a 'short allele.' The long allele is correlated with high activity of the serotonin transporter system, while the short allele is correlated with low activity.[27]

The researchers sought to understand the genetic and environmental interaction between the *SLC64A* gene variants and stressful life events. Specifically, they were interested in examining why some individuals become depressed and suicidal when faced with stressful life events, while other subjects appear to be more resilient. They hypothesized that the long allele of the *SLC64A* gene served as functional protection for carriers against the effects of stressful life events. Caspi and his colleagues once again reported a sophisticated gene and environmental interaction highly correlated with the differences in coping with stressful life events. They concluded that '[i]ndividuals with one or two copies of the short allele … exhibited more depressive symptoms, diagnosable depression, and suicidality in relation to stressful life events than individuals homozygous for the long allele.'[28]

This research may have significant potential application for criminal law. In much the same way that genetic predisposition evidence has previously been presented, these more specific interactions provide a more detailed understanding of differences in human behaviour and potentially more compelling testimony for consideration by juries. For example, a defendant charged with a violent crime may claim his behaviour could in part be attributed to the interaction of his genes (e.g., the 3-repeat *MAOA* gene causing low activity of the MAOA enzyme) and his life experiences (severe child abuse). On the other hand, a prosecutor may claim that the defendant's genetic makeup simply means he is a 'born killer' and should surely be incarcerated.

Legal Precedents regarding *MAOA* and *SLC6A4*

To date, testimony regarding these research findings in U.S. criminal cases has been quite limited. In May 2004, the faculty of Vanderbilt Forensic Psychiatry (a component of the Vanderbilt University School of Medicine Department of Psychiatry in Nashville, Tennessee) started to include genetic testing as part of their comprehensive pre-trial forensic psychiatric evaluation of defendants charged with homicide. As of February of 2006, this team has conducted *MAOA* and *SLC64A* genotyping on nine men and one woman charged with first-degree murder. Since August 2005, this team has testified regarding *MAOA* and/or *SLC64A* genotyping of four defendants in U.S. criminal cases. The details of this testimony will be reported in a future publication, once the legal outcome of these cases has been resolved.

In earlier unrelated cases, one criminal defendant sought *MAOA* testing, while several other defendants have introduced claims based on serotonin levels. The 1994 criminal case of Stephen A. Mobley is the sole reported case in the U.S. referencing *MAOA* genotyping prior to the Caspi et. al. studies of 2002 and 2003.[29] At trial, Mobley, who

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was convicted of murder and other related offences, filed a motion seeking funds to hire expert witnesses to assess his potential deficiency in *MAOA* enzymatic activity, based on the then-recent studies suggesting 'a possible genetic basis for violent and impulsive behaviour in certain individuals,' and his family history of violence. The trial court denied Mobley's motion, finding that the link between *MAOA* and violence lacked scientific verifiability sufficient for either the guilt or sentencing phases of his capital trial.

SLC6A4 genotyping has not been referenced in any published U.S. case as evidence presented during trial. Instead, expert testimony regarding a defendant's serotonin levels, a more tenuous claim, has been introduced in several U.S. criminal cases, usually to establish a link between a defendant's low serotonin levels and impulse control or intermittent explosive disorder.[30] The defendants in these cases claim to suffer from an inability to form the requisite intent for the alleged crime, or claim to have diminished culpability for purposes of sentencing. The defendants in these cases have enjoyed some success, such as a reduction from first to second degree murder, or potential mitigating effect during sentencing. However, because the link between serotonin levels and violence or impulse control is poorly understood, these claims have only had limited success when challenged by other expert testimony.

In short, we stand on the cusp of the introduction of this new behavioural research into criminal trials. Expert testimony regarding the research on *MAOA* and *SLC6A4*, together with a presentation of the relevant environmental factors could play a significant role in criminal cases going forward.

Future Directions

We are not proposing that the science of behavioural genetics will favour either the defence or the prosecution in criminal trials. We are simply predicting that research in this area will flourish and will identify more interactions among specific genes and specific life experiences, which promote specific behavioural outcomes. As the data amasses, the conclusions regarding the biological contribution to behaviour will become more precise, and the degree of scientific probability will become more robust.

Criminal defence attorneys, for example, may seek to present testimony regarding behavioural genetics in several circumstances:

- As mitigating evidence during capital sentencing hearings;
- To bolster the argument that a defendant may have been unable to subjectively form the mental state required for a particular crime, particularly with respect to premeditation for first degree murder;
- As evidence to inform the defendant's competence to assist in his defence or to waive Miranda rights;
- In the juvenile justice system to demonstrate that the juvenile's behaviour was partly determined by factors that were beyond his control (such as his genes and his history of child abuse) and that may be treatable, to support retaining the case in juvenile court rather than moving to criminal court.

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The prosecution may also make use of behavioural genetics evidence. For example, the prosecution could rely on behavioural genetics evidence to suggest that a criminal defendant poses a continuing threat to society or to support a finding of future dangerousness. Prosecutors could also use such evidence to malign the jury against the criminal defendant. This likelihood has already been realized in one case,[31] where the prosecutor referred to the defendant's family history of crime during his closing statement to the jury as demonstrating that the defendant came from a 'family of crime.'[32] Although the court acknowledged that in some contexts, 'this statement might be inappropriate, as it might indicate (for instance) a genetic predisposition to crime,' in the case at hand the court was unconcerned because it considered the statement merely hyperbolic, not grossly denigrating.[33]

Consequently, behavioural genetics evidence may be a double-edged sword for criminal defendants. Indeed, recent opinions in U.S. cases demonstrate that the introduction of behavioural genetics testimony by defendants could be adversely interpreted. The opinion issued by the United States Court of Appeals for the Ninth Circuit in *Landrigan v. Stewart*[34] provides a stark example of this phenomenon. Jeffrey Landrigan filed a petition for federal habeas corpus relief, claiming ineffective assistance of counsel during the penalty phase of his capital case because his attorneys, following the defendant's explicit instruction, failed to present mitigating evidence during the penalty phase of Landrigan's trial.[35] Four years after sentencing, however, Landrigan argued that notwithstanding his instructions at trial, he would have cooperated had his attorneys attempted to offer mitigating evidence demonstrating that his 'biological background made him what he is.'[36] The Ninth Circuit found such testimony unmoving, holding instead that 'although Landrigan's new evidence can be called mitigating in some slight sense, it would also have shown the court that it could anticipate that he would continue to be violent.'[37] At this stage of scientific progress regarding behavioural genetics, and the limited treatment options that may be available, defence lawyers should carefully consider whether evidence of an alleged genetic defect would help or hurt the defendant.

We predict that in the future, genetic testing will play an increasingly central role in criminal trials. For example, new research designs make likely that specific groups of genes will be identified that contribute to the development of schizophrenia and bipolar disorder. Criminal defendant will likely seek testing for these gene variants to support a claim of legal insanity. Prosecutors may likewise use a defendant's lack of these gene variants to support the contention that the defendant is malingering a psychiatric disorder and therefore not legally insane. Alternately, genetic testing may play an increased role in the evaluation and disposition of sexual offenders. For instance, specific gene-environment interactions may be correlated with a predisposition toward sexual disorders such as paedophilia. Future research could be used to support the contention that individuals with these factors are more likely to be recidivists, while individuals without these factors be more likely to be rehabilitated with treatment. The defendant's genetic makeup could thus become a central issue with respect to parole or indefinite commitment decisions.

The future promises a deluge of gene-environment research on human behaviour, and such evidence has and will continue to be introduced in the criminal court room. Paul S. Appelbaum, recently concluded that, '[r]ecent research findings … suggest that

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behavio[u]ral genetics may be the next frontier for the world of criminal justice, and mental health professionals are likely to play a critical role in helping the courts make sense of the new date.'[38] The recent use of such evidence in the criminal courtroom suggests that his prediction is beginning to be realized.

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[1] See, e.g., id.; State v. Boushack, No. 94-1389-CR, 1995 Wisc. App. LEXIS 378, at *4–*8 (Wis. Ct. App. Mar. 21, 1995) (rejecting defendant's argument that that his genetic defect limited his self control generally and made his intoxication involuntary).

[2] E.g. United States v. Moore, 486 F.2d at 1150.

[3] Von Dohlen v. State, 602 S.E.2d 738, 743 (S.C. 2004), cert. denied, 125 S. Ct. 1645 (2005).

[4] Id. at 740.

[5] Id. at 742.

[6] Id. at 743.

[7] No. M1999-02496-CCA-R3-CD, 2001 Tenn. Crim. App. LEXIS 341 (Tenn. Crim. App. May 8, 2001).

[8] Id. at *18.

[9] Id. at *12.

[10] Id. at *19.

[11] Id. at *19–*26.

[12] Kenley v. State, 759 S.W.2d 340, 344–48 (Mo. Ct. App. 1988) (rejecting defendant's ineffective assistance of counsel claim because it was reasonable trial strategy for attorney to exclude psychiatric testimony regarding defendant's genetic background and childhood history of violence because it did not satisfy the legal requirements for insanity).

[13] For example, in Robinson v. Johnson, 151 F.3d 256 (5th Cir. 1998), an expert abstained from testifying at trial because he believed Robinson's behavior to be drug-induced. On appeal, the expert filed an affidavit that he now believed Robinson's behavior to be caused by schizophrenia rather than drugs, because of new evidence that Robinson's sister and other family members had been diagnosed as manic depressives and schizophrenics, demonstrating Robinson's genetic predisposition to the disease.

[14] E.g., People v. Sapp, 73 P.3d 433, 469–73 (Cal. 2003), cert. denied, 541 U.S. 1011 (2004) (introducing as mitigating evidence the defendant's psychological and neurological factors contributing to the homicide).

[15] Hill v. Ozmint, 339 F.3d 187 (4th Cir. 2003).

[16] 813 So.2d 68 (Fla. 2002) [hereinafter Crook I] (vacating death sentence for failure to consider Crook's brain damage and mental retardation as mitigating factors); 908 So.2d 350 (Fla. 2005) [hereinafter Crook II] (vacating death sentence after re-sentencing by finding the death sentence was disproportionate in light of evidence of extreme mitigation).

[17] E.g., id. (finding that an expert's testimony explained how the defendant's fit of rage exhibited in the homicide was causally related to his behavioural predisposition to rage and impulse control).

[18] A. Caspi et al. Role of Genotype in the Cycle of Violence in Maltreated Children. Science 2002; 297:851-854.

[19] These conclusions were drawn from the Dunedin Multidisciplinary Health and Development Study (DMHDS). The DMHDS followed about 440 male children from birth to age 26. The children were assessed every two or three years, providing the research team with a wealth of information regarding psychosocial variables including child abuse. For example, the researchers established that 8% of the children experienced 'severe' maltreatment, 28% experienced 'probable' maltreatment, and 64% experienced no childhood maltreatment.

[20] HG Brunner et al. Abnormal Behavior Associated with a Point Mutation in the Structural Gene for Monoamine Oxidase A. Science 1993; 262:578-580.

[21] Caspi et. al., supra note 18, at 853.

[22] DL Foley et al. Childhood Adversity, Monoamine Oxidase A Genotype, and Risk for Conduct Disorder. Arch Gen Psychiatry 2004; 61:738-744.

[23] Y Huang et al. An Association Between a Functional Polymorphism in the Monoamine Oxidase A Gene Promoter, Impulsive Traits and Early Abuse Experiences. Neuropsychopharmacology 2004;

29:1498-1505.

[24] SR Jaffee et al. Nature x Nurture: Genetic Vulnerabilities Interact with Physical Maltreatment to Promote Conduct Problems. Development and Psychopathology 2005; 17:67-84.

[25] KW Nilsson. Role of Monoamine Oxidase A Genotype and Psychosocial Factors in Male Adolescent Criminal Activity. Biological Psychiatry 2006; 59:121-127.

[26] A Caspi et al. Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene. Science 2003; 301:386-389.

[27] In the DMHDS study population, 31% of the subjects had two copies of the long allele (l/l homozygotes), 17% had two copies of the short allele (s/s homozygotes), and 51% had one copy of each allele (s/l heterozygotes).

[28] Id. at 386.

[29] Mobley v. State, 455 S.E.2d 61, 65 (Ga. 1995).

[30] E.g., Hall v. State, 2005 WL 22951 (Tenn. Crim. App. 2005); State v. Payne, 2002 WL 31624813 (Tenn. Crim. App. 2003); State v. Godsey, 2001 WL 1543474 (Tenn. Crim. App. 2002); People v. Uncapher, 2004 WL 790329 (Mich. App. 2004); Hill v. Ozmint, 339 F.3d 187 (4th Cir. 2003).

[31] Johnston v. Love, 940 F.Supp. 738 (E.D. Pa. 1996).

[32] Id. at 753 n.17.

[33] Id.

[34] 272 F.3d 1221 (9th Cir. 2001), reh'g en banc granted, vacated, 397 F.3d 1235 (9th Cir. 2005).

[35] Id. at 1224.

[36] Id. at 1228.

[37] Id. at 1228–29 (internal citations omitted).

[38] PS Appelbaum. Law and Psychiatry: Behavioral genetics and the Punishment of Crime. Psychiatric Services 2005; 56:25-27.

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