

When Neurogenetics Hurts: Examining the Use of Neuroscience and Genetic Evidence in Sentencing Decisions Through Implicit Bias

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Courts increasingly use neuroscience and genetic evidence (“neurogenetic evidence”) to shed light on various aspects of a defendant’s mental state and behavior. The evidence is particularly prevalent in cases involving defendants with mental illnesses and is used to determine issues of mental capacity, personal responsibility, and treatability. However, using neurogenetic evidence risks framing mental illness through a narrow explanatory model—one relying solely on biological causes. Such evidence elicits both stigma-reducing and stigma-enhancing implicit biases against mental illness, which can manifest themselves in beliefs that a person with mental illness is less blameworthy for his condition, but also more dangerous and less receptive to treatment. These implicit biases affect jurors (and potentially judges) and may influence sentencing decisions in cases involving defendants with mental illnesses, including ultimate sentencing decisions in capital cases.

While there has been vast literature on (1) the merging fields of neurogenetics and the law, (2) sentencing decisions in cases involving defendants with mental illnesses, and (3) implicit bias against mental illness, no article has connected the literature to provide an interdisciplinary account of these processes. This Note argues that the use of neurogenetic evidence in the courtroom may harm defendants with mental illnesses because the nature of the

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evidence primes negative implicit biases against mental illness. This Note then explores how this dynamic plays out during the sentencing phase in capital cases involving defendants with mental illnesses.

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INTRODUCTION

In 1977, Randall Dale Adams was convicted of murdering police officer Robert W. Wood.¹ At sentencing, the prosecution relied on expert witnesses, Doctors John Holbrook and James Grigson, who evaluated Adams and testified that he had antisocial personality disorder and the profile and characteristics of a sociopath.² They explained to the jury that they “would expect little or no change in this diagnosis” and that Adams would likely commit criminal acts of violence in the future.³ The expert witnesses contended that Adams would present a continuing threat to society and that failing to execute him would endanger police officers, who protect society from anarchy.⁴ The jury recommended the death sentence,⁵ which the Texas Court of Criminal Appeals affirmed, stating that the “testimony, when considered with the evidence of the

1. See *Ex parte Adams*, 768 S.W.2d 281, 283 (Tex. Crim. App. 1989) (en banc); *Adams v. State*, 577 S.W.2d 717, 719 (Tex. Crim. App. 1979) (en banc), *rev'd in part*, 448 U.S. 38 (1980).

2. *Adams*, 577 S.W.2d at 731.

3. *Id.*

4. Kathleen Wayland & Sean D. O'Brien, *Deconstructing Antisocial Personality Disorder and Psychopathy: A Guidelines-Based Approach to Prejudicial Psychiatric Labels*, 42 HOFSTRA L. REV. 519, 520 (2013) (citing Charles Musser, *Film Truth, Documentary, and the Law: Justice at the Margins*, 30 U.S.F. L. REV. 963, 974 (1996)).

5. *Adams*, 577 S.W.2d at 719.

crime itself, which was a particularly senseless and motiveless killing, [was] sufficient to support the jury's determination that appellant would constitute a continuing threat to society."⁶ In 1989, after twelve years in prison, Adams was exonerated when a judge learned that the eyewitness who initially identified Adams in a police line up offered fabricated and perjured testimony.⁷

As in *Adams*, parties may utilize expert witnesses during the sentencing phase of capital cases. The experts offer mitigating or aggravating evidence to sway the jury to recommend life imprisonment or the death penalty.⁸ While *Adams* concerned a somewhat lucky case (if one can consider exoneration after twelve years in prison lucky),⁹ for those who are not or cannot be exonerated, such mitigating or aggravating evidence can literally result in life-or-death consequences. The influence of mitigating or aggravating evidence is more complicated in cases involving defendants with mental illnesses, where the evidence is more likely to comprise of neuroscience and genetic (hereinafter "neurogenetic") explanations of the defendants' behaviors and actions.¹⁰

For the purposes of this Note, "neurogenetic" refers to both "neuroscience evidence," such as brain scans and neuroimaging, and "genetic evidence," such as evidence linking a particular gene to an observable behavior or trait. The two types of evidence pose similar problems for a defendant with mental illness as they both attribute behavior to biological causes (i.e., the brain and genes, respectively). Accordingly, this Note uses the term "neurogenetic" when discussing issues involving both types of evidence, and issues associated with

6. *Id.* at 731.

7. *See Ex parte Adams*, 768 S.W.2d 281, 291, 294 (Tex. Crim. App. 1989) (en banc).

8. *See Adams*, 577 S.W.2d at 731; John M. Fabian, *Death Penalty Mitigation and the Role of the Forensic Psychologist*, 27 L. & PSYCHOL. REV. 73, 80 (2003) (explaining that during the penalty phase of a capital case, the jury hears from both the prosecution and defense about how the defendant's crime, personal disposition, prior criminal history, or psychological makeup constitutes either an aggravating or a mitigating factor); Meghan Shapiro, Comment, *An Overdose of Dangerousness: How "Future Dangerousness" Catches the Least Culpable Capital Defendants and Undermines the Rationale for the Executions It Supports*, 35 AM. J. CRIM. L. 145, 146 n.2 (2008) (explaining that most states with the death penalty consider a defendant's future dangerousness to society as a statutory or non-statutory aggravating factor); *see, e.g.*, 18 U.S.C. § 3592(a)–(b) (2012); TEX. CODE CRIM. PROC. ANN. art. 37.071 § 2(a)–(b) (West 2013), available at <http://www.statutes.legis.state.tx.us/Docs/CR/pdf/CR.37.pdf>.

9. For similar cases involving defendants who were sentenced to death, in part due to perceptions of future potential dangerousness, and who were later exonerated, *see, for example, Graves v. Dretke*, 442 F.3d 334 (5th Cir. 2006) and *Guerra v. Collins*, 916 F. Supp. 620 (S.D. Tex. 1995).

10. *See generally* Jay D. Aronson, *The Law's Use of Brain Evidence*, 6 ANN. REV. L. & SOC. SCI. 93, 97 (2010) (examining how advances in neuroscience have been utilized in the legal system to help determine mental states, detect lies, judge insanity defenses, and to serve as mitigating or aggravating evidence during sentencing). Note that in *Adams*, the expert witnesses conducted a psychiatric diagnosis of Adams and did not rely on neurogenetic evidence, as defined *infra*, to support their evaluation. *See Adams*, 577 S.W.2d at 731. *Adams* illustrates the significant influence expert witnesses may exert over jurors, and this Note argues that expert testimony utilizing neurogenetic evidence in particular can magnify such influence (and the associated harms to defendants).

either “neuroscience evidence” or “genetic evidence” can generally apply to the other as well.

Any biological explanation of behavior will elicit the same type of implicit biases, or unconscious attitudes and beliefs, against a defendant with mental illness, but the magnitude of those biases can vary depending on the nature of the biological explanation offered. In other words, while a psychiatric diagnosis, for example, can elicit the same type of implicit biases against a defendant with mental illness, the biases are not likely to be as strong compared to those elicited by neurogenetic evidence because the psychiatric diagnosis does not provide an immediate link between a tangible, physical source of causation and observable behavior.¹¹

Although the intersection of neurogenetics and law is still in its infancy and judges continue to proceed cautiously in admitting such evidence in court,¹² it is imperative to examine the potential prejudicial effects of such evidence as this type of evidence becomes more pervasive. While there has been vast literature on (1) the merging fields of neurogenetics and the law,¹³ (2) sentencing decisions in cases involving defendants with mental illnesses,¹⁴ and (3) implicit bias against mental illness,¹⁵ no article has connected the literature to provide an interdisciplinary account of these processes. This Note argues that the use of neurogenetic evidence in the courtroom may produce unexpected results for defendants with mental illnesses because the nature of

11. For a discussion regarding the increased harm associated with neurogenetic evidence, as opposed to other explanations relying on biological causes, see *infra* Part III.

12. See, e.g., *United States v. Semrau*, 693 F.3d 510 (6th Cir. 2012) (excluding as inadmissible an expert’s proposed testimony regarding functional magnetic resonance imaging testing); Virginia Hughes, *Science in Court: Head Case*, 464 NATURE 340 (2010) (detailing the case of self-proclaimed psychopath, Brian Dugan, and the judge’s decision to allow brain scanning-based testimony to go forward, but to prohibit the showing of the individual scans themselves).

13. See, e.g., Deborah W. Denno, *Courts’ Increasing Consideration of Behavioral Genetics Evidence in Criminal Cases: Results of a Longitudinal Study*, 2011 MICH. ST. L. REV. 967 (examining the use of behavioral genetics evidence between 1994 and 2011); Nita A. Farahany, *Incriminating Thoughts*, 64 STAN. L. REV. 351 (2012) (explaining how neuroscience evidence demands a redefinition of the taxonomy of evidence); Nita A. Farahany & James E. Coleman, Jr., *Genetics and Responsibility: To Know the Criminal from the Crime*, 69 L. & CONTEMP. PROBS. 115 (2006) (describing how practitioners have introduced behavioral genetics evidence in recent criminal cases); Walter Glannon, *The Limitations and Potential of Neuroimaging in the Criminal Law*, 18 J. ETHICS 153 (2014) (examining the probative value and prejudicial effects of neuroimaging evidence); Owen D. Jones & Francis X. Shen, *Law and Neuroscience in the United States*, in INTERNATIONAL NEUROLAW 349, 349 (Tade Matthias Spranger ed., 2012) (“[S]ince 2000, the number of English-language law review articles including some mention of neuroscience has increased fourfold.”).

14. See, e.g., William Bernet et al., *Bad Nature, Bad Nurture, and Testimony Regarding MAOA and SLC6A4 Genotyping at Murder Trials*, 52 J. FORENSIC SCI. 1362 (2007); Michael J. Saks et al., *The Impact of Neuroimages in the Sentencing Phase of Capital Trials*, 11 J. EMPIRICAL LEGAL STUD. 105 (2014) (examining the impact of neuroimages on mock jurors in an experimental setting).

15. See *infra* Part II. Although there is substantial literature on implicit bias generally, much less has been written on implicit bias against mental illness specifically. For a synthesis of implicit bias research in law reviews, see Anthony G. Greenwald & Linda Hamilton Krieger, *Implicit Bias: Scientific Foundations*, 94 CALIF. L. REV. 945 (2006).

the evidence itself primes both stigma-reducing and stigma-enhancing implicit biases against mental illness. As stigma-enhancing implicit biases often result in longer sentences, this Note then recommends potential strategies to manage neurogenetic evidence and reduce implicit bias in the courtroom.

Part I describes historical advancements in neurogenetics and the increasing use of such evidence in the courtroom. This Part highlights potential concerns that the “seductive allure”¹⁶ of scientific evidence may unduly sway jurors when evaluating neurogenetic evidence. Part II explains the process of implicit bias and explores implicit bias against mental illness in particular. This Part then explains how certain implicit biases against mental illness are triggered through “essentialism,” a cognitive process where people categorize groups by underlying essences that imply immutable and deterministic behaviors.

Part III explores conflicting research suggesting that neurogenetic evidence may serve as both a mitigating factor and an aggravating factor in sentencing decisions, and examines these consequences through an illustration of a defendant with psychopathy. This Part demonstrates that while a defense team may intend to utilize neurogenetic evidence during sentencing as a mitigating factor for a defendant with mental illness, the prosecution may utilize the very same evidence as an aggravating factor.

Finally, although normative solutions are generally beyond the scope of this Note, Part IV provides a few recommendations on how judges may manage the use of neurogenetic evidence in the courtroom to minimize its negative consequences for defendants with mental illnesses. This Part recommends (1) requiring a special jury instruction in capital cases utilizing neurogenetic evidence to highlight the variety of contributors of mental illness beyond biological causes, and (2) employing court-appointed implicit bias experts, under Rule 706 of the Federal Rules of Evidence and its state counterparts, to assist jurors in understanding the implicit biases primed by neurogenetic evidence. This Part concludes by offering recommendations to defense attorneys as well, cautioning against the utilization of neurogenetic evidence during the sentencing phase of capital cases in light of the research discussed in this Note.

I.

THE GROWING INTERACTION BETWEEN NEUROGENETICS AND THE LAW

While scholarship addressing the intersection of psychology and the law has existed for nearly a century,¹⁷ scholarship on neurogenetics and the law emerged in the late 1990s through conferences that brought scholars from

16. See Deena Skolnick Weisberg et al., *The Seductive Allure of Neuroscience Explanations*, 20 J. COGNITIVE NEUROSCIENCE 470 (2008).

17. See, e.g., JEROME FRANK, *LAW AND THE MODERN MIND* (1930).

neuroscience and the law together in dialogue, resulting in an increase in interdisciplinary articles, journals, and books.¹⁸ In addition, funding from the Dana Foundation and the Gruter Institute for Law and Behavioral Research further catalyzed this research.¹⁹ Support for the work continued in 2007 when the Law and Neuroscience Project received \$10 million in funding from the MacArthur Foundation.²⁰ Today, neurogenetics and the law work continues to grow at an astounding rate.²¹ The mainstream media has released articles related to law and neurogenetics,²² and neuroscience evidence²³ has been utilized in Supreme Court jurisprudence.²⁴

18. Oliver R. Goodenough & Micaela Tucker, *Law and Cognitive Neuroscience*, 6 ANN. REV. L. & SOC. SCI. 61, 63–64 (2010).

19. *See id.*

20. *See id.*

21. *See* Francis X. Shen, *The Law and Neuroscience Bibliography: Navigating the Emerging Field of Neurolaw*, 38 INT'L J. LEGAL INFO. 352, 357 (2010) (“[T]he field is now in the midst of massive growth, with over 45% of its publications coming in just the past two years. The 127 publications in 2009 represents a 300% increase over the number published just five years earlier, and represents a 2,000% increase over the number published a decade before.”); *see also* Denno, *supra* note 13, at 971 (finding eighty-one criminal cases employed behavioral genetics evidence between 1994 and 2011).

22. *See, e.g.*, Scott T. Grafton et al., *Brain Scans Go Legal*, 17 SCI. AM. MIND, Dec. 2006–Jan. 2007, at 30; Jeffrey Rosen, *The Brain on the Stand*, N.Y. TIMES, Mar. 11, 2007, <http://www.nytimes.com/2007/03/11/magazine/11Neurolaw.t.html>; Barbara Bradley Hagerty, *Can Your Genes Make You Murder?*, NAT'L PUB. RADIO (July 1, 2010, 12:00 AM), <http://www.npr.org/templates/story/story.php?storyId=128043329>.

23. As stated in the Introduction, this Note focuses on “neurogenetic evidence,” which encompasses both neuroscience and genetic evidence. Neuroscience evidence includes any evidence linking the functionality or structure of the brain to behavior. This evidence is often illustrated through its pictorial counterpart, “neuroimaging evidence,” which includes electroencephalography (EEG), computed tomography (CT) scans, and functional magnetic resonance imaging (fMRI), among other techniques. These images illustrate characteristics of the brain including electrical activity or blood flow to specific areas of the brain, brain atrophy, and brain lesions. For a lay explanation of various neuroimaging techniques, see Jane Campbell Moriarty, *Flickering Admissibility: Neuroimaging Evidence in the U.S. Courts*, 26 BEHAV. SCI. & L. 29, 30–35 (2008). Genetic evidence includes evidence linking any particular gene to a behavior. For example, research suggests that low activity of the monoamine oxidase A (MAOA) gene is associated with violent antisocial behavior. *See, e.g.*, Brett Walker, *When the Facts and the Law Are Against You, Argue the Genes?: A Pragmatic Analysis of Genotyping Mitigation Defenses for Psychopathic Defendants in Death Penalty Cases*, 90 WASH. U. L. REV. 1779, 1795 (2013). “Neurogenetic evidence” does not include psychiatric diagnoses such as the expert testimony offered in the case of Randall Dale Adams. *See Adams v. State*, 577 S.W.2d 717, 731 (Tex. Crim. App. 1979) (en banc), *rev'd in part*, 448 U.S. 38 (1980). For example, one common psychiatric diagnosis of psychopathy has relied on the “Psychopathology Checklist—Revised” (PCL-R). Kimberly D. Phillips, *Empathy for Psychopaths: Using fMRI Brain Scans to Plea for Leniency in Death Penalty Cases*, 37 LAW & PSYCHOL. REV. 1, 7–8 (2013). The PCL-R is a checklist of specific personality traits and behaviors associated with psychopathy such as “glibness/superficial charm,” “grandiose sense of self-worth,” and “lack of remorse or guilt.” *Id.* Thus, a psychiatrist rates an individual on each item of the checklist and aggregates a total score, where higher scores indicate greater psychopathic tendencies. *Id.* For more information regarding the psychiatric diagnosis of psychopathy, see *id.* at 5–9.

24. *See Graham v. Florida*, 560 U.S. 48, 68 (2010) (citing brain development research in the Court’s decision on life imprisonment for minors).

Much of the interaction between neurogenetics and the law has revolved around how court proceedings utilize neurogenetic evidence.²⁵ In federal courts and in a majority of state courts, *Daubert v. Merrell Dow Pharmaceuticals* governs the admissibility of scientific evidence.²⁶ In *Daubert*, the Supreme Court provided guidelines for determining the admissibility of expert testimony under Rule 702 of the Federal Rules of Evidence.²⁷ The Court explained that when evaluating the validity of an expert opinion or testimony, judges may assess a variety of factors, including: the falsifiability of the scientific theory or evidence, its error rate, whether it has been peer reviewed and published, and whether the theory or evidence has attracted widespread acceptance within the relevant scientific community.²⁸ Despite these factors, however, the Court granted judges wide discretion in assessing expert testimony, allowing each court to make its own determination regarding the admissibility of scientific evidence in each case.²⁹

Although the law has not incorporated neurogenetic evidence in all legal contexts, courts have generally increased their use of such evidence over the last five years.³⁰ For example, plaintiffs may use neuroscience evidence to prove actual harm in tort or to show that a party lacked sufficient cognitive capacity to form a valid contract.³¹ Neuroscience evidence has also played a role in determining whether someone is telling the truth, or whether someone is competent to stand trial.³² However, debate over the reliability of such

25. See Goodenough & Tucker, *supra* note 18, at 69.

26. See *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579 (1993).

27. See FED. R. EVID. 702 (“A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if: (a) the expert’s scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue; (b) the testimony is based on sufficient facts or data; (c) the testimony is the product of reliable principles and methods; and (d) the expert has reliably applied the principles and methods to the facts of the case.”); *Daubert*, 509 U.S. at 593–95.

28. *Daubert*, 509 U.S. at 593–95.

29. *Id.* at 597.

30. See Shen, *supra* note 21, at 352–53 (highlighting that in 2010, the first *Daubert* hearing was held on the admissibility of functional magnetic resonance imaging lie detection evidence in *United States v. Semrau*, a Florida court admitted quantitative encephalography evidence in *State v. Nelson*, and the Supreme Court cited brain development research in its decision on life imprisonment for minors in *Graham v. Florida*). See generally Denno, *supra* note 13 (describing the increased utilization of behavioral genetic evidence in criminal courts in the last two decades).

31. O. Carter Snead, *Neuroimaging and the “Complexity” of Capital Punishment*, 82 N.Y.U. L. REV. 1265, 1291–92 (2007).

32. See, e.g., *United States v. Kasim*, No. 2:07 CR 56, 2008 WL 4822291, at *17 (N.D. Ind. Nov. 3, 2008) (considering medical testimony and accompanying brain images in finding that Dr. Kasim was incompetent to stand trial for Medicaid fraud); Nobuhito Abe, *The Neurobiology of Deception: Evidence from Neuroimaging and Loss-of-Function Studies*, 22 CURRENT OPINION NEUROLOGY 594 (2009) (finding that the prefrontal cortex in the brain plays a predominant role in deception); Joshua D. Greene & Joseph M. Paxton, *Patterns of Neural Activity Associated with Honest and Dishonest Moral Decisions*, 106 PROC. NAT’L ACAD. SCI. 12506 (2009) (finding that levels of activity in control-related regions of the prefrontal cortex correlated with the frequency of dishonesty in individuals).

evidence remains. Although at least one court has admitted lie detection evidence for the defense in a post-conviction proceeding,³³ several academic commentators remain skeptical of the reliability of current lie detection evidence.³⁴ Moreover, in two recent cases, the judges excluded neuroscience evidence because of the questionable scientific validity of the testing.³⁵

Courts have been particularly receptive to neurogenetic evidence in the context of capital cases, especially during the sentencing phase where such evidence can shed light on the future dangerousness of a defendant.³⁶ The increased use of neurogenetic evidence during capital sentencing hearings, results, in part, from the reduced evidentiary burdens at this stage of trial.³⁷ In addition, courts may be more willing to admit neurogenetic evidence during the sentencing phase of capital cases simply because “death is different.”³⁸

The increasing utilization of neurogenetic evidence in the courtroom presents potential problems as jurors and judges may not possess the requisite knowledge to understand such evidence and therefore may be unduly influenced by it. For instance, scholars have found that laypeople generally possess limited knowledge about genetics. One study found that despite conversational familiarity with genetic terminology, most participants lacked an understanding about basic genetic science (e.g., over half of the surveyed population did not know that genes were located in cells).³⁹ Moreover, another study observed that “public discourse on genetics is plagued by genetic fatalism in such a way that any association between genes and behavior is seen to imply predetermined, immutable behavior.”⁴⁰ In other words, public

33. See *Harrington v. State*, 659 N.W.2d 509, 516 (Iowa 2003) (noting the admission of “computer-based brain testing” to probe the existence of discrete facts stored in the defendant’s brain).

34. See, e.g., Henry T. Greely, *Premarket Approval Regulation for Lie Detection: An Idea Whose Time May Be Coming*, 5 AM. J. BIOETHICS 50 (2005) (calling for regulation of lie detection evidence).

35. See *United States v. Semrau*, 693 F.3d 510, 522–23 (6th Cir. 2012) (finding that the evidence would not satisfy the *Daubert* test necessary to meet Rules 702 and 703 of the Federal Rules of Evidence); *Wilson v. Corestaff Servs., L.P.*, 900 N.Y.S.2d 639, 642 (Sup. Ct. 2010) (noting concerns about the scientific validity of lie detection testing).

36. See Bernet et al., *supra* note 14, at 1363–65 (listing examples of capital cases utilizing testimony regarding MAOA and SLC6A4 (a serotonin transporter gene) genotyping evidence during the sentencing phase); Snead, *supra* note 31, at 1300 n.175 (listing examples of capital cases utilizing various forms of neuroscience evidence as mitigating evidence during the sentencing phase).

37. See 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, 1846 (2003) (“Evidentiary rules do not generally apply at sentencing hearings.” (citations omitted)); Snead, *supra* note 31, at 1325 (explaining that courts essentially ignore evidentiary standards for expert testimony concerning future dangerousness and exercise wide discretion in admitting such evidence during this phase).

38. Moriarty, *supra* note 23, at 49 (quoting *Ford v. Wainwright*, 477 U.S. 399, 411 (1986)).

39. See Angela D. Lanie et al., *Exploring the Public Understanding of Basic Genetic Concepts*, 13 J. GENETIC COUNSELING 305, 313 (2004).

40. Ilan Dar-Nimrod & Steven J. Heine, *Genetic Essentialism: On the Deceptive Determinism of DNA*, 137 PSYCHOL. BULL. 800, 803 (2011); see also Joseph S. Alper & Jonathan Beckwith, *Genetic Fatalism and Social Policy: The Implications of Behavior Genetics Research*, 66 YALE J. BIOLOGY & MED. 511 (1994).

discourse incorrectly frames genetic evidence as linked to inevitable character traits rather than as one factor in explaining a behavior.

A lack of understanding about neurogenetics becomes increasingly problematic when coupled with the seemingly unquestionable nature of the science and its resulting “seductive allure.” Early researchers used “seductive allure” to describe their observation that neuroscience explanations of psychological phenomena seemed to draw large public interest.⁴¹ They investigated whether even irrelevant neuroscience information could draw such interest; specifically, they investigated whether irrelevant neuroscience information could produce higher evaluations from laypeople on the soundness or logic of a particular explanation.⁴² For example, an explanation without neuroscience stated: “The researchers claim that this ‘curse’ happens because subjects have trouble switching their point of view to consider what someone else might know, mistakenly projecting their own knowledge onto others.”⁴³ The explanation with irrelevant neuroscience stated: “Brain scans indicate that this ‘curse’ happens because of the frontal lobe brain circuitry known to be involved in self-knowledge. Subjects have trouble switching their point of view to consider what someone else might know, mistakenly projecting their own knowledge onto others.”⁴⁴ In the second explanation, the neuroscience information was logically irrelevant as it did not alter the underlying logic of the explanation itself or add any additional explanatory power.

The researchers found that participants judged explanations with irrelevant neuroscience information to be more satisfying than the same explanations that did not include the superfluous neuroscience information.⁴⁵ Under this “allure,” participants not only judged otherwise logical statements as more persuasive when they included neuroscience information, but also judged originally “bad” explanations (i.e., statements involving circular reasoning) as good ones, “masking otherwise salient problems in these explanations.”⁴⁶

This “seductive allure” may affect laypeople in general. The researchers found that both non-experts and students who had taken a semester of cognitive neuroscience also responded positively to superfluous neuroscience information.⁴⁷ As another scholar explained, neuroscience evidence may possess a “‘seductive allure’ to the lay public in part because it combines the luster of real science, the ontological solidity of physical causation, and the

41. See generally Weisberg et al., *supra* note 16.

42. See *id.* at 471.

43. *Id.*

44. *Id.* (emphasis omitted).

45. *Id.* at 475.

46. *Id.* at 470.

47. *Id.* at 475.

often illusory concreteness and precision of brain-based explanation.”⁴⁸ In other words, neuroscience evidence can be “alluring” to laypeople because it reduces the level of abstraction involved in explaining a behavior by providing a physical source of causation—the brain.

By the same logic, genetic evidence can be “alluring” to laypeople because genes are the physical source of causation for behavior.⁴⁹ Whereas other explanations of mental illness such as a psychiatric diagnosis can still require an abstract mental jump linking intangible mental states to a behavior,⁵⁰ neurogenetic evidence allows laypeople to understand a behavior as a natural extension of more tangible, physical aspects such as the brain or genes.

In another study, the researchers found that presenting brain images with an explanation of a psychological phenomenon produced a similar “allure” and resulted in higher ratings of scientific merit compared to an explanation without the brain images, even when the images were irrelevant or superfluous.⁵¹ Neuroimaging evidence may be particularly persuasive because laypeople are more likely to view an image of the brain as a “visual truth” and “as factually correct.”⁵²

Despite early evidence of the “seductive allure” of neuroscience, recent research conflicts as to the strength of the effect across different legal contexts.⁵³ Accordingly, empirical researchers and legal scholars should

48. See Nick Haslam, *Genetic Essentialism, Neuroessentialism, and Stigma: Commentary on Dar-Nimrod and Heine* (2011), 137 PSYCHOL. BULL. 819, 822 (2011) (citations omitted).

49. While no study has replicated the findings by Weisberg et al., *supra* note 16, utilizing irrelevant genetic evidence, the “seductive allure” logically extends to genetic evidence.

50. See, e.g., Phillips, *supra* note 23, at 5–9 (describing how one common tool for psychiatric diagnoses of psychopathy, the PCL-R, is a checklist of specific personality traits and behaviors associated with psychopathy such as “glibness/superficial charm”).

51. David P. McCabe & Alan D. Castel, *Seeing is Believing: The Effect of Brain Images on Judgments of Scientific Reasoning*, 107 COGNITION 343 (2008); see also Adina L. Roskies et al., *Neuroimages in Court: Less Biasing than Feared*, 17 TRENDS COGNITIVE SCI. 99, 100 (2013). Roskies et al. also found this effect, explaining that the “seductive allure” is largely due to a lack of sufficient education. The bias toward neuroscience evidence may be “due to the lay intuition that neuroscience is a ‘harder’ science than the behavioral sciences.” Roskies et al., *supra*. However, “[i]f one is skeptical of the diagnosis of schizophrenia on the basis of clinical observation, there is no reason to be any less skeptical about neurological evidence for schizophrenia, because the neuroscientific relevance is predicated on the psychiatric diagnosis.” *Id.* (for example, finding differences in brain activation between two patients with schizophrenia is useful only to the extent that there are observable behavioral or psychological differences that might be associated).

52. Phillips, *supra* note 23, at 44 (quoting Laurence R. Tancredi & Jonathan D. Brodie, *The Brain and Behavior: Limitations in the Legal Use of Functional Magnetic Resonance Imaging*, 33 AM. J. L. & MED. 271, 279 (2007)).

53. See Daniel D. Langleben & Jane Campbell Moriarty, *Using Brain Imaging for Lie Detection: Where Science, Law, and Policy Collide*, PSYCHOL. PUB. POL’Y & L., at 6 (2012), *advance online publication available at* <http://papers.ssrn.com/abstract=2139408>.

More recent data have suggested that such images are not as overwhelmingly influential to a jury as originally believed. [A recent study] examined the influence of neuroscience expert testimony and neuroimaging testimony on mock juries determining guilt in a criminal case in which the defendant claimed not to have requisite intent to harm the victim. Schweitzer et al. concluded that “the overwhelming consistent finding has been a lack of any impact of

continue to explore these dangers associated with neurogenetic evidence as it becomes increasingly used in the courtroom. It is “increasingly important that judges and jurors be sufficiently educated to understand the ways in which neuro[genetic] evidence can be relevant to legal questions and to recognize when they are not.”⁵⁴ As this Note explains in Parts II and III, this “seductive allure” of neurogenetic evidence can create further problems because the evidence primes negative implicit biases against mental illness and the perceived persuasiveness of the underlying evidence enhances the strength of the biases.

II.

IMPLICIT BIAS AGAINST MENTAL ILLNESS

Over the past few decades, researchers have explored the powerful influence of unconscious thought and have found that implicit (as opposed to explicit) preferences and beliefs drive much of behavior.⁵⁵ Neurogenetic evidence primes particular implicit beliefs toward mental illness, which can manifest themselves in evaluative judgments and decisions toward a person with mental illness. This Part begins by explaining implicit bias generally, then discusses implicit biases toward mental illness in particular, and concludes by explaining how neurogenetic evidence primes particular implicit biases through the process of essentialism.

To understand implicit bias, it is helpful to first learn several key terms. Preferences, also termed “attitudes,” are “evaluative disposition[s]—that is, the tendency to like or dislike, or to act favorably or unfavorably toward, someone or something.”⁵⁶ Beliefs, also termed “social stereotype[s],” are “mental association[s] between a social group or category and a trait.”⁵⁷ “Bias[es]” are “displacement[s] of people’s responses along a continuum of possible judgments.”⁵⁸ These biases can be conceptualized as the real-world application of a person’s preferences and beliefs, manifesting themselves in actual

neuroimages on the decisions of our mock jurors.” In the [study], Schweitzer et al. did find that a neurological explanation for defendant’s mental state—with or without brain images—was more influential to the jurors than a clinical psychological explanation. Although this study is compelling, there is more to be done in the area, a point well explained by Schweitzer et al.

Id. (citing N.J. Schweitzer et al., *Neuroimages as Evidence in a Mens Rea Defense: No Impact*, 17 PSYCHOL. PUB. POL’Y & L. 357, 382 (2011)). *But see* Saks et al., *supra* note 14, at 123–24 (finding that while neuroimages may have no impact in the context of a *mens rea* defense and various forms of insanity defenses, neuroimages reduced judgments of responsibility and death sentences for defendants diagnosed as psychopathic, and increased judgments of responsibility for defendants diagnosed as schizophrenic).

54. *See* Roskies et al., *supra* note 51.

55. *See generally* Greenwald & Krieger, *supra* note 15.

56. *Id.* at 948 (defining “attitude”).

57. *Id.* at 949 (defining “social stereotype”).

58. *Id.* at 950 (defining “bias”).

judgments, decisions, and actions.⁵⁹ While biases can operate on an explicit, conscious level, subliminal stimuli can trigger implicit biases, which occur “more rapidly than can be mediated by conscious activity.”⁶⁰

Implicit biases, which can be favorable or unfavorable to a group, may produce particularly dangerous consequences because they are automatic by definition, and therefore operate outside one’s conscious control.⁶¹ In addition, people may possess implicit preferences, beliefs, and biases because the brain categorizes information to more efficiently process external information and stimuli.⁶² In other words, the immense amount of information in the external world necessitates stereotyping to perceive, process, and retain information.

Several characteristics of implicit bias are particularly important in understanding how these biases influence people in everyday life. First, implicit bias is pervasive and widespread.⁶³ Second, while people may believe they do not possess any implicit biases, implicit bias operates absent any conscious intent to favor or disfavor a particular social group.⁶⁴ A meta-analysis of 126 individual studies found substantial variability in the strength of the relationship between implicit and explicit cognitions, suggesting that implicit biases do not mirror explicit biases.⁶⁵ Third, implicit bias is predictive of behavior. “[I]mplicit bias against a social category . . . predicts disparate behavior toward individuals mapped to that category,” regardless of contrary conscious intent.⁶⁶ Fourth, implicit bias is malleable. Researchers have found that implicit biases may be minimized upon exposure to counter-stereotypical out-group members.⁶⁷ For example, a negative implicit bias toward African American faces (relative to European American faces) may be minimized with sufficient prior exposure to faces such as Martin Luther King Jr. (an association of an African American face and a positive concept). These four characteristics are key in understanding the harm caused by the pervasive and unconscious

59. See Dale Larson, Comment, *Unconsciously Regarded as Disabled: Implicit Bias and the Regarded-As Prong of the Americans with Disabilities Act*, 56 UCLAL. REV. 451, 470 (2008).

60. Anthony G. Greenwald & Mahzarin R. Banaji, *Implicit Social Cognition: Attitudes, Self-Esteem, and Stereotypes*, 102 PSYCHOL. REV. 4, 5 (1995) (citations omitted).

61. See *id.* at 4–5.

62. See generally Linda Hamilton Krieger, *The Content of Our Categories: A Cognitive Bias Approach to Discrimination and Equal Employment Opportunity*, 47 STAN. L. REV. 1161 (1995).

63. See Kristin Lane et al., *Implicit Social Cognition and Law*, 3 ANN. REV. L. & SOC. SCI. 427, 433 (2007); see also Brian A. Nosek et al., *Pervasiveness and Correlates of Implicit Attitudes and Stereotypes*, 18 EUR. REV. SOC. PSYCHOL. 36, 52 (2007) (finding that approximately three in four individuals who take the race Implicit Association Test, discussed *infra* Part II.A, show some degree of race-based bias).

64. See Lane et al., *supra* note 63, at 431–32.

65. See *id.*

66. Jerry Kang, *Trojan Horses of Race*, 118 HARV. L. REV. 1489, 1514 (2005); see also Greenwald & Krieger, *supra* note 15, at 954 (arguing that implicit bias affects how we interpret social situations, perform on objectively measured tests, and interact with others).

67. See Lane et al., *supra* note 63, at 438.

influence of implicit biases primed by neurogenetic evidence,⁶⁸ as well as the potential to combat these harms due to the malleable nature of implicit bias.⁶⁹

A. *The Implicit Association Test*

Utilization of the Implicit Association Test (IAT) is largely responsible for the depth of implicit bias research in recent years. The IAT is a web-based tool developed in 1998 that can be used to test either implicit attitudes or implicit biases by measuring automatic group-valence and group-trait associations that participants cannot discern.⁷⁰ For example, the most widely used IAT assesses implicit attitudes toward African Americans relative to European Americans.⁷¹ To measure this attitudinal preference, the IAT compares automatic associations between African American or European American faces and positive or negative concepts.⁷² To achieve this, a participant first pairs two concepts, such as an African American face and the word “good” or a European American face and the word “good.”⁷³ The test then switches which group is associated with which characteristic. The IAT evaluates the relative speed at which the participant makes each pairing as a relative attitudinal preference.⁷⁴ Therefore, if a participant is quicker in associating European American faces with positive concepts than pairing African American faces with positive concepts, the participant has a closer implicit association of the former two concepts, indicating an implicit bias in favor of European Americans.⁷⁵

B. *Implicit Bias Against Mental Illness*

Although a number of studies have examined implicit bias against physical disability,⁷⁶ little research has looked at implicit biases against mental

68. See *infra* Part III.

69. See *infra* Part IV.

70. Greenwald & Krieger, *supra* note 15, at 952. “Group-valence” refers to associations between a group and a positive or negative evaluative disposition. “Group-trait associations” refer to associations between a group and a variety of both positive and negative traits. See *id.* at 950–53.

71. *Id.* at 952.

72. See Kang, *supra* note 66, at 1509–10.

73. See *id.* (describing testing with terms such as “violent,” “lazy,” “smart,” and “kind”).

74. See *id.* The IAT relies on speed as a measure because faster responses indicate pairings that are more strongly associated in the brain. *Id.* at 1508–10.

75. See generally Greenwald & Krieger, *supra* note 15.

76. See, e.g., PROJECT IMPLICIT, <http://www.projectimplicit.org> (last visited Mar. 17, 2015). Researchers at Project Implicit measured implicit bias against disability by comparing associations between images associated with people with disabilities or images associated with people without disabilities, and positive and negative words. Images associated with people with disabilities included a handicap wheelchair sign, a drawing of crutches, a drawing of a blind man with a walking cane, and a seeing-eye dog. Images associated with people without disabilities included a drawing of a man skiing and a drawing of a man running. PROJECT IMPLICIT, <https://implicit.harvard.edu/implicit/selectatest.html> (access the Disability IAT on this page).

illness in particular.⁷⁷ IAT studies on implicit bias against disability have found that “[p]reference for people without disabilities compared to people with disabilities was among the strongest implicit and explicit effects across the social group domains.”⁷⁸ Problematically, studies suggest that people may endorse even more critical evaluations and exhibit stronger implicit biases against mental illness than physical illness.⁷⁹

Research on implicit bias against mental illness has demonstrated that laypeople possess strong negative biases against mental illness. Studies show that people possess both negative explicit and implicit attitudes and beliefs about people with mental illnesses and laypeople tend to associate the concepts “bad,” “blameworthy,” and “helpless” with mental illness.⁸⁰ Moreover, an association with the label “mentally ill” can make it more difficult for a person to obtain work and housing, and to gain acceptance from peers and coworkers.⁸¹ Negative attitudes toward mental illness have even led one legal scholar to coin the term “sanism,” which he defines as an “irrational prejudice [against mental illness] of the same quality and character of other irrational prejudices that cause (and are reflected in) prevailing social attitudes of racism, sexism, homophobia, and ethnic bigotry.”⁸²

The full extent of implicit bias against mental illness may vary, however, depending on how the mental illness is explained or conceptualized. Within the

77. See Bethany A. Teachman et al., *Implicit and Explicit Stigma of Mental Illness in Diagnosed and Healthy Samples*, 25 J. SOC. & CLINICAL PSYCHOL. 75, 77 (2006) (“Although stigma of mental illness has been measured using a variety of self-report and behavioral indicators, there has been little investigation into the role that implicit, less strategic or unintentional, processes play in the expression of bias.”).

78. Nosek et al., *supra* note 63, at 54 (finding that 76 percent of the sample demonstrated an implicit preference for those without disabilities while 9 percent showed a pro-disabled preference, and the bias was consistent across genders, ethnicities, age groups, and political orientations).

79. See, e.g., Teachman et al., *supra* note 77, at 89 (finding strong evidence of negative implicit biases toward mental illness as well as relatively more negative explicit biases toward mental illness as compared to explicit biases toward physical illness); see also Stephen P. Hinshaw & Andrea Stier, *Stigma as Related to Mental Disorders*, 4 ANN. REV. CLINICAL PSYCHOL. 367, 372 (2008) (“Additional attitudinal studies over the past three decades have continued to yield evidence for major stigmatization of mental disorders. For example, for decades research has shown that when compared with a host of other stigmatized conditions, mental illness is typically the worst ranked or near the bottom . . .”).

80. See Hinshaw & Stier, *supra* note 79, at 372–73, 378 (suggesting strong explicit biases against mental illness persist as “many of the most disparaging expressions used by children and adults to castigate unliked or devalued peers involve colloquial terms related to mental illness or mental retardation (e.g., retard, psycho, nuts, deranged)”; Teachman, *supra* note 77, at 83–84 (finding that subjects rated people with mental illnesses as helpless when rating them as “helpless” versus “competent”).

81. Teachman, *supra* note 77, at 77; see also Patrick Corrigan, *How Stigma Interferes with Mental Health Care*, 59 AM. PSYCHOLOGIST 614, 616 (2004); Stewart Page, *Effects of the Mental Illness Label in 1993: Acceptance and Rejection in the Community*, 7 J. HEALTH & SOC. POL’Y 61, 64–65 (1995) (finding that property owners are extremely unlikely to make available apartments ready for viewing when they believe that prospective renters have a history of mental illness).

82. Michael L. Perlin, “Baby, Look Inside Your Mirror”: *The Legal Profession’s Willful and Sanist Blindness to Lawyers with Mental Disabilities*, 69 U. PITT. L. REV. 589, 590 (2008).

last few decades, medical professionals and researchers have utilized “biological”⁸³ rather than “psychosocial”⁸⁴ explanations for mental illness, rebranding mental illness as a medical disease in an attempt to reduce negative biases against people with mental illnesses.⁸⁵ For example, many healthcare professionals and patient advocacy campaigns, such as the National Alliance for the Mentally Ill and the National Alliance on Mental Illness, have sought to encourage thinking of mental illnesses as medical diseases rather than as psychological ones that can be altered by environmental factors.⁸⁶ Moreover, in one study, researchers measured endorsement of various causes of schizophrenia in Germany in 1990 and 2001 and found that endorsement of biological causes such as “brain disease” and “heredity” increased from 51 percent to 70 percent, and from 41 percent to 60 percent, respectively. On the other hand, endorsement for psychosocial causes, such as “broken home,” decreased from 55 percent to 39 percent.⁸⁷

Proponents of this conceptual shift toward explaining mental illness in biological terms initially intended to reduce stigma against mental illness and to dispel perceptions that people with mental illnesses are responsible for their own conditions.⁸⁸ One team of researchers explained that “[b]y framing mental . . . disorders as diseases, these programs anticipate that stigma and discrimination should decrease; that is, if mental disorders are attributed to factors outside of an individual’s personal control, blameworthiness should decrease, reducing society’s negative view toward such disorders.”⁸⁹ However, this biological model of mental illness has produced varying outcomes, subduing some implicit biases but exacerbating others. While some research

83. For this Note, “biological” is defined as any explanation or conceptualization of mental illness grounded in biological explanations; that is, mental illness as a disease similar to physical diseases or mental illness as a product of genes or brain abnormalities. While “biological” encompasses neurogenetic evidence, the term is broader because it attributes behavior to *any* biological cause. For example, a psychiatric diagnosis that relies on biological factors would constitute “biological” evidence, but would not constitute “neurogenetic” evidence unless it also utilizes either neuroscience or genetic evidence (such as brain scans or links to particular genes).

84. For this Note, “psychosocial” is defined as any explanation or conceptualization of mental illness grounded in environmental explanations; that is, mental illness as a product of early childhood experiences and mental illness as a product of social interactions and environmental stressors, among others.

85. See Bernice A. Pescosolido et al., “A Disease Like Any Other”? A Decade of Change in Public Reactions to Schizophrenia, Depression, and Alcohol Dependence, 167 *AM. J. PSYCHIATRY* 1321, 1321 (2010). *But see* Hinshaw & Stier, *supra* note 79, at 370 (“Whereas such ascriptions to biological etiologies might be thought to reduce stigmatization because these causes are noncontrollable, the actual evidence is far from clear . . .”).

86. See Danny C.K. Lam & Paul M. Salkovskis, *An Experimental Investigation of the Impact of Biological and Psychological Causal Explanations on Anxious and Depressed Patients’ Perception of a Person with Panic Disorder*, 45 *BEHAV. RES. & THERAPY* 405, 405–06 (2007).

87. See J. Read et al., *Prejudice and Schizophrenia: A Review of the ‘Mental Illness is an Illness Like Any Other’ Approach*, 114 *ACTA PSYCHIATRICA SCANDINAVICA* 303, 307 (2006).

88. See Lam & Salkovskis, *supra* note 86.

89. Andrew J. Howell et al., *Psychological Essentialism and Its Association with Stigmatization*, 50 *PERSONALITY & INDIVIDUAL DIFFERENCES* 95, 99 (2011) (citations omitted).

suggests that biological conceptualizations of mental illness can reduce blame and the extent to which the public holds people with mental illnesses responsible for their illnesses, other studies have found links between biological explanations and “prognostic pessimism”—the belief that mental illnesses are relatively incurable and unlikely to remit.⁹⁰

C. “Essentialism” as an Explanation for Why the Biological Model of Mental Illness Elicits Conflicting Implicit Biases

A cognitive process known as “essentialism”—how people perceive “natural” categories, such as minerals and living organisms, as having an “underlying, nontrivial, fundamental nature that makes them what they are”—triggers the implicit biases associated with the biological model of mental illness.⁹¹ This underlying nature or essence causes natural categories to be what they are by generating the shared characteristics of the members of the category.⁹² For example, a kiwi’s underlying essence causes it to have a brown, fuzzy exterior, a green interior with black seeds, and a sweet taste. Moreover, while there may be changes in the visible characteristics of members of a category (e.g., a kiwi that has been dyed from its original brown exterior to a pink exterior), these do not necessarily imply changes in the kiwi’s essence.

Studies show that, in addition to categories found in nature, people essentialize socially constructed categories, such as race and gender, by perceiving members as sharing an essential group of traits.⁹³ One researcher highlighted the pervasive nature of this process:

[E]ssentialism is a general human tendency, and evidence for it has been found among children and adults in an array of diverse cultures including impoverished neighborhoods in Brazil, pastoral herdsmen in Mongolia, Vevo children in Madagascar, Menominee community members in Wisconsin, and middle-class children and adults in the

90. See Haslam, *supra* note 48, at 820–21; see, e.g., Benjamin Goldstein & Francine Rosselli, *Etiological Paradigms of Depression: The Relationship Between Perceived Causes, Empowerment, Treatment Preferences, and Stigma*, 12 J. MENTAL HEALTH 551, 558 (2003) (finding that participants who endorsed biological factors for depression were less likely to blame people with depression for their condition); Jo C. Phelan, *Geneticization of Deviant Behavior and Consequences for Stigma: The Case of Mental Illness*, 46 J. HEALTH & SOC. BEHAV. 307, 316–17 (2005) (finding that attributing schizophrenia or major depression to genetic factors increased the perceived seriousness and persistence of the illnesses); Jo C. Phelan et al., *Genes and Stigma: The Connection Between Perceived Genetic Etiology and Attitudes and Beliefs About Mental Illness*, 6 PSYCHIATRIC REHABILITATION SKILLS 159, 178–79 (2002) (finding that attributing schizophrenia to genetic or hereditary causes reduced perceptions that the person with schizophrenia was responsible for his illness, but also decreased perceptions that the illness could improve with treatment).

91. See Dar-Nimrod & Heine, *supra* note 40, at 801.

92. See *id.*

93. *Id.* See generally Myron Rothbart & Marjorie Taylor, *Category Labels and Social Reality: Do We View Social Categories as Natural Kinds?*, in LANGUAGE, INTERACTION AND SOCIAL COGNITION 11–36 (Gün R. Semin & Klaus Fiedler eds., 1992).

United States.⁹⁴

Because the underlying essence of a category is unobservable and abstract, people often use an “essence placeholder” to reduce the abstract nature of an essence and draw causal inferences between a group’s essence and an observable characteristic.⁹⁵ Research has studied the degree to which genes may serve as an essence placeholder resulting in “genetic essentialism,” where people believe members of a particular group share a distinct genetic makeup and infer behaviors and traits on the basis of assumed shared genes.⁹⁶ “[G]enetic essentialism reduces the self to a molecular entity, equating human beings, in all their social, historical, and moral complexity, with their genes.”⁹⁷

Research suggests that genetic essentialism creates the view that characteristics and behaviors of a group are immutable and determined, rather than influenced by the environment and within an individual’s control.⁹⁸ Such essentialism can lead people to perceive groups that share a genetic foundation as homogenous, where all members of the group possess an associated characteristic or behavior. Problematically, because “[e]ssentializing social groups increases the perceived homogeneity and immutability of the members of a group . . . [s]uch essentializing is associated with increased stereotypical thinking and attitudes.”⁹⁹

94. Dar-Nimrod & Heine, *supra* note 40, at 801 (citations omitted).

95. *Id.* (citation omitted).

96. *Id.* at 801–02. Genetic essentialism is thus a type of essentialism that replaces the general, abstract essence of a group with a genetic explanation.

97. *Id.* at 801 (quoting DOROTHY NELKIN & M. SUSAN LINDEE, *THE DNA MYSTIQUE: THE GENE AS A CULTURAL ICON* (1995)).

98. *Id.* at 803.

99. *Id.* at 801. Dar-Nimrod explains that the consequences of genetic essentialism are pervasive throughout both naturally and socially constructed categories:

People come to identify with different cultures if they learn that genes underlie their race; people become more prejudiced when they learn that members of ethnic/racial out[-]groups differ in their genes; women perform worse on math tests when they hear that men possess “math genes” . . . ; criminals are viewed as less culpable if they are perceived to possess genes linked to their crime; mental illnesses are perceived as more serious if genes have been implicated; and people eat more cookies when they learn of “obesity genes.”

Id. at 809 (citations omitted). See also Nick Haslam et al., *Psychological Essentialism, Implicit Theories, and Intergroup Relations*, 9 *GROUP PROCESSES & INTERGROUP REL.* 63, 69 (2006) (citing research showing how essentialist beliefs are associated with the endorsement of prejudiced attitudes); Johannes Keller, *In Genes We Trust: The Biological Component of Psychological Essentialism and its Relationship to Mechanisms of Motivated Social Cognition*, 88 *J. PERSONALITY & SOC. PSYCHOL.* 686, 690, 694 (2005) (finding that participants who believed that “[t]he fate of each person lies in his or her genes” were more likely to possess prejudice, negative racial stereotyping, nationalism, and patriotism). *But see* Dar-Nimrod & Heine, *supra* note 40, at 802 (“[H]omosexuality may be viewed more positively if it is perceived to be the outcome of a natural, genetic predisposition rather than as a consciously made life choice.”), 806–07 (referencing studies suggesting that homosexuality may be tolerated more if sexual orientation genes are believed to exist). For more information regarding how essentialist beliefs may elicit conflicting biases against sexual orientation, see Nick Haslam & Sheri R. Levy, *Essentialist Beliefs About Homosexuality: Structure and Implications for Prejudice*, 32 *PERSONALITY & SOC. PSYCHOL. BULL.* 471, 482 (2006) (“In relation to antigay attitudes, at least, only beliefs in the discreteness, fundamentality, and informativeness of sexual orientation were associated

Research has also explored how the brain may serve as an essence placeholder resulting in “neuro-essentialism,” the concept that the brain defines the essence of a person.¹⁰⁰ Neuro-essentialism reduces observable behavior, traits, and illnesses to localized portions of the brain.¹⁰¹ For example, if a person learned that particular abnormalities in neurotransmitter function are associated with major depression, or that particular structural abnormalities in the frontal lobe of the brain are associated with psychopathy, the person could essentialize members of these groups as sharing the same deterministic neuronal characteristics.

In the context of mental illness, research shows that both neuro and genetic essentialism (hereinafter “neurogenetic essentialism”) can elicit both stigma-reducing and stigma-enhancing implicit biases. The primary stigma-reducing implicit bias is one of reduced blame, where a biological explanation of mental illness reduces implicit attributions of a person’s ability to control certain behaviors and, therefore, responsibility of the person with mental illness for his behavior.¹⁰²

However, neurogenetic essentialism produces a variety of stigma-enhancing implicit biases against mental illness as well. A biological explanation of mental illness is likely to elicit an implicit bias that the illness is immutable and incurable because it “resides deep within the individual’s biological core.”¹⁰³ The biological model produces an implicit bias that someone with a mental illness is unlikely to overcome it with treatment.¹⁰⁴ This bias, in turn, can produce a belief that violent behavior may emerge at any moment and in an uncontrolled manner, resulting in enhanced social distance (i.e., the desire not to interact with people with mental illnesses) and enhanced perceived dangerousness of people with mental illnesses.¹⁰⁵ The bias, whether implicit or explicit, that people with mental illnesses are more dangerous than other members in society is, however, unfounded, as “only a few forms of

with prejudice. In contrast, beliefs in the immutability, biological basis, and historical and cross-cultural universality were associated with tolerance.”). For a detailed discussion of how genetic essentialism produces biases against various social groups including race and ethnicity, gender, sexual orientation, criminality, and mental illness, see Dar-Nimrod & Heine, *supra* note 40.

100. Eric Racine et al., *Contemporary Neuroscience in the Media*, 71 SOC. SCI. & MED. 725, 728 (2010) (“Neuro-essentialism designates interpretations that the brain is the self-defining essence of a person, a secular equivalent to the soul.”) (citation omitted).

101. *See id.*

102. *See* Haslam, *supra* note 48, at 821.

103. *See* Hinshaw & Stier, *supra* note 79, at 380; *see also id.* at 370 (“A key point in this regard is that [biological] model accounts are often reductionistic, failing to take into account (a) ecological perspectives involving person-environment fit, (b) views that incorporate both social deviance and mental dysfunction in an evolutionary sense, or (c) biopsychosocial and developmental models emphasizing interaction and transaction across individual vulnerability and contextual influence in the genesis of mental disturbance.” (citations omitted)).

104. *See* Haslam, *supra* note 48, at 820.

105. Phelan, *supra* note 90 (finding that exposure to genetic attributions for mental illness increased the perceived seriousness and persistence of the illness); *see* Haslam, *supra* note 48, at 820.

mental [illness] show any increased risk for dangerous behavior over base rates.”¹⁰⁶

In addition to these negative implicit biases, the biological model of mental illness appears to deepen perceived differences between those with mental illnesses and those without them. Because neurogenetic essentialism creates a belief that mental illnesses are fixed and deeply rooted, those without mental illnesses are more likely to view those with mental illnesses as belonging to a discrete out-group.¹⁰⁷ This “otherization” of the mentally ill is particularly problematic because people are more prone to develop implicit biases against another group if they view the group as different from them.¹⁰⁸ In other words, because the biological model accentuates group differences on a fundamental level, this conceptualization of mental illness may not only elicit stronger negative implicit biases related to dangerousness and prognostic pessimism, but also entrench these biases to a greater extent by categorizing people with mental illnesses as a discrete and neurogenetically disparate out-group.

Because the biological model of mental illness encompasses neurogenetic evidence, as this evidence is increasingly admitted in court cases, the implicit biases associated with the biological model will become increasingly prevalent. Part III explores how these biases manifest themselves in the sentencing context, particularly for capital defendants.

III.

IMPLICIT BIASES PRIMED BY NEUROGENETIC EVIDENCE AND UNEXPECTED CONSEQUENCES FOR A DEFENDANT WITH MENTAL ILLNESS DURING SENTENCING

As courts increasingly admit neurogenetic evidence, the nature of the evidence is likely to prime particularly strong implicit biases associated with the biological model of mental illness rather than the psychosocial model of mental illness. Any biological explanation of mental illness (e.g., a psychiatric diagnosis relying on biological factors) can trigger essentialist thinking of the group and prime the implicit biases discussed above. However, not all

106. Hinshaw & Stier, *supra* note 79, at 376 (finding that increased risk of dangerousness only applied to “antisocial personality disorder and psychopathy, intermittent explosive disorder, alcohol and substance abuse, and a particular form of psychosis involving delusions of being under attack” (citations omitted)); *see also* Linda A. Teplin et al., *Crime Victimization in Adults with Severe Mental Illness: Comparison with the National Crime Victimization Survey*, 62 ARCHIVES GEN. PSYCHIATRY 911, 916–18 (2006).

107. *See, e.g.*, Sheila Mehta & Amerigo Farina, *Is Being “Sick” Really Better? Effect of the Disease View of Mental Disorder on Stigma*, 16 J. SOC. & CLINICAL PSYCHOL. 405, 416 (1997) (finding that “[v]iewing those with mental disorders as diseased sets them apart and may lead to our perceiving them as physically distinct. Biochemical aberrations make them almost a different species”).

108. *See generally* Greenwald & Krieger, *supra* note 15; Dar-Nimrod & Heine, *supra* note 40.

biological explanations will trigger neurogenetic essentialism.¹⁰⁹ Moreover, the strength of the essentializing process and the associated implicit biases depends on the persuasiveness of the link between the underlying essence and mental illness.¹¹⁰

Neurogenetic evidence poses increased risk of bias formation because the nature of the evidence immediately provides a tangible, physical source of causation for mental illness (i.e., the brain or genes) and may be unduly persuasive to people interpreting the evidence.¹¹¹ For example, functional magnetic resonance imaging (fMRI), currently one of the most advanced techniques for measuring and depicting brain function, operates by tracking changes in blood flow in different parts of the brain while the subject performs various mental activities.¹¹² Courts have used such evidence in a variety of contexts, such as diagnosing brain injury; determining mental capacity as evidence for an insanity defense, *mens rea*, and competency; and detecting deception by witnesses.¹¹³ Recently, a court also utilized fMRI evidence during the sentencing phase of a capital case.¹¹⁴ In addition, for defendants diagnosed with psychopathy, some courts have employed research that shows a connection between particular genes and violent behavior to help jurors better understand the mental illness.¹¹⁵ Explaining a mental illness with neurogenetic evidence such as fMRI findings or genetic links to behavior can trigger neurogenetic essentialism, potentially enhancing the essentializing process further if people are “seduced” to perceive the evidence as more credible and persuasive, and ultimately eliciting stronger implicit biases associated with the biological model of mental illness.

Given that neurogenetic evidence may be unduly influential and can elicit stronger implicit biases associated with the biological model of mental illness, neurogenetic evidence can bias sentencing decisions involving defendants with mental illnesses. As discussed in Part I *supra*, courts may utilize neurogenetic evidence in both civil and criminal cases. For example, in civil cases, litigants may use neuroscience evidence to prove actual harm in tort or to show that a

109. For example, while a psychiatric diagnosis can rely on biological causes, it does not provide the same connection between a tangible, physical source of causation (i.e., the brain or genes) and behavior as does neurogenetic evidence. *See, e.g.*, Phillips, *supra* note 23, at 5–9 (listing personality and behavioral traits on the PCL-R checklist used for diagnosing psychopathy).

110. *See* Dar-Nimrod & Heine, *supra* note 40, at 809 (citing various studies that have found that stronger genetic attributions for mental illness are associated with increased prognostic pessimism for the person with mental illness as well as an increased desire for social distance from the person with mental illness).

111. *See supra* Part I, discussing the “seductive allure” of neurogenetic evidence.

112. Neal Feigenson, *Brain Imaging and Courtroom Evidence: On the Admissibility and Persuasiveness of fMRI*, 2 INT’L J. L. CONTEXT 233, 234 (2006).

113. *See id.*

114. *See* Hughes, *supra* note 12, at 340–42.

115. *See* Walker, *supra* note 23, at 1799–1803 (discussing a few cases that have utilized this genotyping evidence).

party lacked capacity to form a contract.¹¹⁶ In criminal cases, courts may utilize neurogenetic evidence in both the guilt phase and sentencing phase.¹¹⁷ To more effectively engage with current literature, which has focused more on the influence of neurogenetic evidence on sentencing decisions, this Note narrows its focus to analyze how the implicit biases primed by neurogenetic evidence affect the sentencing phase in capital cases.

A. *An Illustration: Background on Sentencing in Capital Cases Involving Defendants with Psychopathy*

To illustrate how implicit biases may influence decisions in the courtroom, consider the example of a capital case involving a defendant with psychopathy. By way of background, a capital trial proceeds in two phases: the guilt phase and the sentencing phase.¹¹⁸ In the guilt phase, the prosecution must prove the elements of the crime beyond a reasonable doubt.¹¹⁹ If the jury finds the defendant guilty of the charged crime, then the trial moves on to the sentencing phase, where the jury decides whether the defendant should receive a life sentence or the death penalty.¹²⁰ The defense presents mitigating factors that go against the imposition of the death penalty, while the prosecution presents aggravating factors that support the imposition of the death penalty.¹²¹ Aggravating factors that may influence jurors to believe a defendant deserves the greater punishment of the death penalty commonly include the defendant's "prior convictions, future dangerousness, [and] lack of remorse."¹²² Conversely, mitigating factors that attempt to persuade jurors that a defendant is less than fully culpable and should receive a life sentence commonly include a "family history of physical or substance abuse, mental health issues, and showing of remorse."¹²³

116. See Snead, *supra* note 31, at 1291–92.

117. See *id.* at 1292–93 & nn.135–37 (citing criminal cases that have admitted neuroimaging evidence in connection with claims of mental incompetence, diminished capacity, and insanity defenses).

118. See Fabian, *supra* note 8, at 80.

119. See *id.*

120. See *id.*

121. *Id.* (stating that the jury hears from both the prosecution and defense about how the defendant's crime, personal disposition, prior criminal history, or psychological makeup constitutes either an aggravating or a mitigating factor).

122. Walker, *supra* note 23, at 1787; see also Snead, *supra* note 31, at 1322 n.292 ("Twenty-one of the thirty-eight states with the death penalty include future dangerousness as an aggravating factor.").

123. Walker, *supra* note 23, at 1787; see also Snead, *supra* note 31, at 1319 ("Mitigation evidence is presented in order to 'inspire[] compassion . . . offer[ing] neither justification, nor excuse for the capital crime.'" (alterations and omission in original) (citation omitted)).

B. An Illustration Continued: The Paradoxical Effect of a Biological Explanation for Psychopathy as Mitigating and Aggravating Evidence

Arguing for aggravating or mitigating factors becomes particularly complicated in potential capital cases involving a defendant with psychopathy because characteristics of the mental illness itself (e.g., risk of violent crime) are synonymous with the factors considered to aggravate a sentence to the death penalty.¹²⁴ Psychopathy, at least among males, is defined as “a lifelong persistent condition characterized . . . by aggression beginning in early childhood, impulsivity, resistance to punishment, general lack of emotional attachment or concern for others, dishonesty[,] and selfishness in social interactions.”¹²⁵ While the precise cause of psychopathy is currently unknown, researchers have identified several possible contributors, including genetics, brain abnormalities, and prior substance abuse.¹²⁶ Recent brain scanning technology has also revealed a connection between certain genes and the manifestation of psychopathic characteristics, increasing the use of neurogenetic evidence in cases involving defendants with psychopathy.¹²⁷

Despite the increasing use of neurogenetic evidence during the sentencing phase, particularly by the defense,¹²⁸ research in implicit bias suggests such evidence may produce unexpected consequences for the defendants. As illustrated in Part II *supra*, the biological model of mental illness elicits both stigma-reducing (e.g., reduced attributions of blame) and stigma-enhancing

124. For example, psychopathy is correlated with a high risk of committing violent crimes and although the illness is prevalent in only 1 percent of the population, psychopathy is present in 15 to 20 percent of the incarcerated population. See Christina Lee, Note, *The Judicial Response to Psychopathic Criminals: Utilitarianism over Retribution*, 31 LAW & PSYCHOL. REV. 125, 127 (2007).

125. Grant T. Harris et al., *The Construct of Psychopathy*, 28 CRIME & JUST. 197, 197–98 (2001).

126. Walker, *supra* note 23, at 1784.

127. In the unpublished case, *Tennessee v. Waldroup*, the defense presented genetic evidence of the defendant’s psychopathy (such as low MAOA gene activity, which is linked to violent behavior) and the jury rendered a lesser charge of voluntary manslaughter. See Walker, *supra* note 23, at 1800–01 (citing Hagerty, *supra* note 22); Bernet et al., *supra* note 14, at 1363–65 (describing how low activity of the MAOA and SLC6A4 genes may be linked to violent or aggressive behavior); see also Kent A. Kiehl, *A Cognitive Neuroscience Perspective on Psychopathy: Evidence for Paralimbic System Dysfunction*, 142 PSYCHIATRY RES. 107, 114 (2006) (finding that in fMRI tests, psychopaths had minimal neural responses to stimuli of different degrees, suggesting deficits in paralimbic functioning, which is the portion of the brain that regulates emotional responses); Kent A. Kiehl et al., *Psychopathy and Semantic Processing: An Examination of the N400*, 40 PERSONALITY & INDIVIDUAL DIFFERENCES 293 (2006); Kent A. Kiehl et al., *Temporal Lobe Abnormalities in Semantic Processing by Criminal Psychopaths as Revealed by Functional Magnetic Resonance Imaging*, 130 PSYCHIATRY RES. NEUROIMAGING 27 (2004).

128. One author suggests that one practical reason why defense teams utilize neurogenetic evidence more during the sentencing phase could be that “it would be physically difficult for the state to compel a brain scan of an unwilling person.” Teneille Brown & Emily Murphy, *Through a Scanner Darkly: Functional Neuroimaging As Evidence Of a Criminal Defendant’s Past Mental States*, 62 STAN. L. REV. 1119, 1133 (2010). See also *infra* note 135 (discussing that in Denno’s longitudinal study, there were no instances of the prosecution utilizing behavioral genetics evidence as an aggravating factor during sentencing).

(e.g., reduced perceptions of treatability and rehabilitation) implicit biases against mental illness. While the defense might utilize neurogenetic evidence to argue that a defendant should not be further blamed for his actions because his psychopathy is driven by a process outside of his control, the prosecution could use the same evidence to argue that the defendant possesses a genetic predisposition to violence, will always be violent, and should therefore be executed.¹²⁹ As a result, a defense team may intend to utilize neurogenetic evidence as a mitigating factor, but implicit biases primed by neurogenetic evidence may actually cause the evidence to operate as an aggravating factor against the defendant by increasing the perceived future dangerousness of the defendant. As illustrated in the case of Randall Dale Adams, future dangerousness can be a very persuasive aggravating factor against defendants with psychopathy, as jurors may believe society is better protected with the defendant out of the world entirely.¹³⁰

Although this Note challenges the assumption that neurogenetic evidence generally benefits defendants with mental illness and should be utilized during sentencing, one recent empirical study suggests that the use of neurogenetic evidence in the sentencing phase does, in fact, mitigate rather than aggravate sentences. The study asked 181 United States state trial court judges to impose a prison term on a hypothetical defendant with psychopathy.¹³¹ All judges were provided expert testimony diagnosing the defendant as a psychopath.¹³² However, judges who were provided additional expert testimony that neurogenetic factors¹³³ could explain the defendant's psychopathic behavior handed down a shorter sentence (12.83 years on average) than judges who did not receive the additional neurogenetic explanation (13.93 years on average).¹³⁴

These findings, however, are far from conclusive. First, it is unclear how persuasive neurogenetic evidence may be as aggravating evidence because prosecutors have not relied on such evidence in the sentencing phase of

129. See, e.g., Erica Beecher-Monas & Edgar Garcia-Rill, *Genetic Predictions of Future Dangerousness: Is There a Blueprint for Violence?*, 69 LAW & CONTEMP. PROBS. 301, 302 (2006); Snead, *supra* note 31; Walker, *supra* note 23, at 1783; Abram S. Barth, Note, *A Double-Edged Sword: The Role of Neuroimaging in Federal Capital Sentencing*, 33 AM. J.L. & MED. 501 (2007); Amanda R. Evansburg, Note, *"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 (2001).

130. *Adams v. State*, 577 S.W.2d 717, 731 (Tex. Crim. App. 1979) (en banc), *rev'd in part*, 448 U.S. 38 (1980); Shapiro, *supra* note 8, at 146 n.2 ("Future dangerousness is a requisite sentencing factor in two states, an optional statutory aggravating factor in four states, and an articulated non-statutory aggravating factor in at least two dozen states and the federal system." (internal citations omitted)).

131. See Lisa G. Aspinwall et al., *The Double-Edged Sword: Does Biomechanism Increase or Decrease Judges' Sentencing of Psychopaths?*, 337 SCIENCE 846 (2012).

132. *Id.* at 846.

133. *Id.* (explanations of "low MAOA activity, atypical amygdala function, and other neurodevelopmental factors").

134. *Id.* at 847.

criminal cases.¹³⁵ Second, although in a hypothetical scenario judges purport to give lower sentences to defendants with mental illnesses, recent empirical studies continue to find that neurogenetic explanations of mental illness prime stigma-enhancing implicit biases such as greater perceived future dangerousness. These findings are consistent with the concept that such biases could operate as aggravating evidence and translate into extended incarcerations.¹³⁶

Finally, it is possible that neurogenetic evidence produces a net-aggravating bias for jurors, but does not bias judges in the same manner. The study by Aspinwall et al. focused only on judges¹³⁷ whereas the study by Appelbaum et al. tested members of the general public, which “constitute the pool from which jurors are drawn.”¹³⁸ If the findings of these studies reflect reality, neurogenetic evidence may prime a net-mitigating bias for judges, which can benefit a defendant with mental illness in a non-capital case, potentially leading to a shorter sentence. However, defendants may remain at risk of neurogenetic evidence priming net-aggravating biases in capital cases, where jurors, not judges, recommend either a life sentence or the death penalty during the sentencing phase.¹³⁹ This could explain why, despite the increasing use of neurogenetic evidence by defense teams in capital cases, “more often than not, jurors in these cases have ‘imposed or recommended a sentence of death.’”¹⁴⁰ In light of this inconclusive research, it is imperative to continue to examine how neurogenetic evidence may produce implicit biases and serve as both an aggravating and a mitigating factor in the sentencing of defendants with mental illnesses.

135. For example, although Denno’s longitudinal studies tracked the use of genetic evidence in eighty-one capital cases involving defendants with psychopathy, in all eighty-one cases, there was no instance of the prosecution utilizing the same evidence as an aggravating factor. Denno, *supra* note 13, at 997; *see also* Brown & Murphy, *supra* note 128, at 1133 (explaining that it may simply be practically difficult for the prosecution to compel neurogenetic evidence from a defendant).

136. *See, e.g.*, Paul S. Appelbaum & Nicholas Scurich, *Impact of Behavioral Genetic Evidence on the Adjudication of Criminal Behavior*, 42 J. AM. ACAD. PSYCHIATRY & L. 91, 96 (2014) (critiquing Aspinwall et al., *supra* note 131, because the study used neurogenetic data to support a diagnosis of psychopathy rather than as a direct explanation of criminal behavior, suggesting that the actual difference in sentencing determinations (12.83 versus 13.93 years) would have a limited real-world impact that would not have been detectable in their study).

137. *See* Aspinwall et al., *supra* note 131, at 846.

138. *See* Appelbaum et al., *supra* note 136, at 92–93.

139. *See supra* Part III.A.1.

140. *See* Phillips, *supra* note 23, at 39; *see also* Snead, *supra* note 31, at 1307–08 (“By the metric of whether defendants receive a life sentence or the death penalty, however, [neurogenetic evidence] has proven to not be as successful. There are many cases in which juries were presented with neuroimaging evidence and nevertheless imposed or recommended a sentence of death.”). For an extensive list of capital cases imposing the death penalty over a life sentence despite the introduction of neurogenetic evidence as a mitigating factor, *see* Snead, *supra* note 31, at 1308 n.215.

IV. FUTURE STEPS

This Note primarily explains how implicit biases primed by neurogenetic evidence can harm defendants with mental illnesses and challenges the assumption that neurogenetic evidence is beneficial to defendants with mental illnesses. Although normative solutions are generally beyond the scope of this Note, this Part recommends a few strategies that both judges and defense attorneys can apply to manage the various dangers discussed herein. Additional guidance on how best to manage neurogenetic evidence in the courtroom will arise with continued research into the implicit biases associated with the biological model of mental illness.

First, in capital cases that utilize neurogenetic evidence, judges should consider requiring special jury instructions to highlight that mental illness is a product of a variety of factors beyond biological ones. A special jury instruction could include language that properly frames the explanation of mental illness. Such an instruction could reduce associated implicit biases by highlighting contributors of mental illness beyond the biological explanation, such as psychosocial factors. As defined earlier in this Note,¹⁴¹ “psychosocial” refers to any explanation or conceptualization of mental illness grounded in environmental explanations, such as early childhood experiences, social interactions, and environmental stressors. A comprehensive jury instruction would not only explain mental illness more accurately by acknowledging the complexity of neurogenetic and environmental interactions, but would also reduce implicit biases¹⁴² elicited through neurogenetic essentialism.¹⁴³

To illustrate an example of a more comprehensive jury instruction in cases involving neurogenetic evidence, consider once again a defendant with psychopathy. As illustrated in Part III.B *supra*, defense teams may wish to introduce genetic evidence linking the defendant’s violent behavior to low activity of the MAOA and SLC6A4 genes.¹⁴⁴ In this case, a judge could require a special jury instruction that highlights both biological and psychosocial causes of violent behavior:

141. See *supra* note 84.

142. See Lane et al., *supra* note 63, at 438 (describing the malleable nature of implicit bias).

143. See, e.g., Dar-Nimrod & Heine, *supra* note 40, at 809 (“One study found that messages that portray mental illness as an outcome of both genes and the environment may reduce genetic essentialist reactions. [That study] included a combined genetic and social perspective manipulation []. They found that exposure to the combined perspective significantly reduced perceptions of danger associated with individuals with schizophrenia compared with exposure to a purely genetic account.”); see also Beecher-Monas & Garcia-Rill, *supra* note 129, at 340–41 (“A far better solution is to require that experts testifying about human behavior acknowledge the complexity of the environmental (nurture) and biological (nature) interactions, and ultimately recognize that human beings can and do change their behavior.”); Denno, *supra* note 13 (finding the mitigating effect of biological evidence in capital cases involving defendants with psychopathy was strongest when the evidence was combined with other types of evidence that support mitigating factors).

144. See Bernet et al., *supra* note 14, at 1363–65.

While genes influence behavior, they do not govern or determine it. An individual's genes may predispose the individual to behavioral tendencies, but whether the individual will act out on those tendencies depends on how the genes interact with environmental factors including, but not limited to, childhood abuse, substance abuse, low family income, growing up in a disrupted family, and growing up in high-crime neighborhoods. While research suggests that the MAOA gene is associated with violent behavior, individuals with low MAOA activity more often exhibit violent behavior if they were abused or mistreated as children. Similarly, research suggests that the SLC6A4 gene is associated with aggression, but that aggressive manifestations of the gene depend on the degree of stressful environmental triggers in an individual's life such as employment, finance, housing, health, and relationships.¹⁴⁵

Because the primary goal of the sample language above is to reduce implicit biases associated with the biological model of mental illness, utilizing court-appointed experts, as authorized by Rule 706 of the Federal Rules of Evidence and its state counterparts, is another option.¹⁴⁶ In cases involving neurogenetic evidence, judges could appoint experts in implicit bias to explain to jurors the research discussed in this Note and the particular implicit biases primed by the neurogenetic evidence offered.¹⁴⁷

Second, defense attorneys should carefully consider whether and how to utilize neurogenetic evidence during the sentencing phase of capital cases. Despite the increasing use of neurogenetic evidence by defense teams, "more often than not,"¹⁴⁸ jurors continue to impose or recommend the death penalty.¹⁴⁹ Defense attorneys may intend to utilize neurogenetic evidence as a mitigating factor by arguing that the defendant's mental illness is outside of his control so he should not be further blamed for his actions. However, as illustrated in this Note, neurogenetic evidence could also inadvertently operate as an aggravating factor by priming implicit biases and increasing the perceived future dangerousness of the defendant.

Accordingly, prior to utilizing neurogenetic evidence in cases involving a defendant with mental illness, defense attorneys should consult an implicit bias expert to evaluate whether the evidence will actually benefit the defendant. If

145. This hypothetical jury instruction relies heavily on empirical research cited in Walker, *supra* note 23, at 1811–13.

146. E. Spencer Compton, Note, *Not Guilty by Reason of Neuroimaging: The Need for Cautionary Jury Instructions for Neuroscience Evidence in Criminal Trials*, 12 VAND. J. ENT. & TECH. L. 333, 338 (2010).

147. FED. R. EVID. 706(a) ("The court may appoint any expert that the parties agree on and any of its own choosing.").

148. Phillips, *supra* note 23, at 39.

149. See Snead, *supra* note 31, at 1308 n.215 (providing an extensive list of capital cases imposing the death penalty over a life sentence despite the introduction of neurogenetic evidence as a mitigating factor).

the defense team and implicit bias expert ultimately decide to utilize neurogenetic evidence, the team should introduce the evidence as part of a more comprehensive explanation of the defendant's mental illness, employing additional research regarding psychosocial causes of the mental illness as well.

CONCLUSION

Neurogenetic evidence is not going anywhere. As technology advances and neurogenetic evidence becomes more accurate, courts will increasingly admit such evidence. Neurogenetic evidence may be unduly persuasive because it provides a tangible, physical source of causation (i.e., the brain or genes) to link to observable behaviors. Furthermore, the evidence explains mental illness through a biological model and triggers neurogenetic essentialism, eliciting both stigma-reducing and stigma-enhancing implicit biases against mental illness. Accordingly, while a defense team may intend to utilize neurogenetic evidence during sentencing as a mitigating factor for a defendant with mental illness, the prosecution may utilize the very same evidence as an aggravating factor. The implicit biases associated with neurogenetic evidence affect jurors (and potentially judges) and may affect sentencing decisions in cases involving defendants with mental illnesses, potentially aggravating sentences to the death penalty in capital cases. As a result, scholars must continue research into how neurogenetic evidence should be managed, how implicit biases against mental illness can be reduced, and ultimately how to provide greater protection for defendants with mental illnesses in our legal system.

