

**WHY NOT BOTH NATURE AND NURTURE: USING BEHAVIORAL
GENETIC MARKERS AS SENTENCING FACTORS**

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I. INTRODUCTION

[1] On the evening of July 1, 2013, Amos Wells was upset that his pregnant girlfriend, Chanice Reed, would not answer his calls.¹ He then drove to where she, her mother Annette, and ten-year-old brother Eddie, lived.² After arguing with Chanice and yelling at the top of his voice in a “bone-chilling scream,” Wells retrieved a handgun from his Chevrolet Tahoe parked in front of the house, and shot Chanice in the front yard as she screamed, “No, no, no.”³ Her mother then tried to bat the gun away before he shot her too. Further shots were heard before Wells finally drove off.⁴

[2] Following 9-1-1 calls, responding officers found three individuals at the scene.⁵ They found Annette on the ground screaming, who later succumbed to two gunshot head injuries at the hospital.⁶ Lying unresponsive in the yard, Chanice had been shot four times, once in the head, and three times in the torso.⁷ She and her unborn child did not survive; post-mortem testing revealed that Wells was the biological father.⁸ In the hallway inside the house, her younger brother was shot four times, and did not survive.⁹

¹ Wells v. State, 611 S.W.3d 396, 403 (Tex. Crim. App. 2020).

² *Id.*

³ *Id.*

⁴ *Id.*

⁵ *Id.*

⁶ Wells, 611 S.W.3d at 403.

⁷ *Id.*

⁸ *Id.*

⁹ *Id.*

[3] Later that evening on a phone call with his brother, Wells repeatedly said “he did not know why he did it.”¹⁰ After turning himself in, Wells kept blurting out things like “[p]ut me in jail; kill me.”¹¹ The police noted that a couple of times Wells appeared to go into a trance, “like he went to another planet.”¹² In a later interview, Wells said, “[t]here’s no explanation that I could give anyone, or anybody could give anyone, to try to make it seem right, or make it seem rational, to make everybody understand.”¹³ Genetic research into impulsive, extremely violent behavior can give individuals like Wells a scientific explanation.

[4] Violent behavior and crime continues to be a pressing public health and safety concern to citizens in the United States and around the world.¹⁴ Approximately five million violent victimization events occur annually in the United States with a large percentage of these crimes involving the use of lethal weapons.¹⁵ The financial burden alone produced by this violent behavior is extraordinarily high—each murder costs taxpayers more than \$17 million.¹⁶ Furthermore, methods used to reduce violent crime, such as

¹⁰ Brief for the State at 12, *Wells v. State*, 611 S.W.3d 396 (Tex. Crim. App. 2020) (No. AP-77,070), 2018 WL 6624304, at *15.

¹¹ *Id.*

¹² *Wells*, 611 S.W.3d at 411.

¹³ Robbie Gonzalez, *How Criminal Courts Are Putting Brains—Not People—on Trial*, WIRED (Dec. 2, 2017, 1:01 PM), <https://www.wired.com/story/how-criminal-courts-are-putting-brains-not-people-on-trial/> [<https://perma.cc/WKJ7-WL3M>].

¹⁴ Kevin Beaver et al., *The 2-Repeat Allele of the MAOA Gene Confers an Increased Risk for Shooting and Stabbing Behaviors*, 85 PSYCHIATRY Q. 257, 258 (Sept. 2014).

¹⁵ *Id.*

¹⁶ *Id.*

stop-and-frisk policies¹⁷ and three strike rules,¹⁸ do not always succeed. Cases like *Wells v. State* continue to occur.¹⁹

[5] Looking to the roots of violent behavior, scientists demonstrate that parts of this behavior are innate, or present at birth, while other parts are acquired after birth through environmental influences, as codified in the age-old nature vs. nurture debate.²⁰ However, the reality of that debate is more nuanced, especially with behaviors as complex as violence.²¹ With the advent of human genome sequencing and the use of supercomputers for statistical analysis, scientists are able to determine that there are heritable genetic components that lead to violent behavior.²² While the precise genetic variations that are related to extreme acts of violence remain elusive, scientists have honed in on a particular gene, monoamine oxidase A

¹⁷ See John MacDonald et al., *The Effects of Local Police Surges on Crime and Arrests in New York City*, 11 PLOS ONE 1, 11 (2016) (stating that the stop and frisk policy in New York resulted in “excess stops that had little crime suppression benefits”).

¹⁸ See Franklin E. Zimring & Sam Kamin, *Facts, Fallacies, and California’s Three Strikes*, 40 DUQ. L. REV. 605, 606 (2002) (finding that California’s three strikes laws only reduced crime by six tenths of one percent).

¹⁹ *Wells v. State*, 611 S.W.3d 396, 401-04 (Tex. Crim. App. 2020).

²⁰ Mairi Levitt, *Perceptions of Nature, Nurture and Behaviour*, 9 LIFE SCIS., SOC’Y & POL’Y 1, 2 (2013).

²¹ See, e.g., Kent W. Nilsson et al., *Gene-Environment Interaction of Monoamine Oxidase A in Relation to Antisocial Behaviour: Current and Future Directions*, 125 J. NEURAL TRANSMISSION 1601, 1601 (2018).

²² See, e.g., Adrian Raine, *From Genes to Brain to Antisocial Behavior*, 17 CURRENT DIRECTIONS PSYCH. SCI. 323, 323 (2008) (stating that reviews of over 100 studies provide clear evidence that about 50% of the variance in antisocial behavior is attributable to genetic influences); Nathan J. Kolla & Marco Bortolato, *The Role of Monoamine Oxidase A in the Neurobiology of Aggressive, Antisocial, and Violent behavior: A Tale of Mice and Men*, 194 PROGRESS NEUROBIOLOGY 1, 3 (2020) (stating that ample research shows that aggression has a robust genetic underpinning and that aggressive antisocial behaviors are highly heritable).

(MAOA), known as the “warrior” gene, as the most reliable candidate.²³ Like its use in *Wells*, this genetic marker has appeared in numerous criminal cases throughout the world—most often in the sentencing phase.²⁴

[6] Sentencing reform throughout the modern era has been focused on increasing fairness, from the creation of the Sentencing Commission to reforms of the federal Fair Sentencing Act.²⁵ When looking at sentencing factors, many of the factors cover the “nurture” side of the equation, such as having “been the victim of domestic violence.”²⁶ Additionally, courts frequently rely on the presentencing report, where the offender is assessed on a variety of “nurture” factors, such as criminal history, community reputation, childhood, employment record, adult achievements, substance abuse problems and efforts to break such habits, and rehabilitative progress.²⁷ While courts increasingly confront the genetic components of violent behaviors in the criminal justice system, results are haphazard. Courts must deliberately include *both* nature and nurture components that contribute to an individual’s propensity for particular behaviors to determine appropriate sentences.

[7] This Article proceeds in three Parts. Part II starts with an overview of genetic markers and the legal and scientific thresholds required to qualify a behavioral genetic marker as valid. These standards are then applied to the MAOA genetic marker. Part III delves into how the MAOA genetic marker

²³ Kolla & Bortolato, *supra* note 22, at 3.

²⁴ Sally McSwiggan et al., *The Forensic Use of Behavioral Genetics in Criminal Proceedings: Case of the MAOA-L Genotype*, 50 INT’L J.L. PSYCHIATRY 17, 20–23 (2017) (discussing nine cases in the United States that all relied upon the low expression MAOA genetic variant).

²⁵ Steven Breyer, *The Original U.S. Sentencing Guidelines and Suggestions for a Fairer Future*, 46 HOFSTRA L. REV. 799, 800 (2018); see NATHAN JAMES, CONG. RSCH. SERV., THE FIRST STEP ACT OF 2018: AN OVERVIEW (2019).

²⁶ 730 ILL. COMP. STAT. 5/5-5-3.1 (2022).

²⁷ Daniel J. Freed, *Federal Sentencing in the Wake of Guidelines: Unacceptable Limits on the Discretion of Sentencers*, 101 YALE L.J. 1681, 1722 (1992).

should be used as a sentencing factor. Usage in future dangerousness determinations and diminished mental capacity mitigations are both examined, though ultimately, mitigation is a better fit. Part IV proceeds with some practicalities of using genetic markers in sentencing, by listing additional topics of consideration that will need to be addressed.

II. THE SCIENCE OF BEHAVIORAL GENETIC MARKERS

[8] Each cell in the human body contains DNA which are the codes or instructions needed for an organism to develop, survive, and reproduce.²⁸ DNA sequences can be broken up into small sections known as genes that contain a specific “code” to make a specific protein.²⁹ Once created, these proteins then carry out particular functions in the body.³⁰ In the way that letters strung together in a certain order make a word, the correct nucleotide “code” strung together will make a protein.³¹

[9] When cells reproduce, the DNA must be copied.³² Sometimes errors occur during this process that change the DNA sequence in the new cell.³³

²⁸ *Deoxyribonucleic Acid (DNA) Fact Sheet*, NAT’L HUM. GENOME RSCH. INST. (Aug. 24, 2020), <https://www.genome.gov/about-genomics/fact-sheets/Deoxyribonucleic-Acid-Fact-Sheet> [<https://perma.cc/S5XP-FA55>] [hereinafter *DNA Fact Sheet*].

²⁹ *Id.*

³⁰ See also H. Kuivaniemi & G. Tromp, *Type III collagen (COL3A1): Gene and Protein Structure, Tissue Distribution, and Associated Diseases*, 707 GENE 151, 151 (2019) (explaining that, as an example, one type of collagen, a well-known structural protein in the body, is encoded by the COL3A1 gene).

³¹ *Id.* at 154 (noting that, as an example, the first fifteen nucleotides of the COL3A1 gene DNA sequence are ATGATGAGCTTTGTG); see also *DNA Fact Sheet*, *supra* note 28 (noting that these nucleotides are chemical building blocks of four varieties: adenine (A), thymine (T), guanine (G) and cytosine (C)).

³² See *id.*

³³ See Chee Seng Ku et al., *The Discovery of Human Genetic Variations and Their Use as Disease Markers: Past, Present and Future*, 55 J. HUM. GENETICS 403, 404 (2010).

Like in a game of telephone, these errors may pass on to offspring, giving rise to genetic variation within a population.³⁴ Depending on the size of these errors, the encoded protein can be significantly altered.³⁵ Sometimes the error consists of a single nucleotide (SNP) that has been added, deleted, or replaced.³⁶ Other errors can include longer additions or deletions of multiple nucleotides (indels) or even a grouping of nucleotides accidentally repeated (tandem repeats).³⁷ There are two types of tandem repeats: short tandem repeats (STRs) usually are tandem repeats in which the sequence length is eight nucleotides or less; longer tandem repeats are labeled as variable number tandem repeats (VNTRs).³⁸ Each of these errors, depending on the gene or genes in which they occur, can lead to a variety of outcomes such as physical disorders or profound behavior changes.³⁹

[10] With the advent of DNA sequencing and the mapping of the human genome, these genetic variations can now be easily manipulated and pinpointed to a known physical location on a chromosome, known as a genetic marker.⁴⁰ Identifying gene location enables further study and

³⁴ *Id.* at 411.

³⁵ *Id.* at 409 (explaining that genetic variation can affect protein structure).

³⁶ *Id.*

³⁷ *Id.*

³⁸ Ku et al., *supra* note 33, at 411.

³⁹ See, e.g., Andres Bendesky & Cornelia I. Bargmann, *Genetic Contributions to Behavioural Diversity at the Gene–Environment Interface*, 12 NATURE REVIEWS: GENETICS 804, 804 (2011) (explaining that, rarely, single gene mutations can cause changes in behavior such as sleep disorders and overeating).

⁴⁰ *Genetic Marker*, NAT'L HUM. GENOME RSCH. INST., <https://www.genome.gov/genetics-glossary/Genetic-Marker> [<https://perma.cc/GD6Z-PBUG>].

manipulation of specific genes.⁴¹ Genetic markers can help link a particular trait, or even a disease, with the responsible gene.⁴²

A. Scientific Threshold for a Behavioral Genetic Marker

[11] While early humans did not understand the mechanisms of inheritance or the concept of DNA, they intuitively understood that genetic inheritance shapes behavior.⁴³ By controlling animal mating, humans succeeded in domesticating cattle, horses, and dogs; selective breeding was a key insight in human civilization, even if the underlying science was not yet understood.⁴⁴ Following a series of scientific breakthroughs, it is evident that both genes and the environment together influence behavior.⁴⁵ Genes, via their influence on development and anatomy, create a framework upon which the environment acts to shape the behavior of an individual.⁴⁶ Additionally, genes create the scaffold for learning, memory, and cognition, which further allows individuals to acquire and store information about their environment for use in shaping future behavior.⁴⁷

⁴¹ See BETSY FOXMAN, *MOLECULAR TOOLS AND INFECTIOUS DISEASE* 100 (2010) (providing examples in table 7.1 of study potential enabled by identifying gene location).

⁴² See NAT'L HUM. GENOME RSCH. INST., *supra* note 40.

⁴³ Michael D. Breed & Leticia Sanchez, *Both Environment and Genetic Makeup Influence Behavior*, *NATURE EDUC. KNOWLEDGE* (2010), <https://www.nature.com/scitable/knowledge/library/both-environment-and-genetic-makeup-influence-behavior-13907840/> [<https://perma.cc/76FS-9CQW>].

⁴⁴ *Id.*

⁴⁵ See *generally id.* (explaining the various ways in which genes and the environment have influenced behavior).

⁴⁶ See *id.*

⁴⁷ *Id.*

[12] Research on the cause of behavior is a challenging field. Most complex behaviors, including mental disorders, are likely caused by the joint effects of genetic and environmental factors.⁴⁸ A gene–environment interaction ($G \times E$) refers to genetic variant differences in susceptibility to an environmental exposure and can be characterized through different types of scientific studies, which include candidate gene studies, meta-analyses, longitudinal studies, and genome-wide association studies.⁴⁹ Each of these studies varies in its experimental methodology, and each presents opportunities and challenges with its findings.

[13] Candidate gene studies are a common approach to studying these $G \times E$ phenomena. These studies require a hypothesis describing the biological mechanisms of a “candidate” gene’s impacts on a particular behavior ($cG \times E$).⁵⁰ Researchers also record environmental variables along with genetic variants of the candidate gene.⁵¹ Through multivariate statistics, these studies determine whether $G \times E$ interactions contribute to the particular behaviors.⁵² Because the candidate gene, environmental factors, and impacted behavior are all based on pre-study conjectures, the results of these studies are sometimes limited. Multiple $cG \times E$ studies are

⁴⁸ See, e.g., J. Kim-Cohen et al., MAOA, *Maltreatment, and Gene–Environment Interaction Predicting Children's Mental Health: New Evidence and a Meta-Analysis*, 11 MOLECULAR PSYCHIATRY 903, 906 (2006) (concerning the relationship between a specific gene in youth boys and their exposure to similar social environments).

⁴⁹ *Id.* at 904 (defining gene-environment correlations and providing examples of a meta-analysis and a longitudinal study); Laramie E. Duncan & Matthew C. Keller, *A Critical Review of the First 10 years of Candidate Gene-by-Environment Interaction Research in Psychiatry*, 168 AM. J. PSYCHIATRY 1041, 1041–42 (2011) (discussing candidate gene studies and genome-wide association studies).

⁵⁰ Duncan & Keller, *supra* note 49.

⁵¹ See *id.* at 1042.

⁵² *Id.*; Kim-Cohen et al., *supra* note 48, at 904 (noting different scientific findings can also be attributed to methodology artifacts, such as measurement error and measurement range restriction).

likely to show a highly variable pattern of findings.⁵³ Sometimes the $G \times E$ phenomena is small; a large number of variables producing noisy data can make detection difficult.⁵⁴ Additionally, the limited availability of human participants with particular environmental factors degrades the robustness of the statistical power of findings.⁵⁵ Therefore, replication of the findings from candidate gene studies is particularly important to determine an overall estimate of the statistical significance of any given $G \times E$ interaction.⁵⁶

[14] Another way to study behavioral genetic markers is meta-analysis, where the purpose is “to provide formal statistical methods to synthesize findings across studies of the same treatment or phenomena.”⁵⁷ Meta-analyses uses statistical conversion methods to compare the findings of multiple studies by converting the multiple studies’ results into odds, ratios, or correlations.⁵⁸ The researcher then appropriately weighs the studies in order to obtain overall statistical values.⁵⁹ This type of study is most commonly used in the assessment of clinical trials from multiple researchers and requires a large body of research to compare across multiple studies.⁶⁰

⁵³ See Duncan & Keller, *supra* note 49, at 1044-45.

⁵⁴ *Id.* at 1047-48.

⁵⁵ *Id.* at 1044.

⁵⁶ *Id.* at 1047-48.; see also John K. Hewitt, *Editorial Policy on Candidate Gene Association and Candidate Gene-by-Environment Interaction Studies of Complex Traits*, 42 BEHAV. GENETICS 1, 1-2 (2011) (noting the change in policy for Behavioral Genetics to only publish $cG \times E$ studies that are “rigorously conducted, adequately powered,” and pass a direct replication analysis).

⁵⁷ Alan Taylor & Julia Kim-Cohen, *Meta-Analysis of Gene-Environment Interactions in Developmental Psychopathology*, 19 DEV. & PSYCHOPATHY 1029, 1030 (2007).

⁵⁸ *Id.*

⁵⁹ *Id.*

⁶⁰ See *id.*; Kim-Cohen et al., *supra* note 48, at 904.

[15] Longitudinal studies can be used to characterize behavioral genetic markers. These studies use continuous or repeated measures to track individuals over extended time periods.⁶¹ Longitudinal studies are helpful to evaluate “the relationship between risk factors and the development of disease,” as well as the outcomes over different time lengths.⁶² Standard challenges with this type of study include the attrition of subjects over time, the introduction of new, extraneous variables previously unaccounted for, and the variabilities arising from individual differences.⁶³

[16] Genome-wide association studies (GWAS) are another type of study that can determine if specific gene variants among people influence their genetic susceptibility to particular traits or diseases.⁶⁴ In GWAS studies, researchers find a large number of human subjects with the particular trait in question, match them to individuals without that trait, and then search across the DNA sequences of both groups to locate statistically significant variations.⁶⁵ One of the benefits of this type of study is that researchers do not create hypotheses prior to the study.⁶⁶ However, where a genetic variant that contributes to a trait only occurs in a small percentage of the population, such as less than ten percent, GWAS studies lack predictive power.⁶⁷ Additionally, genetic markers located on the X chromosome are commonly

⁶¹ Edward Joseph Caruana et al., *Longitudinal Studies*, 7 J. THORACIC DISEASE E537, E537 (2015).

⁶² *Id.*

⁶³ *Id.* at E538.

⁶⁴ Psychiatric GWAS Consortium Coordinating Committee, *Genomewide Association Studies: History, Rationale, and Prospects for Psychiatric Disorders*, 166 AM. J. PSYCHIATRY 540, 540 (2009).

⁶⁵ See *Genome-Wide Association Studies (GWAS)*, NAT’L HUM. GENOME RSCH. INST. (Aug. 11, 2022), <https://www.genome.gov/genetics-glossary/Genome-Wide-Association-Studies> [<https://perma.cc/LW7W-K5ME>].

⁶⁶ See *id.*

⁶⁷ See Psychiatric GWAS Consortium Coordinating Committee, *supra* note 64, at 550.

excluded from standard GWAS methods, making certain $G \times E$ interactions difficult to characterize using these studies.⁶⁸

[17] To evaluate whether a behavioral genetic marker meets scientific muster, there is no exact number of studies or type of study that is required. Each methodology has advantages and disadvantages, and each type of study has outcomes which must be broadly compared. To pass a scientific threshold, statistically significant behavioral genetic markers require replication across multiple types of studies.

B. Legal Threshold for a Behavioral Genetic Marker

[18] Behavioral genetic markers have been introduced in criminal court cases as proof for a variety of behaviors, including alcoholism, mental illness, sexual sadism, or predisposition to violence.⁶⁹ Typically, behavioral genetics evidence can be used in two ways: first, during the guilt-or-innocence trial phase, which involves a factual determination about whether a defendant committed the crime, and second, during the penalty or sentencing phase, after the defendant has been found guilty, where additional information is considered to determine the sentence.⁷⁰ In the sentencing phase, behavioral genetic markers are often used as mitigating factors or as evidence of future dangerousness to extend the defendant's sentence.⁷¹

⁶⁸ Anastasia L. Wise et al., *eXclusion: Toward Integrating the X Chromosome in Genome-Wide Association Analyses*, 92 AM. J. HUM. GENETICS 643, 643 (2013).

⁶⁹ Deborah W. Denno, *Courts' Increasing Consideration of Behavioral Genetics Evidence in Criminal Cases: Results of a Longitudinal Study*, 2011 MICH. ST. L. REV. 967, 1003–05 (2011).

⁷⁰ *Id.* at 976.

⁷¹ Hannah L. Bedard, *The Potential for Bioprediction in Criminal Law*, 18 COLUM. SCI. & TECH. L. REV. 268, 275 (2017).

[19] For the trial phase, the legal hurdle to overcome for any genetic marker is admissibility through expert testimony.⁷² Following *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, trial judges must ensure that the principles underlying the expert's testimony are scientifically valid and relevant to the facts at issue.⁷³ To determine scientific validity, judges consider the following factors: (1) testability, (2) peer review and publication, (3) the existence of methodological standards (including known or potential error rate), and (4) general acceptance.⁷⁴ Once the scientific principles pass the *Daubert* test, an expert would then provide testimony to the trier-of-fact on the behavioral genetic marker and how it applies to the case.⁷⁵

[20] However, the *Daubert* standard only applies during the guilt phase—the standards for evidence at sentencing are murkier.⁷⁶ Addressing future dangerousness testimony in *Barefoot v. Estelle*, the Supreme Court determined that reliability was the cornerstone for admissibility at sentencing.⁷⁷ The court permitted psychiatrists to testify in a capital sentencing hearing about the defendant's future behavior, that he “would probably commit further acts of violence and represent a continuing threat to society,” even though such predictions were shown to be wrong two out

⁷² *See id.* at 298.

⁷³ *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 597 (1993); *see also* *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997) (reaffirming that trial judges must review testimony for scientific validity and “fit”); *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 158 (1999) (extending the scope of *Daubert* to technical evidence).

⁷⁴ *Bedard*, *supra* note 71, at 299.

⁷⁵ *See id.* at 309 (describing how an expert would analyze a genetic market).

⁷⁶ *See Bedard*, *supra* note 71, at 275 (explaining some courts allowed mitigating evidence that “may otherwise be inadmissible”).

⁷⁷ *Barefoot v. Estelle*, 463 U.S. 880, 901 (1983) (holding the adversary process could be “trusted to sort out the reliable from the unreliable evidence”).

of three times.⁷⁸ Furthermore, in *Kansas v. Marsh*, the Supreme Court reaffirmed a very open standard for mitigating evidence in capital cases, which allows defendants to present evidence 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.”⁷⁹ Generally, most jurisdictions lack guidelines for judges on what types of scientific evidence should be admitted in sentencing hearings past the standards in *Barefoot* and *Marsh*.⁸⁰

C. Monoamine Oxidase A (MAOA) As A Genetic Marker

[21] After examining the scientific and legal thresholds for behavioral markers, these standards must be applied to the behavioral genetic marker at hand: monoamine oxidase A (MAOA). The MAOA gene encodes the production of the MAOA enzyme which metabolizes (breaks down) certain monoamine neurotransmitters, such as dopamine and serotonin.⁸¹ Without enzymes to metabolize the neurotransmitters, neural pathways get repeatedly activated, causing severe dysfunction.⁸²

[22] The MAOA gene is located on the X chromosome and has a 30-nucleotide variable number of tandem repeats (VNTRs).⁸³ Different numbers of these repeated regions in a person's DNA correlate to different

⁷⁸ *Id.* at 884, 904 (permitting psychiatrists to testify about defendant's future behavior); *Id.* at 920 (Blackburn, J., dissenting) (stating such predictions were shown to be wrong two out of three times).

⁷⁹ *Kansas v. Marsh*, 548 U.S. 163, 174 (2006) (quoting *Lockett v. Ohio*, 438 U.S. 586, 604 (1978)).

⁸⁰ Bedard, *supra* note 71, at 275.

⁸¹ Beaver et al., *supra* note 14.

⁸² Heiko Brennenstuhl et al., *Inherited Disorders of Neurotransmitters: Classification and Practical Approaches for Diagnosis and Treatment*, 50 NEURO-PEDIATRICS 2, 2 (2019).

⁸³ *Id.*

activity levels for the MAOA enzyme in the brain.⁸⁴ These genetic variations can be divided into two groups: one that corresponds to low MAOA activity or low neurotransmitter metabolism, and a second that corresponds to high MAOA activity or normal neurotransmitter metabolism.⁸⁵ As MAOA is located on the X chromosome, males have only one copy of the MAOA gene whereas females have two.⁸⁶ The impact of two different MAOA genetic variants in women is unclear; historically, many investigators have selected only males for ease of scientific study.⁸⁷

i. Scientific Threshold of MAOA

[23] The first scientific study of MAOA involvement in antisocial and aggressive behavior occurred in 1993 when scientists characterized Brunner syndrome. In a large Dutch family, every affected male demonstrated disruptive, violent outbursts that manifested in a variety of ways including attempted murder, rape, and arson.⁸⁸ When attempting to diagnose these individuals, urine testing showed a marked disturbance of monoamine metabolism, indicating that the MAOA enzyme was not working correctly.⁸⁹ Additional testing revealed that these individuals had a

⁸⁴ *Id.*

⁸⁵ Maurizio Manca et al., *The Regulation of Monoamine Oxidase A Gene Expression by Distinct Variable Number Tandem Repeats*, 64 J. MOLECULAR NEUROSCIENCE 459, 460 (2018) (explaining how two, three, and five repeats are generally defined as low expression variants (MAOA-L), while the three and a half and four repeat VNTRs have been considered high expression variants (MAOA-H)); *see also* Nilsson et al., *supra* note 21, at 1602.

⁸⁶ Nilsson et al., *supra* note 21, at 1602.

⁸⁷ *Id.*

⁸⁸ *See* H. G. Brunner et al., *Abnormal Behavior Associated with a Point Mutation in the Structural Gene for Monoamine Oxidase A*, 262 SCIENCE 578, 578 (1993); H. G. Brunner et al., *X-Linked Borderline Mental Retardation with Prominent Behavioral Disturbance: Phenotype, Genetic Localization, and Evidence for Disturbed Monoamine Metabolism*, 52 AM. J. HUM. GENETICS 1032, 1032 (1993) [hereinafter *Brunner Human Genetics*].

⁸⁹ *Brunner Human Genetics*, *supra* note 88, at 1036.

complete and selective deficiency in the MAOA enzyme due to a single nucleotide mutation in the MAOA gene.⁹⁰

[24] In 2002, Caspi *et al.* published a landmark G x E study examining the role of the MAOA gene in the development of antisocial behaviors.⁹¹ Motivated by the earlier Brunner syndrome evidence,⁹² they found that associations between childhood maltreatment and antisocial behavior were modified differently by genetic variants of MAOA: those having the low-activity variant were more responsive to the effects of maltreatment than the high-activity group.⁹³ These findings attracted media and scientific interest,⁹⁴ and others attempted to replicate the findings of this study. Like other G × E studies, results were mixed. Most studies confirmed the original

⁹⁰ *Id.* at 1036–37.

⁹¹ A. Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 *SCI.* 851, 851 (2002).

⁹² See generally J. Samochowiec et al., *Association of a Regulatory Polymorphism in the Promoter Region of the Monoamine Oxidase a Gene with Antisocial Alcoholism*, 86 *PSYCHIATRY RES.* 67, 69–70 (1999) (The frequency of the 3R MAOA-L was significantly increased in 59 antisocial alcoholics compared to 185 control subjects (51 vs. 35%; P = 0.031), suggesting that genetic variant confers increased susceptibility to antisocial behavior rather than alcohol dependence per se in alcohol-dependent males.).

⁹³ A. Caspi et al., *supra* note 91, at 853.

⁹⁴ See, e.g., Tim Radford, *Scientists Identify Gene that may Trigger Violence in Abused Child.*, *GUARDIAN* (Aug. 2, 2002), <https://www.theguardian.com/uk/2002/aug/02/childprotection.medicalscience> [<https://perma.cc/C5B5-4FFS>].

findings,⁹⁵ while other studies found no interaction,⁹⁶ or in some cases a reversal in activity among female subjects.⁹⁷ Following these cG × E studies, several meta-analyses found evidence for a consistent G × E effect involving MAOA and child maltreatment.⁹⁸ Even though the MAOA gene is on the X chromosome, limiting the number of available GWAS studies,⁹⁹ at least one study confirmed that the low-expression MAOA variants are associated with extremely violent behavior.¹⁰⁰ A separate longitudinal study

⁹⁵ See, e.g., Beaver et al., *supra* note 14, at 257 (finding African American males with 2R MAOA-L are significantly more likely to engage in shooting and stabbing behaviors); but see Dean A. Stetler et al., *Association of Low-activity MAOA Allelic Variants with Violent Crime in Incarcerated Offenders*, 58 J. PSYCHIATRIC RES. 69, 69 (2014) (finding a robust statistical association between low-activity MAOA-L and crime).

⁹⁶ See, e.g., B. Haberstick et al., *MAOA Genotype, Childhood Maltreatment, and Their Interaction in the Etiology of Adult Antisocial Behaviors*, 75 BIOLOGICAL PSYCHIATRY 25, 25, (2014) (finding no effect of the MAOA genotype on self-reported antisocial behavior for 3356 white men (aged 24-34)).

⁹⁷ See, e.g., P. Hollerbach et al., *Main and Interaction Effects of Childhood Trauma and the MAOA uVNTR Polymorphism on Psychopathy*, 95 PSYCHONEUROENDOCRINOLOGY 106, 109 (2018) (finding in a larger study on 4278 Finnish individuals, MAOA-L homozygous female carriers exhibited slightly higher levels of psychopathy than their MAOA-H counterparts).

⁹⁸ See Kim-Cohen et al., *supra* note 48, at 903 (finding the association between maltreatment and mental health problems is significantly stronger in the group of males with low vs high MAOA activity in analyzing previous studies and in a new 975 subject study); Amy L. Byrd & Stephen B Manuck, *MAOA, Childhood Maltreatment and Antisocial Behavior: Meta-analysis of a Gene-environment Interaction*, 75 BIOLOGICAL PSYCHIATRY 9, 15 (2014) (finding an interaction of MAOA variation and childhood maltreatment predicting antisocial outcomes more strongly in persons of low, compared to high, activity MAOA).

⁹⁹ Z. Liu et al., *MAOA Variants and Genetic Susceptibility to Major Psychiatric Disorders*, 53 MOLECULAR NEUROBIOLOGY 4319, 4319 (2016).

¹⁰⁰ J. Tiihonen et al., *Genetic Background of Extreme Violent Behavior*, 20 MOLECULAR PSYCHIATRY 786, 786 (2015) (defining extremely violent behavior as committing at least 10 homicides, attempted homicides, or batteries, and stating that no substantial signal was observed for MAOA among non-violent offenders, indicating that findings were specific for violent offending.)

of 398 men observed from age 16 until age 30 was able to replicate these results, showing that individuals exposed to childhood abuse with the low-expression MAOA variants were significantly more likely to report later offending, conduct problems, and hostility, even when controlling for a range of potentially confounding factors.¹⁰¹ With multiple types of scientific studies showing the same results, MAOA shows promise as a genetic marker that predicts violent behavior.

ii. Legal Threshold of MAOA

[25] Following the original study publication, attorneys immediately began using MAOA in a courtroom. In 1994, Stephen Mobley was convicted of murder, armed robbery, aggravated assault, and possession of a firearm during the commission of a crime.¹⁰² Following the guilty verdict, Mobley's counsel sought traditional mitigation evidence, but found no physical abuse, sexual abuse, neglect, or childhood poverty in Mobley's childhood.¹⁰³ After reading the Brunner study, Mobley's counsel investigated the use of this genetic link to violence as a possible mitigation defense, filing a "Motion for Continuance and Motion to Provide Funds for Expert Witness Assistance to Conduct Preliminary Analysis for MAOA Deficiency and other Genetic Analysis as Information Becomes Available[,]" supporting the motion with research, affidavits from doctors, and testimony from the family historian.¹⁰⁴ The trial court denied the motion, finding that the theory behind the request did not pass scientific muster at the time (a mere year after the original Brunner study), and sentenced the defendant to death.¹⁰⁵ The Supreme Court of Georgia upheld

¹⁰¹ See David M. Fergusson et al., *MAOA, Abuse Exposure and Antisocial Behaviour: 30-year Longitudinal Study*, 198 BRITISH J. PSYCHIATRY 457, 457 (2011).

¹⁰² *Mobley v. State*, 455 S.E.2d 61, 61 (Ga. 1995).

¹⁰³ *Turpin v. Mobley*, 502 S.E.2d 458, 463 (Ga. 1998).

¹⁰⁴ *Id.* at 465.

¹⁰⁵ *Mobley*, 455 S.E.2d at 65–66.

the ruling, finding that Mobley was not entitled to funding for expert witnesses to conduct MAOA testing.¹⁰⁶

[26] Over the next fifteen years, courts grew much more comfortable with allowing MAOA genetic marker testimony, based on the large body of available research. During a court-appointed joint custody visit with his children, the defendant in *State v. Waldroup* killed his ex-wife's best friend and held his ex-wife and children against their will while he attacked and viciously beat them.¹⁰⁷ The defense claimed that because the defendant had a low-expression variant of the MAOA gene, was severely abused as a child, and had recently endured stressful life experiences, he was unable to stop himself from committing violent crimes.¹⁰⁸ The lead prosecutor, Cynthia Lecroy-Schemel, noted that "[t]here were numerous things he did around the crime scene that were conscious choices . . . One of them was [that] he told his children to 'come tell your mama goodbye,' because he was going to kill her. And he had the gun, and he had the machete."¹⁰⁹ Even with victim testimony and the sheer violence of the crime, the jury convicted Waldroup of voluntary manslaughter instead of murder and sentenced him for second-degree attempted murder instead of first-degree attempted murder.¹¹⁰ One juror observed that "[e]vidently it's just something that doesn't tick right . . . Some people without this would react totally different

¹⁰⁶ *Id.* at 66 ("[T]he theory behind the request for funds will not have reached a scientific stage of verifiable certainty in the near future and that Mobley could not show that such a stage will ever be reached.") (citing *Harper v. State*, 292 S.E.2d 389, 395 (Ga. 1982)).

¹⁰⁷ *State v. Waldroup*, No. E2010-01906-CCA-R3-CD, 2011 WL 5051677, at *1–2 (Tenn. Crim. App. Oct. 20, 2011).

¹⁰⁸ Barbara Bradley Hagerty, *Can Your Genes Make You Murder?*, NPR (July 1, 2010), <https://www.npr.org/templates/story/story.php?storyId=128043329> [<https://perma.cc/A5LN-M4V4>].

¹⁰⁹ *Id.*

¹¹⁰ *Id.*

than he would . . . A diagnosis is a diagnosis, it's there . . . A bad gene is a bad gene.”¹¹¹

[27] Courts most recently examined MAOA genetic marker testimony in *State v. Yopez*.¹¹² The defendant, Anthony Blas Yopez, along with “his girlfriend, Jeannie Sandoval (Sandoval), lived with George Ortiz (Ortiz), the boyfriend of Sandoval’s adoptive mother.”¹¹³ On October 29, 2012, the defendant “killed Ortiz during an argument, after which Yopez and Sandoval set fire to Ortiz’s body.”¹¹⁴ “Ortiz’s autopsy concluded that his cause of death was homicidal violence and thermal injuries;” New Mexico “charged Yopez with (1) first-degree murder, (2) conspiracy to commit first-degree murder, (3) tampering with evidence, and (4) unlawful taking of a motor vehicle.”¹¹⁵ Yopez sought to introduce evidence that he had a low-activity MAOA gene variant.¹¹⁶ The trial court denied his motion, stating that the proffered testimony was “insufficiently reliable or relevant on the issue of whether Yopez formed the specific intent to kill Ortiz.”¹¹⁷

[28] After a lengthy scientific examination, the Supreme Court of New Mexico held that the district court did not abuse its discretion by excluding the proposed expert testimony.¹¹⁸ While the court agreed with the conclusion that the scientific findings of low-activity MAOA genotypes moderating the effects of childhood maltreatment to increase the likelihood

¹¹¹ *Id.*

¹¹² *State v. Yopez*, 483 P.3d 576, 578 (N.M. 2021).

¹¹³ *Id.*

¹¹⁴ *Id.*

¹¹⁵ *Id.*

¹¹⁶ *Id.* at 581.

¹¹⁷ *Yopez*, 483 P.3d at 582.

¹¹⁸ *Id.* at 589.

of antisocial and aggressive behavior in males satisfied the *Daubert* reliability factors, it drew a distinction between the science and the case at hand.¹¹⁹ The court noted that even though Yepez had a low-activity MAOA genetic variant, evidence of mere genetic susceptibility to a given mental condition was not relevant to the issue of deliberate intent.¹²⁰

[29] These cases illustrate attempts to introduce MAOA evidence at different phases of criminal trials, with varying levels of success. Directly after the Brunner study, in *Mobley*, the MAOA genetic marker had not undergone enough scientific study to even pass the lower legal threshold to be admitted as evidence in sentencing.¹²¹ Fifteen years later, the MAOA genetic marker was admitted at the trial phase in *Waldroup*, passing the *Daubert* standard.¹²² More recently in *Wells* and *Yepez*, the MAOA genetic marker again passes the sentencing standard of reliability as well as the *Daubert* standard.¹²³ Critically, when the MAOA genetic marker passed the *Daubert* threshold at trial, it was found not relevant to intent.¹²⁴ While the MAOA genetic marker now passes the legal threshold for both trial and sentencing phases, applying it in the sentencing phase ensures that the evidence will be relevant to the issue at hand.

¹¹⁹ *Id.* at 587.

¹²⁰ *Id.* at 589.

¹²¹ *Turpin v. Mobley*, 502 S.E.2d 458, 466 (Ga. 1998).

¹²² *Hagerty*, *supra* note 108; *State v. Waldroup*, No. E2010-01906-CCA-R3-CD, 2011 WL 5051677, at *7 (Tenn. Crim. App. Oct. 20, 2011).

¹²³ *See Wells v. State*, 611 S.W.3d 396, 423, 430 (Tex. Crim. App. 2020); *Yepez*, 483 P.3d at 587.

¹²⁴ *Yepez*, 483 P.3d at 587.

III. USING BEHAVIORAL GENETIC MARKERS FOR VIOLENCE IN SENTENCING

[30] The historic usage of behavioral genetic markers at sentencing sheds light on where such markers should be used in future cases. In a survey of thirty-three criminal cases from 2007 to 2011 in which parties used behavioral genetics evidence, all but one of the cases began as a capital case where the defendant was initially sentenced to death by a judge or jury.¹²⁵ Attorneys employed two basic rationales for presenting this evidence: (1) to support a claim of ineffective assistance of counsel, and (2) to provide proof and diagnosis of a defendant's mitigating condition.¹²⁶ The court in all of the cases used behavioral genetics evidence for mitigation; in fact, only the courts in three cases utilized behavioral genetics evidence for future dangerousness, and all were prior to 2007.¹²⁷

[31] Going further, a widely publicized study actually put the question to active judges to examine the use of behavioral genetics evidence at sentencing.¹²⁸ A set of facts about a hypothetical defendant was presented to 181 state trial judges.¹²⁹ In these facts, the defendant was found guilty of aggravated battery against a restaurant manager who suffered brain damage

¹²⁵ Denno, *supra* note 69, at 993.

¹²⁶ *Id.* at 994–95.

¹²⁷ *Id.* at 995–96.

¹²⁸ See, e.g., Benedict Carey, *Study of Judges Finds Evidence From Brain Scans Led to Lighter Sentences*, N.Y. TIMES, (Aug. 16, 2012) <https://www.nytimes.com/2012/08/17/science/brain-evidence-sways-sentencing-in-study-of-judges.html> [<https://perma.cc/39L9-MCPC>]; Alix Spiegel, *Would Judge Give Psychopath With Genetic Defect Lighter Sentence?*, NPR, (Aug. 17, 2012, 2:58 AM EST), <https://www.npr.org/sections/health-shots/2012/08/17/158944525/would-judge-give-psychopath-with-genetic-defect-lighter-sentence> [<https://perma.cc/XBN2-VLMR>].

¹²⁹ Lisa Aspinwall et al., *The Double-edged Sword: Does Biomechanism Increase or Decrease Judges' Sentencing of Psychopaths?*, 337 SCI. 846, 846 (2012), <https://www.jstor.org/stable/pdf/23268380> [<https://perma.cc/4GXE-3RGQ>].

as a result of an attack.¹³⁰ From these facts, along with a psychopath diagnosis from a psychiatrist, the researchers asked the judges to give a sentence.¹³¹ The judges were randomly assigned to groups based on two criteria: (1) whether the expert evaluation was presented by the prosecution or by the defense, and (2) whether additional testimony was provided about the genetic explanation for psychopathy development.¹³² While judges presented with this information gave an average sentence of nine years for aggravated battery, judges without the additional evidence sentenced the defendant to an average of 13.93 years; the authors concluded that psychopathy was an aggravating factor based on increased future dangerousness.¹³³ Separately, the content analysis of the judges' reasoning indicated that the genetic evidence increased the proportion of judges listing mitigating factors, from 29.7% to 47.8%.¹³⁴ While a scientific study could never fully replicate a court case, and the methodology suffered a few flaws, this study makes clear that understanding and applying behavioral genetics can make a difference in sentencing.¹³⁵ But how to apply behavioral genetic markers remains at issue—using the MAOA genetic marker as a test case, different applications of behavioral genetic marker in sentencing can be examined.

¹³⁰ *Id.*

¹³¹ *Id.* at 846.

¹³² *Id.*

¹³³ *Id.* at 846–47.

¹³⁴ Aspinwall et al., *supra* note 129, at 846.

¹³⁵ Deborah W. Denno, *What Real-World Criminal Cases Tell Us About Genetics Evidence*, 64 HASTINGS L.J. 1591, 1596–1604 (2013) (pointing out numerous flaws to include: (1) psychopathy is not listed in the Diagnostic and Statistical Manual of Mental Disorders: Fifth Edition (“DSM-V”) and is not fully recognized or diagnostically accepted in the medical community, (2) the hypothetical case is not based on a capital crime, and (3) the study defines aggravating and mitigating factors contrary to their legal definitions).

A. The Case Against Future Dangerousness

[32] When behavioral genetic markers are used as an aggravating factor, they are exclusively used as a predictor of future dangerousness.¹³⁶ Broadly, aggravating factors can be statutory or non-statutory, and must furnish “clear and objective standards’ that provide ‘specific and detailed guidance.’”¹³⁷ Even with this requirement, as shown in *Barefoot*, the Supreme Court continues to uphold increasingly vague aggravating factors, such as the much-criticized variations of the “heinous, atrocious, or cruel” aggravating factor and the “future danger” aggravating factor.¹³⁸

[33] Even though mental health and legal professionals agree on the unreliability of current methods to predict future dangerousness, courts and legislatures continue to use these predictions in sentencing.¹³⁹ These methods can take the form of a tool such as the Violence Risk Assessment Guide (VRAG), which uses factors such as the defendant’s relation to the victim or marital status to calculate a probability value of future criminal behavior; alternately, the evaluation might take the form of an assessment by a clinical psychiatrist.¹⁴⁰ Studies of all prediction tools, from

¹³⁶ See Deborah W. Denno, *Behavioral Genetics Evidence in Criminal Cases: 1994-2007*, in *THE IMPACT OF BEHAVIORAL SCIENCE ON CRIMINAL LAW* 317, 346–349 (Nita A. Farahany ed., 2009).

¹³⁷ *Lewis v. Jeffers*, 497 U.S. 764, 774 (1990) (quoting *Godfrey v. Georgia*, 446 U.S. 420, 428 (1980)).

¹³⁸ Jeffrey L. Kirchmeier, *Aggravating and Mitigating Factors: The Paradox of Today's Arbitrary and Mandatory Capital Punishment Scheme*, 6 WM. & MARY BILL RTS. J. 345, 363 (1998).

¹³⁹ Bedard, *supra* note 71, at 281.

¹⁴⁰ Erica Beecher-Monas & Edgar Garcia-Rill, *Genetic Predictions of Future Dangerousness: Is There a Blueprint for Violence?*, L. & CONTEMP. PROBS. 301, 318–20 (2006), https://www.researchgate.net/publication/254588966_Genetic_Predictions_of_Future_Dangerousness_Is_There_a_Blueprint_For_Violence [<https://perma.cc/983A-KWQT>].

psychological clinical assessments to actuarial instruments, report high false positive rates.¹⁴¹ The prediction tools currently employed are not sufficiently accurate to be used for deprivations of life or liberty.¹⁴² For this reason, some states, including California and Florida, prohibit prosecutors from introducing expert testimony on the issue of future dangerousness.¹⁴³ Some legal experts believe that if bio-based evidence, such as behavioral genetic markers coupled with environmental factors, can make these predictions more accurate, they should be implemented.¹⁴⁴ Unfortunately, knowing an offender's MAOA variant status is not a better predictor of future criminal activities. Even though the MAOA genetic marker coupled with a history of maltreatment indicates the likelihood of violent behavior, it does not create a causal link to criminal violence.¹⁴⁵ Therefore, the MAOA genetic marker should not be used for this purpose.

[34] Furthermore, using behavioral genetic markers like MAOA for predictions of future dangerousness takes society closer to genetic determinism.¹⁴⁶ Scientists typically separate genetic determinism into three categories: (1) strong genetic determinism, where the gene (G) almost always leads to the development of trait (T) (G increases the probability of T and the probability of T, given G, is 95% or greater); (2) moderate genetic determinism, where more often than not G leads to the development of T (G increases the probability of T and the probability of T, given G, is greater than 50%); and (3) weak genetic determinism, where G sometimes leads to

¹⁴¹ Bedard, *supra* note 71, at 281–82.

¹⁴² *Id.*

¹⁴³ Erica Beecher-Monas & Edgar Garcia-Rill, *Danger at the Edge of Chaos: Predicting Violent Behavior in a Post-Daubert World*, 24 *CARDOZO L. REV.* 1845, 1849 (2003).

¹⁴⁴ Bedard, *supra* note 71, at 282.

¹⁴⁵ *Id.* at 286–87.

¹⁴⁶ Beecher-Monas & Garcia-Rill, *supra* note 143, at 305.

the development of T (G increase the probability of T, but the probability of T is still less than 50%).¹⁴⁷

[35] Stronger genetic determinism should be rejected in this concept's application to the MAOA genetic marker. Even in the most extreme cases of Brunner syndrome with no expression of MAOA, moderate genetic determinism would be the most apt—all the inflicted individuals presented violent and aggressive behaviors, but these behaviors manifested in different ways, some of which were not criminal.¹⁴⁸ On the other hand, defendants with mere low-expression MAOA variants would likely fall under weak genetic determinism.

[36] The rejection of strong genetic determinism, however, does not require the rejection of moderate or weak genetic determinism. Genes alone may not determine behavior, but that does not imply that genetic impairments of volition lack any bearing on whether a person can control violent impulses.¹⁴⁹ Genes in conjunction with the environment may influence behavior by producing physiological conditions that make controlling violent behavior abnormally difficult.¹⁵⁰ In rejecting the theory of strong genetic determinism, courts should not use the MAOA genetic marker in the aggravating sentencing factor of predicting future violence.

¹⁴⁷ David B. Resnik & Daniel B. Vorhaus, *Genetic Modification and Genetic Determinism*, PHIL., ETHICS, & HUMS. MED. (2006), <https://doi.org/10.1186/1747-5341-1-9> [<https://perma.cc/AG2Y-6G9R>].

¹⁴⁸ *Brunner Human Genetics*, *supra* note 88, at 1032.

¹⁴⁹ Amanda R. Evansburg, "*But Your Honor, It's in His Genes*"- *The Case for Genetic Impairments as Grounds for a Downward Departure Under the Federal Sentencing Guidelines*, 38 AM. CRIM. L. REV. 1565, 1578 (2001).

¹⁵⁰ *See id.* at 1574, 1579.

B. The Case for Diminished Mental Capacity

[37] Unlike aggravating factors, mitigating factors are often subjective, based in empathy for the defendant.¹⁵¹ For example, a trial judge noted that one of the non-statutory mitigating factors that worked in the defendant's favor was "that [the defendant's] family loved him."¹⁵² Statutory and non-statutory mitigating factors can range from circumstances at the scene of the crime to the defendant's childhood. Essentially, as illustrated in an Arizona statute, any factor can be used that "is relevant to the defendant's character or background or to the nature or circumstances of the crime and that the court finds to be mitigating."¹⁵³

[38] Federal and state mitigation factors often cover the nurture side of the nature-nurture debate. For example, in Illinois, if the defendant "is the parent of a child or infant whose well-being will be negatively affected by the parent's absence" or "is or had been the victim of domestic violence," these circumstances act to mitigate the sentence.¹⁵⁴ The nature side of the nature-nurture equation—genetic markers—should also be included in sentencing.

[39] One possible use for behavioral genetic markers such as MAOA is in the Diminished Mental Capacity factor in the U.S. Sentencing Guidelines. Under § 5K2.13, "[a] downward departure may be warranted if (1) the defendant committed the offense while suffering from a significantly reduced mental capacity; and (2) the significantly reduced mental capacity contributed substantially to the commission of the offense."¹⁵⁵ The Federal

¹⁵¹ Denno, *supra* note 69, at 979.

¹⁵² Schriro v. Landrigan, 550 U.S. 465, 470 (2007).

¹⁵³ See Ariz. Rev. Stat. Ann. § 13-701(D)(27) (2018).

¹⁵⁴ 730 Ill. Comp. Stat. 5/5-5-3.1 (2022); see also Tenn. Code Ann. § 40-35-113 (West 2010) (listing suffering from a mental or physical condition that significantly reduced the defendant's culpability for the offense as a mitigating factor).

¹⁵⁵ U.S. SENT'G GUIDELINES MANUAL § 5K2.13 (U.S. SENT'G COMM'N 2004).

Sentencing Commission further notes that for “the purposes of the policy statement, ‘[s]ignificantly reduced mental capacity’ means the defendant, although convicted, has a significantly impaired ability to (A) understand the wrongfulness of the behavior comprising the offense or to exercise the power of reason; or (B) control behavior that the defendant knows is wrongful.”¹⁵⁶ Here, the Commission permits judges to impose a reduced sentence where the offender was significantly impaired in his ability to resist criminal behavior, subtly acknowledging that not all criminal behaviors are subject to the same volition of control.¹⁵⁷

[40] Downward departures for offenders with lower thresholds for violence, caused by genetics and exacerbated by environmental factors, could be included in the Diminished Mental Capacity category. This category would more accurately reflect the relationship between genes and criminal violence; while genetic impairments predispose certain individuals to violent behavior, making it substantially harder to refrain from acts of violence, genetic impairments are not deterministic forces that foreclose all possibility of adherence to the law.¹⁵⁸

[41] Additionally, the Sentencing Commission has interpreted “reduced mental capacity” to include volitional impairments.¹⁵⁹ In the 1994 Amendment, the Commission “defines ‘significantly reduced mental capacity’ in accordance with the decision in *United States v. McBroom*.¹⁶⁰ The Supreme Court concluded that “significantly reduced mental capacity” included both cognitive impairments, which is the inability to understand the wrongfulness of the conduct or to exercise the power of reason; and volitional impairments, which is the inability to control behavior that the

¹⁵⁶ *Id.*

¹⁵⁷ Evansburg, *supra* note 149, at 1569–70.

¹⁵⁸ *Id.*

¹⁵⁹ U.S. SENT’G GUIDELINES MANUAL § 5K2.13 cmt. n.1 (U.S. SENT’G COMM’N 2004).

¹⁶⁰ *Id.*

person knows is wrongful.¹⁶¹ The application note specifically includes both types of impairments in the definition of “significantly reduced mental capacity.”¹⁶² The inclusion of volitional impairments opens the door to behavioral genetic markers such as MAOA in the diminished capacity mitigation factors.

IV. IMPLICATIONS OF BEHAVIORAL GENETIC FACTORS IN SENTENCING

[42] While sentence reduction through mitigation factors is important, eventually the goal must be to rehabilitate individuals with low-expression MAOA genetic variants. The Regional Psychiatric Centre in Canada could be a model for treatment of such individuals.¹⁶³ This program works exclusively with violent offenders and uses group and individual therapy sessions with a psychoeducational aspect to reduce recidivism rates.¹⁶⁴ In order to get individuals into these types of programs, genetic factors like MAOA must be applied at sentencing, implicating a number of issues and restrictions.

A. Preliminary Scientific & Legal Threshold Must Be Met

[43] While MAOA has repeatedly passed the scientific and legal threshold, other behavioral genetic markers must do the same prior to their use in sentencing. The case summarized in Part I of this Article is particularly instructive on this point.¹⁶⁵ The defendant in *Wells* sought to introduce seven specific gene variants that increased his propensity for

¹⁶¹ United States v. McBroom, 124 F.3d 533, 546, 548 (3d Cir. 1997).

¹⁶² U.S. SENT’G GUIDELINES MANUAL § 5K2.13 (U.S. SENT’G COMM’N 2004).

¹⁶³ Mark E. Olver et al., *Risk Reduction Treatment of High-Risk Psychopathic Offenders: The Relationship of Psychopathy and Treatment Change to Violent Recidivism*, 4 PERSONALITY DISORDERS: THEORY, RSCH., & TREATMENTS 160, 160 (2013).

¹⁶⁴ *Id.*

¹⁶⁵ See *Wells v. State*, 611 S.W.3d 396, 425 (Tex. Crim. App. 2020).

violence; the trial court only allowed evidence of the MAOA genetic marker.¹⁶⁶ Without ruling on the MAOA genetic marker, the Court of Criminal Appeals of Texas upheld the trial court's rejection of all six behavioral genetic markers, noting that the "proposed testimony did not serve the purposes for which it was offered, that is, objectively to assist the jury in assessing . . . culpability."¹⁶⁷

[44] The MAOA genetic marker provides a good example of the required scientific threshold; multiple studies, from candidate gene studies and meta-analyses, to longitudinal studies and GWAS studies, show the same replicated results.¹⁶⁸ None of the other genetic markers come close to the scope of findings behind the MAOA genetic marker.¹⁶⁹ One of the genetic markers, the 5-HTT gene variants, sometimes known as the SLC6A4 gene, have been found to contribute to depressive symptoms by increasing the reuptake of serotonin; however, its association with aggressive and violent criminal behavior is much weaker.¹⁷⁰ Without scientific studies to back up the behavioral genetic markers, these markers must be rejected at trial, even during the sentencing phase.

¹⁶⁶ *Id.* at 424–25. (attempting to show variants of 5HTTLPR, rs25531, STin2, rs4680 (COMT), rs1800955, and DRD4-2/11 increased a propensity towards violence, in addition to MAOA).

¹⁶⁷ *Id.* at 428 (stating that the expert admitted that a combination of the variants exponentially increases a person's propensity for violence was not supported by any studies whatsoever).

¹⁶⁸ *See supra* Part II(c)(i).

¹⁶⁹ *See id.*

¹⁷⁰ Tufik Y. Shayeb, *Behavioral Genetics & Criminal Culpability: Addressing the Problem of Free Will in The Context of the Modern American Justice System*, 19 UDC L. Rev. 1, 36 (2016).

B. Restrictions Of Sentencing Factors with Behavioral Genetic Markers

[45] Once the scientific threshold is met, the court must examine the sentencing factors available in relation to the details of the behavioral gene marker. As discussed previously, the diminished mental capacity mitigation factor fits best for the MAOA genetic marker. The current language of this section requires that “(1) the defendant committed the offense while suffering from a significantly reduced mental capacity; and (2) the significantly reduced mental capacity contributed substantially to the commission of the offense.”¹⁷¹ With a genetic impairment such as MAOA, the defendant would have to show by a preponderance of the evidence that (1) he actually has a genetic impairment that has been found to predispose the bearer to violent behavior, and (2) this genetic impairment significantly contributed to the commission of the violent crime for which he was convicted.¹⁷² Specifically for MAOA, the genetic disorder being contemplated is one that impairs the ability to control violent impulses.¹⁷³ Therefore, the MAOA genetic impairment departure should apply only to offenders convicted of violent crimes.

[46] The biggest hurdle for practitioners to overcome in the use of behavioral genetic markers is proving the genetic impairment in question. In *Mobley*, the Supreme Court of Georgia determined that the defendant was not entitled to funding for expert witnesses to conduct MAOA testing, rendering it impossible for the defendant to make use of this possible argument. While the science behind the MAOA genetic marker has come a long way in twenty-five years, the budgets for public defenders have not. In 2011, the DNA-based test for the low-expression MAOA variant alone cost

¹⁷¹ U.S. SENT’G GUIDELINES MANUAL § 5K2.13 (U.S. SENT’G COMM’N 2004).

¹⁷² Evansburg, *supra* note 149, at 1583.

¹⁷³ *Id.* at 1582.

\$250.¹⁷⁴ To have the evidence of a genetic marker admitted for the purpose of sentencing, an official report would likely have to be submitted to the court, supported by an expert willing to testify to its results.¹⁷⁵ Ultimately, before parties can widely test for genetic markers such MAOA, courts must address the financial accessibility of genetic testing and admissibility of the data into evidence.

V. CONCLUSION

[47] Is behavior defined by nature or nurture? Both interact together to create behavior patterns.. With significant scientific background and an ever-growing list of cases, the low-expression MAOA genetic variant along with a history of maltreatment clearly impacts violent, criminal behavior. Using the MAOA genetic marker as a functional test, the sentencing factor of diminishing mental capacity is the best application of this genetic predictor to the sentencing framework. While the MAOA genetic marker leads the way, other genetic markers will need to meet the same scientific and legal standards. Cost and other restrictions need to be addressed as well before more individuals can use these novel techniques in sentencing. In the long run, the goal will be to provide specialized rehabilitation methods to reduce violent crime and recidivism.

¹⁷⁴ Jennifer Brooks-Crozier, *The Nature and Nurture of Violence: Early Intervention Services for the Families of MAOA-Low Children as a Means to Reduce Violent Crime and the Costs of Violent Crime*, 44 CONN. L. REV. 531, 569 (2011).

¹⁷⁵ See *supra* Part III.