



Inherited proclivity: When should neurogenetics mitigate moral culpability for purposes of sentencing?

J Bradley Segal^{*†}

Harvard Medical School, Harvard University, Boston, MA 02115, USA

*Corresponding author. E-mail: jbradley_segal@hms.harvard.edu

ABSTRACT

Certain genes and neurobiology ('neurogenetics') may predispose some people to violent behavior. Increasingly, defendants introduce neurogenetic evidence as a mitigating factor during criminal sentencing. Identifying the cause of a criminal act, biological or otherwise, does not necessarily preclude moral or legal liability. However, valid scientific evidence of an inherited proclivity sometimes should be considered when evaluating whether a defendant is less morally culpable for a crime and perhaps less deserving of punishment. This Note proposes a two-pronged test to understand whether and when neurogenetic evidence should be considered to potentially mitigate an individual's culpability for criminal behavior. The first prong normatively assesses whether a defendant meets a threshold of having meaningfully managed his risk of harming others based on what he knew, or should have known, about his own proclivities to violence. The second prong considers the admissibility of the evidence based on whether the specific neurogenetic proclivity claimed by the defendant is relevant and adequately supported by science so as to be reliable. This proposed two-pronged test, beginning with an ethical threshold and followed by a scientific hurdle, can help judges and juries establish when to accept

[†]J Bradley Segal is an MD candidate at Harvard Medical School. He holds a BA in philosophy and BS in physiology & neuroscience from the University of California, San Diego. He is deeply indebted to Holly Fernandez Lynch and Robert Kinscherff for their invaluable comments and revisions to this manuscript. He is also grateful to Judith Edersheim, Amanda Pustilnik, and Fiery Cushman for inspiring interest in this topic at a seminar organized by the Harvard Medical School Center for Bioethics. Finally, he thanks June Casey for assisting with legal research.

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arguments for neurogenetic mitigation at sentencing, and when to reject them.

KEYWORDS: neurogenetics, culpability, behavioral genetics, sentencing, punishment

INTRODUCTION

In 2012, Alex Duran was a Private First Class in the Marine Corps when he awoke hearing voices in his head.¹ This was not the first time he heard these voices.² Duran grew up being physically abused.³ He slept with a knife under his pillow and was known to sporadically punch walls without reason.⁴ That night in 2012, Duran ran outside shoeless and attacked a guard using a homemade machete, striking him multiple times in the neck.⁵ In a general court martial,⁶ Duran was found guilty of attempted murder, maiming, and assault upon a sentry, for which he was sentenced to 15 years of confinement.⁷

In 2014, Duran appealed, claiming the failure by his defense counsel to investigate for the presence of genes associated with criminality prior to sentencing constituted ineffective assistance of counsel.⁸ The appeal argued that Duran's 'violent' upbringing exposed him to environmental risk factors scientifically known to bring out certain genetic proclivities for violence,⁹ and if a genetic cause for his behavior was known prior to sentencing, it would have constituted mitigating evidence and Duran's punishment would perhaps be less severe.¹⁰ 'We are in the second decade of the 21st century: behavioral genetics is and should be in the mainstream of the criminal justice system',¹¹ opined the defense counsel.

On a population level, individuals with particular gene-environment interactions ('GxE') have a significantly increased probability of perpetrating impulsive violent acts.¹² 'The center of th[e] academic debate is not about whether genes influence behavior but rather how they do so'.¹³ Catechol-o-methyltransferase ('COMT'), dopamine transporter 1 ('DAT1'), and serotonin transporter 5-HTTLPR are genes of

¹ United States v. Duran, No. 201200440, 2014 CCA LEXIS 38, at *1–22 (N-M. Ct. Crim. App. Jan. 31, 2014) (unreported case, though cited as persuasive authority under NMCCA rule of practice and procedure 18.2).

² Gabriel K. Bradley, Supplement to Petition for Grant of Review, in United States v. Duran, No. 201200440, 2014 CCA LEXIS 38, at *3.

³ *Id.*

⁴ *Id.* at *16

⁵ *Id.* at *4.

⁶ Members of a court martial (like a judge in civilian courts) are granted discretion to decide punishment for the convict's charges. 10 U.S.C. § 880, 924–8. Increasingly, behavioral genetics is introduced in courts for non-capital offenses in unexpected ways. See Nita A. Farahany, *Neuroscience and Behavioral Genetics in US Criminal Law: An Empirical Analysis*, J. L. & BIOSCI. 8, 12 (2016) (Advance access published Jan. 14, 2016, doi: 10.1093/jlb/lsv059).

⁷ Duran, *supra* note 1, at *1.

⁸ *Id.*

⁹ Bradley, *supra* note 2, at *12, *13 (citing Caspi et al., *infra* note 12).

¹⁰ *Id.* at *15, *23.

¹¹ *Id.* at *17 (internal citation omitted).

¹² Avshalom Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 SCIENCE 851 (2002).

¹³ Patrick Bateson, *The Corpse of a Wearisome Debate*, 297 SCIENCE 2212 (2002).

growing interest in the legal and bioethical literature on this subject.¹⁴ Monoamine oxidase A ('MAO-A') remains the best-understood and most-studied gene to play a role in violent behavior.¹⁵ In 2002, a team led by Caspi showed that 85% of individuals with the GxE combination of the 'low activity' MAO-A allele and a history of severe childhood maltreatment developed antisocial behaviors.¹⁶ While MAO-A was initially portrayed in the media as a 'warrior gene'¹⁷ and met with cautious skepticism by experts,¹⁸ evidence for the neurogenetic¹⁹ association with impulsive aggression has now garnered wider scientific acceptance.²⁰

Commentators generally agree that an inherited vulnerability to violent conduct should not influence the outcome of the guilt-determination phase of criminal proceedings.²¹ Simply put, such genes do not demonstrably cause people to lose appreciation for the wrongfulness of their actions, nor to lose the capacity to conform one's behavior to the requirements of the law.²² Instead, increasingly there are efforts to introduce genetic evidence during sentencing to convey that a defendant's inherited proclivities to criminal behavior constitute a mitigating condition.²³ Evidence of a mitigating condition is intended to diminish the defendant's moral culpability and therefore lessen the punishment imposed.²⁴

At issue is whether and when a court should be willing to admit neurogenetics evidence of a defendant's mitigated culpability during sentencing. This Note addresses the problem in two parts. Part I focuses on a normative prong: when 'should' the door be open to a claim for mitigated culpability based on a defendant's neurogenetic susceptibility to violent behavior? Part II explores a procedural prong: once that door is opened, what criteria must the specific neurogenetic proclivity claimed by the defendant satisfy in order to be deemed admissible at sentencing?

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- ¹⁴ Eg Paul S. Appelbaum & Nicholas Scurich, *Impact of Behavioral Genetic Evidence on the Adjudication of Criminal Behavior*, 42 J. AM. ACAD. PSYCHIAT. & L 91, 92 (2014).
- ¹⁵ Hayley M. Dorfman, Andreas Meyer-Lindenberg & Joshua W. Buckholtz, *Neurobiological Mechanisms for Impulsive-Aggression: the Role of MAOA*, 17 CURR. TOP. BEHAV. NEUROSCI. 297, 305 (2014).
- ¹⁶ Caspi et al., *supra* note 12, at 852 (statistic compared to individuals with neither risk factor).
- ¹⁷ Eg J. McFadden, *Warrior Blood*, THE TIMES (LONDON), July 10, 2004.
- ¹⁸ Eg Paul S. Appelbaum, *Behavioral Genetics and the Punishment of Crime*, 56 PSYCH. SERVICES 25 (2005).
- ¹⁹ Give the various scientific methodologies required to elucidate a gene's role in something as complex as human behavior, in this Note the term 'neurogenetic' is used to refer to evidence with elements of genetics (such as GxE's) and neuroscience (eg brain imaging). See Farahany, *supra* note 6, at 2, 3; John Pyun, *When Neurogenetics Hurts: Examining the Use of Neuroscience and Genetic Evidence in Sentencing Decisions Through Implicit Bias*, 103 CAL. L. REV. 1019, 1024 (2015).
- ²⁰ See Amy L. Byrd & Stephen B. Manuck, *MAOA, Childhood Maltreatment, and Antisocial Behavior: Meta-Analysis of a Gene-Environment Interaction*, 75 BIOL. PSYCHIAT. 1, 9 (2014); Dorfman, Meyer-Lindenberg & Buckholtz, *supra* note 15; Konstantin A. Pavlov, Dimitry A. Chistiakov & Vladimir P. Chekhonin, *Genetic Determinants Of Aggression And Impulsivity In Humans*, 53 J. APPLIED GENET. 61 (2012).
- ²¹ See Stephen J. Morse, *Genetics and Criminal Responsibility*, 15 TRENDS COGNITIVE SCIENCES 378-380 (2011); Nita A. Farahany & James E. Coleman, Jr., *Genetics and Responsibility: To Know the Criminal from the Crime*, 69 L. & CONTEMP. PROBS. 115, 125-146 (2006).
- ²² *Id.*; Eg Millard v. State, 261 A.2d 227 (Md. Ct. Spec. App. 1970) (rejecting insanity defense that was based on defendant's 'XYY' genotype).
- ²³ Deborah W. Denno, *Courts' Increasing Consideration of Behavioral Genetics Evidence in Criminal Cases: Results of a Longitudinal Study*, 2011 MICH. STATE L. REV. 967 (2011); Farahany, *supra* note 6, at 8, 12-20.
- ²⁴ For discussion of mitigation see Russell Stetler, *The Mystery of Mitigation: What Jurors Need to Make a Reasoned Moral Response in Capital Sentencing*, 11 U. PA. J.L. & SOC. CHANGE 237 (2008).

PART I. MORAL SIGNIFICANCE OF NEUROGENETIC PROCLIVITY

Genetic Determinism

Determinism can be characterized as the view that all events in the universe—including human actions—have a physical cause. ‘Genetic determinism’ is the idea that genes are the root cause of behavior,²⁵ which is reflected in media coverage that asks, ‘Can Your Gene Make You Murder?’²⁶ and ‘Are Some People Just Born Evil?’²⁷ Claiming that someone is less responsible because ‘my genes made my do it’ presumes that an individual’s moral culpability for an act is mitigated or excused by the presence of a genetic ‘cause’ or at least a significant contribution to the criminal behavior—irrespective of the surrounding circumstances.²⁸ If all of our actions are caused, however, it raises the question of how anyone can be held morally or legally responsible for wrongdoing since we are never free to act otherwise.²⁹ On this view, subjective experiences of choice and volition are seen as the psychological byproducts of neurogenetic causation.

By contrast, the law takes a non-determinist approach which presumes that individual actions are the end result of an individual’s volitional decisions and choices—not merely the mechanistically determined outcomes of genes, brain circuitry, or anything else.³⁰ This is why criminal punishment hinges on whether or not a defendant had the requisite *mens rea* (‘guilty mind’ including consideration of premeditation and intent), and not on whether the crime was causally predetermined by the laws of nature or a certain GxE.³¹

Threshold to Consider Neurogenetic Mitigation

The starting point for considering neurogenetic mitigation in criminal proceedings, then, is that neurogenetic vulnerabilities to violence alone do not completely excuse responsibility.³² I suggest a threshold to establish when, from a normative perspective, a defendant should be able to make a claim for mitigated culpability and reduced sentencing on the basis of his³³ neurogenetics. The test of whether neurogenetic information can mitigate culpability for violent behavior should be whether: (i) a defendant knew or should have known that he posed a risk of violently harming others; and (ii) whether a defendant took meaningful steps to safeguard others from harm due to violence—whether or not the defendant knew that his proclivity for violence had a scientifically valid neurogenetic association.

²⁵ Ilan Dar-Nimrod & Steven J. Heine, *Genetic Essentialism: On the Deceptive Determinism of DNA*, 137 PSYCHOL. BULL. 800, 803 (2011).

²⁶ Barbara B. Hagerty, *Can Your Genes Make You Murder?* NAT’L PUB. RADIO, July 1, 2010, <http://www.npr.org/templates/story/story.php?storyId=128043329>.

²⁷ M. Callahan, *Are Some People Just Born Evil?* NEW YORK POST, May 5, 2013, at 26.

²⁸ This argument as presented presumes a cause-and-effect relationship between genes and behavior—a view not supported by current scientific evidence; see Farahany & Coleman, *supra* note 21, at 116.

²⁹ SAUL SMILANSKY, FREE WILL AND ILLUSION 4 (2000).

³⁰ Stephen J. Morse, *The Non-Problem of Free Will in Forensic Psychiatry and Psychology*, 25 BEHAV. SCI. & L. 203, 209 (2007) [hereinafter ‘Free Will’]; Stephen J. Morse, *Determinism and the Death Of Folk Psychology: Two Challenges to Responsibility From Neuroscience*, 9 MINN. J.L. SCI. & TECH. 1, 17 (2008).

³¹ See Mairi Levitt & Neil Manson, *My Genes Made Me Do It? The Implications of Behavioural Genetics for Responsibility and Blame*, 12 HEALTH CARE ANALYSIS 33, 38–39 (2007).

³² Morse, *Free Will*, *supra* note 30, at 203, 209; Farahany & Coleman, *supra* note 21, at 116.

³³ For clarity of writing, this Note uses the male gender pronoun.

In applying this test of ‘meaningfully managed risk’, consideration should initially focus on whether the available evidence demonstrates that the defendant knew or should have known that he posed a risk of violence to other people beyond what is normal or expected. Assessment of the defendant’s recognition of his propensity of violence to others can include previous violent incidents or patterns of violent behavior, steps specifically taken to avoid violent conduct or its impact upon others, and/or warnings or observations by others that the individual is particularly prone to violence.

If the defendant reasonably knew or should have known about his proclivity toward violence (whatever the basis of that proclivity), then the next question is whether or not the defendant has ‘meaningfully managed’ the potential consequences of violence through steps to prevent harmful conduct and/or address risk factors that bring out this proclivity. ‘Meaningful’ steps might include efforts to seek behavioral health evaluation and treatment, demonstrated efforts to avoid or manage situations likely to elicit violent responses, abstaining from alcohol and other substances broadly associated with higher risk of violence or associated with violence for that individual, and/or engagement with social groups (eg self-help groups, peers, faith communities) whose values and expectations shield one against violent conduct.

If the defendant demonstrably pursued meaningful safeguards against a proclivity toward violence that he did or should have recognized, that behavior potentially allows an inference to be made about the defendant’s typical mental state³⁴—specifically, an intention to avoid, or at least to not volitionally intend, the violent criminal act that ultimately occurred and/or the harm this act inflicted upon the victim. Thus, defendants who meaningfully manage their risk to others ought to be able appeal to relevant, scientifically valid neurogenetic evidence (as discussed in Part II) to show why they are less morally culpable and perhaps less deserving of punishment. On the other hand, given what they knew or should have known, defendants who fail to manage a proclivity for criminal violence in a meaningful way should be precluded from introducing such evidence—regardless of its relevance or scientific validity.

Three Hypothetical Claims for Neurogenetic Mitigation

Imagine three defendants have a GxE³⁵ that confers a proclivity toward impulsive violence. Each is driving home from work and each bumps into another car. The other motorists become agitated and confrontational. In the course of the ensuing fights, each defendant kills the other driver by a blow to the head. They all plead guilty and punishment must be determined and imposed for each of them.

First, say Defendant A has absolutely no history of criminality, aggressive outbursts, or alcohol use, and no prior knowledge of his GxE variant. Under these facts, it would be reasonable to conclude that Defendant A had no basis for suspecting he was at higher risk for lashing out in violence.

Defendant B has a long history of fighting, often in response to minor provocations and both more frequently and severely when intoxicated with alcohol. Family members repeatedly told him that he has a ‘very bad temper’, that he is ‘a very mean drunk’, and

³⁴ Katrina L. Sifferd, *In Defense of the use of Commonsense Psychology in the Criminal Law*, 25 L. & PHILOS. 571, 586 (2006).

³⁵ This GxE is presumed to not affect behavior to the extent that the defendants would lack moral agency, could not take effective steps to prevent violence, etc.

that when he ‘goes off on people’ he tends to inflict physical injuries. Despite these cautions, he often spends his nights drinking, gets into fights in bars and on the streets, and frequently comes home belligerent and violent. He was previously warned by judges and probation officers that he needs ‘to get a grip on [his] temper and drinking’. Defendant B staunchly refuses treatment for his ‘anger management problem’, even though his refusal once resulted in a violation of probation for an assault and battery charge. He grew up in a violent home and Defendant B believes he inherited his aggressive nature from his father. Here, it is reasonable to conclude that Defendant B knew or should have known of his proclivity for violence but failed to take safeguards to protect others from himself.

Defendant C is similar to Defendant B but he responded to his family’s pleas that he ‘get help’. Defendant C engages in cognitive behavioral therapy with the goal of preventing impulsive violence, attends group anger management sessions, and stopped drinking after joining AA. He spends time with friends who do not condone violence, and joined a faith community that professes tolerance and peace. Every semester, he speaks to a middle school about how to deal with stress without resorting to violence or alcohol. Years go by without a violent incident until the automobile accident. In this case, it is reasonable to conclude C took meaningful steps prior to the automobile accident intended to safeguard others from his known proclivity toward violence.

All three defendants may argue for some mitigation of their sentence since their neurogenetic proclivities to impulsive violence reflects biology that is inherited and ‘not their fault’. However, only Defendants A and C ought to be permitted to appeal to evidence of a neurogenetic proclivity in support of their claims for mitigated culpability (assuming it is relevant and scientifically reliable, again as discussed in Part II).

Prior to the crime, Defendant A justifiably had no reason to know or even suspect that he posed an increased risk of violence and so it is unreasonable to hold him to any heightened obligation to safeguard others. Without a basis for even suspecting he has an unusually elevated risk, he is no more responsible for mitigating his (unknown and unsuspected) risk of violence than the average person. However, compared to the average person, it was significantly more likely that Defendant A’s criminal act was at least in part a reflection of his neurobiology owing to the GxE, and this deserves consideration as a mitigating condition during sentencing.³⁶

Defendant C had insight into his proclivity for violence—although not necessarily the contribution of his neurogenetics—and made meaningful efforts to avoid violence. His intent to avoid causing harm to other people is inferable from Defendant C’s steps to curtail his risk of violence.³⁷ Yet despite his best efforts, his neurogenetic proclivity to impulsive violence may have made him more vulnerable to the violent act, and therefore perhaps diminishes his moral culpability for the crime—this possibility warrants consideration at sentencing.³⁸

In contrast, Defendant B took no steps to safeguard others against his violence despite ample information from multiple sources over an extended period of time about

³⁶ See Matthew Jones, *Overcoming the Myth of Free Will in Criminal Law: The True Impact of the Genetic Revolution*, 52 DUKE L. J. 1031, 1042 (2003); Joshua Greene & Jonathan Cohen, *for the Law, Neuroscience Changes Nothing and Everything*, 359 PHIL. TRANS. R. SOC. LOND. B 1451 (2004).

³⁷ Sifferd, *supra* note 34, at 575.

³⁸ See Jones, *supra* note 36, at 1042; Greene & Cohen, *supra* note 36.

his significant risk. Without meaningfully addressing his known risk of violence, a defendant has no claim to mitigated culpability—regardless of whether those risks are to a significant degree attributable to neurogenetics. Criminal behavior is not morally pardoned solely because it has a neurogenetic cause.³⁹ If any person does or should reasonably recognize that he poses an increased risk of violence due to factors of any origin, that person has an obligation to try to prevent violently harming others. Accordingly, B should not be permitted to introduce evidence of a neurogenetic proclivity toward violence at sentencing since he has not demonstrated any desire or intent of acting contrary to his inherited proclivity.

Individualized Considerations

What constitutes a ‘meaningful’ effort to minimize risk of harm to other people is highly contextual and fact-specific. However, an assessment of the defendant’s protective behaviors should reflect the ‘probability’ and ‘effect size’ of a GxE—the higher likelihood of more intense violence more likely to result in death or other heinous crimes would demand greater vigilance and therefore more stringent steps to safeguard against violence.

Individual cases will also vary with regard to how much information about a ‘proclivity towards violence’ is required to trigger an obligation by a defendant to take meaningful steps to protect others against him. Note that lack of advance knowledge that one has a particular neurogenetic variant does not preclude such an obligation since an individual might have other experiential knowledge about one’s proclivities. This is similar to the situation of individuals who eat fast food every day—they do not need to understand the genetic etiology of how their vulnerability to heart disease compares to most people to recognize that they should do a better job of keeping their cholesterol under control. Nor do people who tend to become violent when they drink need to be specifically aware of how genetics contributes to their behavior—they can still recognize that they become violent when intoxicated, and are obligated to address that risk (whether or not that propensity has a genetic basis not shared by most people). Even if a defendant’s specific genetic vulnerability only becomes known in the context of criminal sentencing, the key to neurogenetic mitigation is whether a person took meaningful steps, based on what they knew or should have known, to address and manage a proclivity toward violence leading up to the crime.

PART II. PUTTING GENES ON THE STAND

Determining that a particular defendant ought or ought not to be permitted to make a claim for reduced sentencing based on neurogenetics is only the first prong in this analysis. Since not all evidence is relevant to a particular case or scientifically reliable, additional legal considerations must be taken into account for defendants to proceed with a neurogenetic claim for mitigated culpability.

The ‘Ecological Fallacy’

One challenge to using population-level GxE studies to inform individual assessments of criminal responsibility or moral culpability is the ‘ecological fallacy’. Ecological fallacies are errors of inference that occur when individual characteristics are inferred from

³⁹ Morse, Free Will, *supra* note 30, at 203, 209.

population-level science.⁴⁰ Data in these situations are descriptive of a heterogeneous population, and individual conclusions can be fallacious because population-level associations do not apply to all individuals within the population.⁴¹ For example, not all 'low MAO-A + maltreatment' individuals commit violent acts,⁴² and even if a defendant has both risk factors, it does not tell us anything definitive about why the individual committed the crime; it could have been related to the GxE, or perhaps not.⁴³ In other words, unless 'all' individuals with a particular GxE behave in a certain way, it is impossible to know whether defendants with any GxE committed a crime 'because' of their neurogenetics. Nonetheless, even though certainty may be impossible, given the stakes involved in punishment, when a defendant reaches the normative threshold to claim mitigated culpability (as described in Part I), it seems appropriate to admit neurogenetic evidence at sentencing if it meets the standards normally required of scientific evidence, and it is reasonably relevant to why an individual committed a crime.

Scientific Validity

Most states have adopted some version of the Federal Rules of Evidence to determine whether scientific evidence, such as a genetic test, is legally admissible to sentencing.⁴⁴ The United States Supreme Court modified the guidelines in *Daubert v. Merrell Dow Pharmaceutical*⁴⁵ and subsequent cases⁴⁶ to establish a 'gate-keeping' role for judges.⁴⁷ Judges can consider whether scientific evidence or expert testimony is admissible based on 'some general observations'⁴⁸ which may include: (i) whether the basis of the evidence or testimony can be and has been empirically tested; (ii) whether it has been peer-reviewed and published; (iii) its known or theoretical error rate; (iv) maintenance of standards controlling administration or operation of the methods relied upon; and (v) whether it has achieved widespread acceptance in the relevant scientific community.⁴⁹

The *Daubert* analysis is intended to be 'a flexible one' and not 'a definitive checklist or test'.⁵⁰ The US Supreme Court declined to rule on whether each of the *Daubert* conditions are necessary or sufficient for the admissibility of scientific evidence.⁵¹ Instead judges are explicitly granted 'broad latitude' and 'considerable leeway' in deciding whether to admit expert knowledge.⁵²

⁴⁰ David A. Freedman, *Ecological Inference and the Ecological Fallacy*, 6 INT'L ENCYCLOPEDIA SOC. & BEHAV. SCI. 4027 (1999).

⁴¹ Eg Laura A. Baker, Serena Bezdjian & Adrian Raine, *Behavioral Genetics: The Science of Antisocial Behavior*, 69 LAW & CONTEMP. PROBS. 7 (2006).

⁴² Caspi et al., *supra* note 12, at 852.

⁴³ Paul S. Appelbaum, *The Double Helix Takes the Witness Stand: Behavioral and Neuropsychiatric Genetics in Court*, 82 NEURON 946 (2014).

⁴⁴ Paula Kim, *Psychopathy, Genes, and the Criminal Justice System*, 15 COLUM. SCI. & TECH. L. REV. 375, 388 (2014).

⁴⁵ 509 U.S. 579 (1993).

⁴⁶ *General Electric Co. v. Joiner*, 522 U.S. 136 (1997); *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999).

⁴⁷ *Daubert*, 509 U.S. at 597.

⁴⁸ *Id.* at 593.

⁴⁹ *Id.* at 593, 594.

⁵⁰ *Id.*

⁵¹ *Id.* at 594.

⁵² *Kumho*, 526 U.S. at 1176.

The effect of MAO-A + childhood maltreatment is scientifically validated in a manner that may satisfy *Daubert* analysis in most cases,⁵³ but importantly, this is not true for all neurogenetic evidence. For example, a gene called ‘ABCD1’ has not previously been introduced in criminal proceedings,⁵⁴ but has been associated with antisocial behavior,⁵⁵ chronic relapsing opioid addiction,⁵⁶ and poor response to treatment for addiction.⁵⁷ ABCD1 would appear to be a strong candidate for neurogenetic mitigation, but the scientific evidence is less reliable than what is normally required for admissibility at sentencing. For instance, some evidence is based on research that tests whether millions of variants across the genome tend to be slightly more or less prevalent in an antisocial population compared to a control group.⁵⁸ This methodology reveals genetic variants with only minor effects on behavior: one study found that a certain ABCD1 variant was harbored by 16% of individuals with longstanding addiction compared to 28% of matched-controls.⁵⁹ While in this particular study the absence of the ABCD1 variant met statistical significance,⁶⁰ in practice, inferring a mitigating condition from the results of this type of genetic test would be highly prone to error.

Relevance and Capital Punishment

Beyond validity, scientific evidence is only admissible if it is relevant.⁶¹ Relevance is determined by whether the evidence ‘has any tendency to make a fact more or less probable than it would be without the evidence; and the fact is of consequence in determining the action’.⁶²

One example where genetic evidence was deemed irrelevant to criminality (but nonetheless admitted, as explained below) was in the case of Michael Tanzi. He was sentenced to death after he brutally ‘assaulted, abducted, robbed, sexually battered, and killed’ a stranger.⁶³ After conviction and sentencing, in 2014 Tanzi claimed his initial counsel had been ineffective for failing to investigate his XYY genotype as a potential mitigating factor.⁶⁴ However, the court rejected the argument partially because there was no valid evidence linking the XYY genotype to violent behavior,⁶⁵ thereby, rendering it irrelevant to the crime itself; the scientific evidence showed that the XYY

⁵³ Eg Caspi et al., *supra* note 12, at 852; Dorfman, Meyer-Lindenberg & Buckholtz, *supra* note 15, at 305; Pavlov, Chistiakov & Chekhonin, *supra* note 20; Byrd & Manuck, *supra* note 20, at 5, 9.

⁵⁴ Cases searched with LEXIS, US federal and state-court cases library, using the search term, ‘ABCD1’. Search last updated Jan. 7, 2016.

⁵⁵ Jessica E. Salvatore et al., *Genome-Wide Association Data Suggest ABCB1 and Immune-Related Gene Sets May Be Involved in Adult Antisocial Behavior*, 5 *TRANSL. PSYCHIATRY* e558 (2015).

⁵⁶ Beate Beer et al., *Association of Polymorphisms in Pharmacogenetic Candidate Genes (OPRD1, GAL, ABCB1, OPRM1) with Opioid Dependence in European Population: A Case-Control Study*, 8 *PLoS ONE* e75359 (2013).

⁵⁷ Vadim Yuferov et al., *Search for Genetic Markers and Functional Variants Involved in the Development of Opiate and Cocaine Addiction and Treatment*, 1187 *ANNALS N.Y. ACAD. SCI.* 184 (2010).

⁵⁸ Salvatore et al., *supra* note 55, at 2.

⁵⁹ Beate et al., *supra* note 56, at 4.

⁶⁰ *Id.*

⁶¹ *FED. R. EVID.* 402.

⁶² *FED. R. EVID.* 401.

⁶³ *Tanzi v. Florida Department of Corrections*, 772 F.3d 648 (11th Cir. 2014).

⁶⁴ *Id.* at 654.

⁶⁵ *Id.* at 655.

genotype can confer lower IQ, poor socialization skills, learning disabilities, as well as physical traits such as above average height, teeth size, and head circumference,⁶⁶ but not a proclivity to violence per se.⁶⁷

Nonetheless, for capital defendants in particular, the standards for admitting mitigating evidence at sentencing are purposefully lax.⁶⁸ Because a defendant's life can depend on mitigating conditions, the Supreme Court ruled that capital defendants can introduce mitigating evidence during sentencing that is relevant to 'any aspect of [the] defendant's character or record and any of the circumstances of the offense that the defendant proffers as a basis for a sentence less than death'.⁶⁹ The most common use of neurogenetics in the American courts has been as evidence of a mitigating condition,⁷⁰ and for capital offenses the 'courts accept behavioral genetics evidence in the majority of cases in which defense attorneys attempt to offer it'.⁷¹

Thus, in *Tanzi*, despite being irrelevant to the crime, because the developmental and physical abnormalities scientifically associated with the XYY genotype can plausibly inform assessments of the defendant's character, the genetic evidence was considered admissible during the postconviction sentencing proceedings.⁷² However, because the genotype has no association with violent crime and the jury already considered extensive mitigating evidence about Tanzi's character and upbringing, the new information gained from the genotype was considered inconsequential compared to the 'heinous, atrocious and cruel' nature his crime.⁷³ In 2014, Tanzi's death sentence was upheld.⁷⁴

CONCLUSION

The marine attacked by Private First Class Duran was fortunate to survive after suffering the deep lacerations to his neck. In 2014, Duran's appeal based on the failure of the defense counsel to pursue genetic testing for potential mitigating evidence was denied.⁷⁵ The Court suggested that it might be a decade before genetically testing criminal defendants prior to sentencing would become the norm.⁷⁶ Nevertheless, in the interim we can expect that capital and non-capital criminal defendants will increasingly bring their neurogenetic evidence to court.⁷⁷

This Note proposes a two-pronged test, beginning with a normative threshold and followed by a procedural hurdle, to help judges and juries establish when to accept arguments for neurogenetic mitigation. The first step is to assess whether a person

⁶⁶ *Id.* at 654.

⁶⁷ See Kirstine Stochholm et al., *Criminality in Men with Klinefelter's Syndrome and XYY Syndrome: a Cohort Study*, 2 *BMJ OPEN* e000650 (2012); Tanzi, *supra* note 63, at 654 (citing expert witness at postconviction hearing).

⁶⁸ Erica Beecher-Monas, *Circumventing Daubert in the Gene Pool*, 43 *TULSA. L. REV.* 241, 249, 260 (2007).

⁶⁹ *Kansas v. Marsh*, 548 U.S. 163, 174 (2006) (quoting *Lockett v. Ohio*, 438 U.S. 586, 604 (1978)).

⁷⁰ Farahny, *supra* note 6, at 12; Denno, *supra* note 23, at 993.

⁷¹ Denno, *supra* note 23, at 1028.

⁷² Tanzi, *supra* note 63, at 654.

⁷³ *Id.* at 644, 655.

⁷⁴ *Id.* at 656.

⁷⁵ Duran, *supra* note 1, at *1.

⁷⁶ *Id.* at *4, *5 (internal citations omitted).

⁷⁷ See Farahny, *supra* note 6.

has meaningfully managed his risk of harming others based on what they knew, or should have known, about their proclivities to violence. If a defendant meets this threshold, the second step is to use *Daubert* standards in assessing whether the specific neurogenetic proclivity claimed by the defendant is adequately supported by valid, reliable science with sufficient relevance to the case at hand as to be admitted to sentencing. This two-pronged test offers a flexible understanding of when moral culpability for purposes of sentencing is meaningfully informed by neurogenetics—and when it is not.