

Tulsa Law Review

Volume 43

Number 2 *Daubert, Innocence, and the Future of Forensic Science* | Number 2

Winter 2007

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Recommended Citation

Erica Beecher-Monas, *Circumventing Daubert in the Gene Pool*, 43 Tulsa L. Rev. 241 (2013).

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CIRCUMVENTING *DAUBERT* IN THE GENE POOL

Erica Beecher-Monas*

I. INTRODUCTION

Genetic testimony is appearing in a variety of civil and criminal trials. This testimony is being introduced by plaintiffs and defendants in civil cases, and by prosecutors and defendants in criminal cases. The level of judicial scrutiny to which such expert testimony is subjected varies widely according to context, even among *Daubert* jurisdictions. At one extreme, civil judges confronting expert genetic testimony by and large put the testifying experts through their *Daubert* paces in explaining the scientific basis for their testimony, whether for plaintiffs or defendants. At the other extreme, judges confronting defense evidence of biological links to violence in criminal cases rarely exclude it on the basis of a *Daubert* analysis. Rather, they refuse to order genetic testing in the first place, thus precluding the development of evidence, or they exclude it on policy grounds (the statutory exclusion of substance abuse testimony to negate intent or in mitigation in most states is an example), or characterize it as a redundant family of behavioral testimony, unhelpful, or confusing to the jury.

Despite the claims of numerous scholars that behavioral genetics is the future of criminal justice,¹ the courts remain skeptical about this link. This may be due in part to

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1. See e.g. Lori B. Andrews, *Predicting and Punishing Antisocial Acts: How the Criminal Justice System Might Use Behavioral Genetics*, in *Behavioral Genetics: The Clash of Culture and Biology* 116, 1354–35 (Ronald A. Carson & Mark A. Rothstein eds., Johns Hopkins U. Press 1999) (suggesting that, in the future, the state might “take into consideration not just genes for criminal behavior, but genes thought to be associated with precursors to criminal behavior”); Lindsay A. Elkins, Student Author, *Five Foot Two with Eyes of Blue: Physical Profiling and the Prospect of a Genetics-Based Criminal Justice System*, 17 Notre Dame J.L., Ethics & Pub. Policy 269, 271 (2003) (arguing that “DNA analysis could serve as an antidote to racial profiling” and exploring the “broader implications of using genetic research”); BettyAnn H. Kevles & Daniel J. Kevles, *Scapegoat Biology: As Violence Continues to Ravage Our Society, Researchers are Raising Hopes that Science Alone Can Save Us from Our Worst Natures—Again*, Discover 58, 60 (Oct. 1, 1997) (noting that crime joins “many aspects of human life for which it is claimed that biology is destiny”); Nicole H. Rafter, *Seeing and Believing: Images of Heredity in Biological Theories of Crime*, 67 Brook. L. Rev. 71, 96 (2001) (forecasting that “pop criminologists may soon come up with recommendations for genetic engineering”); Nikolas Rose, *The Biology of Culpability: Pathological Identity and Crime Control in a Biological Culture*, 4 Theoretical Criminology 5, 6–7 (2000); Jasmine A. Tehrani & Sarnoff A. Mednick, *Genetic Factors and Criminal Behavior*, 64 Fed. Probation 24, 26 (2000) (concluding that “crime prevention efforts may be most effective when all risks, social and genetic, are evaluated”); David Wasserman, *Is There Value in Identifying Individual Genetic Predispositions to Violence?* 32 J.L., Med. & Ethics 24, 24 (2004) (forecasting the expanded use of behavioral genetics in crime control); Rhonda J. Yen, *Tourette’s Syndrome: A Case Example for Mandatory Genetic Regulation of Behavioral Disorders*, 27 L. & Psychol. Rev. 29, 29 (asserting that “the vision of Gattaca [a 1997 movie depicting a society driven by genetic engineering] is closer than many of us realize”); Kathy Hudson & Aaron M. Bailey, Keynote Address, *The Human Genome Project, DNA Science and the Law*:

the long shadow cast by the eugenics movement and eugenicists' racial biases, which has made the topic of behavioral genetics a difficult subject to discuss. Differences in gene expression to environmental challenges, on the other hand, do not carry this negative baggage.² As a result, toxic tort plaintiffs increasingly attempt to overcome the difficulties of proving exposure levels, general causation, and heightened susceptibility through genetic testimony.³ Defendants also seek to use genetic information to show alternative causes for plaintiffs' injuries.⁴

What is remarkable about these cases is the difference in care with which the judges examine the scientific validity of the proffered expert testimony. For example, in *Hall v. State*,⁵ a capital murder case, the court admitted (without any analysis) defense testimony that the defendant had characteristics typical of XYY and other genetic disorders, as well as fetal alcohol syndrome.⁶ Not that it did the defendant much good, since the defendant was convicted of capital murder, and even after the U.S. Supreme Court vacated in light of *Atkins v. Virginia*⁷ and remanded for a mental retardation determination,⁸ the Texas Court of Criminal Appeals affirmed defendant's death sentence.⁹ Rather than analyze the basis for the expert testimony, or hold an evidentiary hearing on the matter, the Court of Criminal Appeals merely noted that the experts had disagreed on retardation (although they agreed on defendant's XYY condition), and the defendant's school records failed to note that he was retarded, so the Court of Criminal Appeals held that the evidence supported the conclusion that the defendant was not mentally retarded, and thus eligible to be executed.¹⁰ No analysis was made at any point in the judicial process of the expert testimony regarding the link between XYY and mental retardation.

Perhaps this absence of analysis is due to judicial incredulity as to a link between biology and behavior. Certainly it is true that many links initially "discovered" between violent behavior and genes have been subsequently discredited.¹¹ The XYY defense is a good example of this phenomenon. Studies in British prisons had observed a higher rate

The American Legal System's Response to Breakthroughs in Genetic Science (Washington, D.C., Oct. 19, 2001), in 51 Am. L. Rev. 431, 443 (2002) (forecasting changes when behavioral genetics enters into the criminal justice system in terms of defenses and predisposition to commit crime).

2. To date, roughly 600 environmentally sensitive genes have been identified by the environmental research arm of the Human Genome project. See Natl. Inst. Env. Health Sci., *Environmental Genome Project*, <http://www.niehs.nih.gov/research/supported/programs/egp/index.cfm> (accessed Apr. 5, 2008).

3. See Gary E. Marchant, *Genetic Data in Toxic Tort Litigation*, 14 J.L. & Policy 7, 8 (2006) (noting that "genetic data has the potential to transform toxic torts").

4. See e.g. *Barrow v. Bristol-Myers Squibb*, 1998 WL 812318 at *37 (M.D. Fla. Oct. 29, 1998) (plaintiffs' expert biomarker testimony in silicone breast implant case was inadmissible in light of defense expert testimony that the same biomarkers can appear in women without silicone implants).

5. 160 S.W.3d 24, 33 (Tex. Crim. App. 2004), *cert. denied*, *Hall v. Tex.*, 545 U.S. 1141 (2005).

6. *Id.* at 33.

7. 536 U.S. 304 (2002) (prohibiting execution of mentally retarded defendants).

8. *Hall v. Tex.*, 537 U.S. 802 (2002).

9. *Hall*, 160 S.W.3d at 40.

10. *Id.* at 39–40.

11. See e.g. Sheila Jasanoff, *Just Evidence: The Limits of Science in the Legal Process*, 34 J.L., Med. & Ethics 328, 338 (2006) (observing that prior attempts to link genes and behavior have been premature and controversial, giving as an example the Violence Initiative of the Alcohol, Drug Abuse, and Mental Health Administration, which "was shot through and through with insupportable assumptions about animal as well as human behavior").

of XYY chromosome abnormality in convicts than was found in the general population.¹² Initial conclusions that this was the “crime gene” were later discredited, through studies demonstrating that XYY individuals are no more aggressive than average.¹³ That research, however, was not the last word on XYY. More recent studies show that men afflicted with this chromosomal abnormality tend to be less intelligent than the norm, more impulsive and hyperactive.¹⁴

But links between genes and violence are not the only connections that have been discredited on further study. Links between diseases and genes also have a history of being “discovered” only to be subsequently discredited.¹⁵ Yet contrast the cursory treatment of expert XYY testimony in *Hall* with the extensive judicial analysis of expert testimony regarding possible genetic influences in the claims that manganese fumes in the welding process caused the plaintiffs’ Parkinson’s disease in the welding fumes cases.¹⁶ There, the court held extensive *Daubert* hearings on the evidence regarding the interaction of environmental stimuli and genetic predisposition to the incidence of Parkinson’s disease, summarizing and analyzing the scientific basis for each expert’s testimony.

Adding further fuel to the fires of controversy is the fundamental question of what implications follow from a genetic link to violence (or disease). The majority and dissent in *Schriro v. Landrigan*,¹⁷ for example, disagreed primarily over what effect the disputed genetic testimony would have had on the jury: The majority thought it would not have helped the defendant (and thus counsel’s failure to develop it would not be ineffective assistance), and the dissent thought that the jury should have been able to weigh this information in its deliberations.¹⁸

The implications to be drawn from genetic data are equally contested in toxic tort cases, however, so that cannot be the basis for the discrepancy in the way the criminal courts analyze admissibility questions. For example, disputes over the significance of biomarkers (chromosomal translocations, change in gene expression, protein concentration or metabolites) were central in cases involving whether exposure to benzene caused plaintiffs’ acute myelogenous leukemia.¹⁹ Similarly, in silicone breast

12. See Patricia A. Jacobs et al., *Aggressive Behavior, Mental Sub-normality, and the XYY Male*, 208 *Nature* 1351, 1351–52 (1965).

13. See David Wasserman & Robert Wachbroit, *Introduction: Methods, Meanings, and Morals*, in *Genetics and Criminal Behavior* 1, 9 (Cambridge U. Press 2001) (discussing XYY chromosome abnormality). Nonetheless, it may be premature to completely disregard a link between jail and genes.

14. These factors (which are genetic) may make them more likely to get caught when they do violate social norms. See Michael J. Rutter et al., *Antisocial Behavior by Young People* (Cambridge U. Press 1998) (discussing studies).

15. See e.g. Kirk E. Lohmuller et al., *Meta-Analysis of Genetic Association Studies Supports a Contribution of Common Variants to Susceptibility to Common Diseases*, 33 *Nature Genetics* 177, 177 (2003) (finding that when a genetic link to a complex disease is first published, the likelihood that other studies will be able to confirm it is roughly one in three); Jack Lucenti, *Gene Association Studies Typically Wrong: Reproducible Gene-Disease Associates are Few and Far Between*, 18 *Scientist* 20, 20 (2004).

16. *In re Welding Fume Prod. Liab. Litig.*, 2006 WL 4507859 at **23–36 (N.D. Ohio Aug. 8, 2006) (providing extensive analysis denying defendants’ motions to exclude plaintiffs’ experts, and giving a chart summarizing the experts, thrust of each expert’s testimony, and court’s finding on validity).

17. 127 S. Ct. 1933 (2007).

18. *Id.* at 1943–55.

19. *Compare Expert Testimony: Jury Returns Verdict for Oil Company after Testimony on Missing Disease*

implant cases, the implications that could be drawn from the presence of biomarkers in plaintiffs was a matter of heated dispute.²⁰

Whatever the reason, civil courts scrutinize the genetic expert testimony far more seriously than the criminal courts appear to. This Essay explores the phenomenon in three parts. Part One discusses the sophisticated analysis engaged in by the civil courts faced with arguments about genetic evidence. Part Two contrasts this approach with that of the criminal courts, which tend to avoid having to analyze the scientific basis of genetic behavioral arguments. Part Three addresses the important question of what the criminal courts should do, faced with the uncertainties inherent in genetic science. It concludes that expert testimony in criminal cases deserves at least the gatekeeping scrutiny given to expert testimony in civil cases; this is far too important an area to be left to the ad hoc avoidance techniques currently prevalent in our justice system.

II. THE CIVIL COURTS GRAPPLE WITH GENETIC SCIENCE

Toxic tort plaintiffs face great difficulties in establishing causation. Both general and specific causation are usually contested, exposure levels are difficult to establish, and genetic testimony is being used to overcome these obstacles.²¹ Now that gene expression assays have become relatively cheap (hundreds of dollars) and quick (several days), we can expect to see increased use of testimony about chromosomal translocations, which indicate exposure (though not to what), changes in gene expression, protein concentration, and the presence of metabolites.²²

The welding fumes cases²³ offer a good example of judicial gatekeeping of expert genetic testimony. There, plaintiffs suffering from Parkinson's disease claimed that their exposure to manganese fumes from welding triggered their injuries.²⁴ In a well-reasoned opinion, the court addressed the defendants' *Daubert* motions, seeking to exclude plaintiffs' experts on a variety of bases, but primarily because each expert was unqualified to testify about the complex issues involved.²⁵ In particular, the defendants objected to the plaintiffs' toxicologist, who proposed to explain the mechanism of manganese absorption and its effects on the body, including injury to the central nervous system.²⁶ This part of the testimony the court ruled admissible, but when the expert proposed to testify about the interplay of genetics with the environment in the etiology of

Marker, 22 Chem. 193 (1998) (discussing *Wells v. Shell Oil Co.* and defense arguments that the absence of biomarkers in plaintiffs' blood showed that benzene could not have caused plaintiffs' acute myelogenous leukemia because benzene-caused disease shows breaks in the fifth and seventh chromosomes, which this plaintiff did not have) with *Lavendar v. Bayer Corp.*, No. 93-C-226-k (W. Va. Cir. May 5, 1998) (rejecting same defense without evidence that absence of biomarkers excluded benzene as a cause).

20. See *Barrow*, 1998 WL 812318 (excluding plaintiffs' expert's biomarker testimony as insufficient to establish causation in light of defense expert testimony that the same biomarker can be found in women without silicone implants).

21. See Marchant, *supra* n. 3, at 19–20.

22. See e.g. Mark Hansen, *DNA Poised to Show its Civil Side*, ABA J. 18–19 (Mar. 2008) (discovering the use of cytokine testing as sort of “genetic fingerprints” to determine whether chemical exposure caused plaintiff's harm).

23. *In re Welding Fume Prod. Liab. Litig.*, 2006 WL 4507859.

24. *Id.* at *2.

25. *Id.*

26. *Id.* at **10–11.

Parkinson's, the court remarked that merely reading the literature does not make someone an expert, and that "a given expert's tendency to opine about areas outside his particular expertise does not . . . disqualify him from testifying about his true, core area of expert knowledge."²⁷ The plaintiff's statistics expert also proposed to testify about genetic data, and to critique the defense's epidemiology studies on a statistical basis.²⁸ This, the court found admissible. An extensive analysis of the expertise of each of the proffered experts and their areas of expertise followed, and each of plaintiffs' experts was permitted to testify, but with certain limitations.²⁹

A similarly thorough analysis was undertaken by the Third Circuit in the *TMI Litigation*³⁰ with respect to radiation biomarker testimony, although the court ultimately excluded the plaintiffs' testimony because plaintiffs had been tested beyond the two-year limit for which the tests were reliable.³¹ Because plaintiffs could not directly establish their radiation levels, they had attempted to demonstrate exposure through the concentration of dicentric chromosomes in lymphocytes in order to establish exposure to radiation.³² While the court in principle endorsed the counting of dicentric chromosomes to establish both radiation exposure and dose, it found no evidence that this was a reliable test beyond the two years for which it had been studied (the plaintiffs were not tested until fifteen years after exposure).³³ The court also excluded evidence of a more stable biomarker for radiation exposure, the FISH test, as having been untimely presented.³⁴

Yet another example of the courts' painstaking analyses in civil cases can be found in the silicone breast implant cases, where plaintiffs argued that they were genetically susceptible to adverse reactions in order to overcome epidemiological studies showing that there was no doubling of the risk (as generally required to demonstrate causation).³⁵ This was admissible evidence in some cases, but excluded where plaintiffs could not show which gene had predisposed them to silicone reactions in *Hall v. Baxter Health Care Corporation*.³⁶ In each of these cases, judges carefully analyzed the testimony presented. They considered the expertise of the expert and the basis for the proffered testimony. They permitted testimony in some areas and excluded some testimony as beyond the expertise of the expert or as unsupported.

The cases with the most sophisticated approach to genetic testimony, however, are vaccine cases, which are not even required to apply *Daubert* in their analyses.³⁷ The

27. *Id.* at *6.

28. *In re Welding Fume Prod. Liab. Litig.*, 2006 WL 4507859 at **15–16.

29. *Id.* at **6–22.

30. *In re TMI Litig.*, 193 F.3d 613 (3d Cir. 1999).

31. *Id.* at 690–93.

32. *Id.* at 690.

33. *Id.*

34. *Id.* at 692–93.

35. See Marchant, *supra*, n. 3, at 25–26.

36. 947 F. Supp. 1387 (D. Or. 1998). See also *Stone v. Williamson*, 2007 WL 1135686 (Mich. App. Apr. 17, 2007) (excluding defense expert testimony of genetic predisposition in a medical malpractice action for lack of evidence that the plaintiff had this predisposition).

37. See e.g. *Hopkins v. Sec. of Health & Hum. Servs.*, 2007 WL 2454038 (Fed. Cl. Aug. 10, 2007) (explaining the procedural stance of a case brought under the National Vaccine Injury Compensation Program).

context is different from an admissibility decision, in that the question before the court is causation rather than admissibility.³⁸ Many of these cases do explicitly apply a *Daubert* standard and analysis, however, and even those that do not, tend to use a sound reasoning methodology.³⁹ In order to prevail under the National Vaccine Injury Compensation Program,⁴⁰ a plaintiff must either suffer from a vaccine-related injury listed as a statutorily presumed cause, or prove that the vaccine was the cause in fact of the harm suffered.⁴¹ Quite frequently, vaccine plaintiffs claim that the vaccine was an environmental trigger that activated an underlying predisposition to a particular disease.⁴²

In analyzing the plaintiffs' claims, the court determines whether the expert's testimony exceeds the scope of expertise demonstrated, and summarizes each expert's position.⁴³ In addition, the court reviews the literature relied upon by the expert, assesses whether the literature supports the expert's testimony, and makes a decision. The court's reasoning is thus clear, accessible, and accountable.⁴⁴ If courts are looking for a good model for making sound *Daubert* decisions, they could do far worse.

III. CIRCUMVENTING ANALYSIS IN THE CRIMINAL COURTS

The central problem for the criminal courts is the lack of a coherent theoretical basis for admitting or excluding expert genetic testimony. In civil cases, the underlying theory making genetic testimony relevant is causation: That an environmental stimulus (such as a chemical produced by the defendant) can trigger genetic predisposition to disease (or alternatively, that the environmental trigger was irrelevant to genetically predisposed disease). For criminal cases, this theory is a non sequitur in a system that sees crime as a chosen course of action rather than a disease. Moreover, the notion of a genetic predisposition comes dangerously close to prohibited propensity evidence—the danger of conflating a propensity to act in a certain way with having acted that way on a given occasion.⁴⁵ This is a valid concern, but not one that the courts explicitly rely on in their analyses. Nor does it affect the majority of genetic testimony, which is proffered in mitigation, or to substantiate some mental illness (either for the insanity defense or that falls short of legal insanity but negates intent).

38. *Id.* at **8–9.

39. *Id.*

40. 42 U.S.C.A. §§ 300aa-10 to 300aa-19 (West Supp. 2003).

41. *Hopkins*, 2007 WL 2454038 at *8.

42. See e.g. *Hopkins*, 2207 WL 1135686 (hearing loss); *Stevens v. Sec. of Health & Hum. Servs.*, 2006 WL 659525 (Fed. Cl. Feb. 24, 2006) (multiple sclerosis); *Bubb v. Sec. of Health & Hum. Servs.*, 2005 WL 1025707 (Fed. Cl. Apr. 29, 2005) (multiple sclerosis); *Lee v. Sec. of Health & Hum. Servs.*, 2005 WL 1125672 (Fed. Cl. May 6, 2005) (fibromyalgia); *Ryman v. Sec. of Health & Hum. Servs.*, 65 Fed. Cl. 35 (2005) (rheumatoid arthritis).

43. See *id.*

44. See *id.*

45. For example, it is difficult to see how the court-appointed expert's testimony that defendant had a "genetic predisposition to psychopathy" that was "essentially untreatable" helped the defendant in *People v. Smith*, 150 P.3d 1224, 1234 (Cal. 2007).

A. Policy Exclusions

There are few more well established links than those between genes and alcohol and between alcohol and violence.⁴⁶ Defense lawyers frequently attempt to draw on this connection to violent behavior, either to negate mens rea (in those jurisdictions that have not abolished this defense⁴⁷) or in mitigation.⁴⁸ Because only a few states permit alcohol or drug abuse to negate intent, or to be a mitigating circumstance in sentencing, however, it is rarely admissible.⁴⁹ Thus, some of the strongest genetic evidence is typically inadmissible for policy reasons.⁵⁰

In addition to the overt policy determinations that derive from legislation, courts make a number of policy-based determinations when they exclude genetic testimony as confusing, or redundant. The most common basis for exclusion is that expert testimony is redundant of family history testimony, which can be obtained from lay witnesses (family members, for example).⁵¹ In this, the courts are mistaken, however, because family testimony about behavioral abnormalities is no more sufficient to establish genetic disease than it would be when any other kind of illness is at issue. Expert testimony is needed to give the fact finder important context, diagnosing and explaining the illness. Family testimony may give information for mitigation, but it does little to explain the significance of biology to the act for which the defendant is being tried or sentenced.

Refusal of the courts to order testing is another of these judicially created policy based exclusions. As soon as studies linking XYY chromosomal abnormalities with criminal propensities became available in the 1970s, defense attorneys attempted to use them to argue that their clients should be exonerated. In the United States, the few cases that attempted such a genetic defense were unsuccessful.⁵² In each of these cases, the

46. See Laura A. Baker et al., *Behavioral Genetics: The Science of Antisocial Behavior*, 69 L. & Contemp. Probs. 7, 29 (2006) (noting that although well studied, the relationship is not well understood).

47. See e.g. *Clarke v. Ariz.*, 548 U.S. 735 (2006) (upholding Arizona's right to preclude expert testimony about mens rea).

48. See e.g. *People v. Mertz*, 842 N.E.2d 618, 644–45 (Ill. 2005) (defense expert testified that defendant had genetic predisposition to alcohol dependence and mood disorder); *State v. Manning*, 885 So. 2d 1044, 1096–97 (La. 2004), cert. denied, *Manning v. La.*, 544 U.S. 967 (2005) (defense psychiatrist proffered mitigation testimony regarding alcohol problems that may have stemmed from a genetic predisposition).

49. See e.g. *Mont. v. Egelhoff*, 518 U.S. 37, 56 (1996) (upholding the Montana legislature's right to exclude evidence of intoxication as a defense to any offense unless the defendant did not know the substance was intoxicating).

50. Even where there is no legislative prohibition on substance abuse testimony, courts may preclude it as ambiguous. See e.g. *Jones v. Schriro*, 450 F. Supp. 2d 1023, 1044–45 (finding no ineffective assistance for failure to develop neuropsychological testimony about defendant's genetic predisposition to alcoholism).

51. See e.g. *Schurz v. Schriro*, 2005 U.S. Dist. LEXIS 22326 at **32–36 (D. Ariz. Sept. 29, 2005) (acknowledging that genetic predisposition to alcoholism testimony should have been developed as mitigation testimony but declining to find ineffective assistance because it would have been cumulative of family history that was presented through lay witnesses).

52. See *State v. Spivey*, 692 N.E.2d 151, 165–66 (Ohio 1998) (defense expert diagnosed defendant as having XYY syndrome and testified that this chromosomal abnormality put defendant at risk for committing criminal acts); *People v. Tanner*, 91 Cal. Rptr. 656, 658–59 (App. 2d Dist. 1970) (finding that neither the link to aggressive behavior nor a chromosomal contribution to legal insanity were established); *Millard v. State*, 261 A.2d 227, 228 (Md. Ct. Spec. App. 1970) (upholding trial court's refusal to submit the genetic issue to the jury because the expert failed to demonstrate a link between the XYY condition and the legal definition of insanity); *People v. Yuki*, 372 N.Y.S.2d 313, 317–20 (Sup. Ct. 1975) (refusing to order genetic testing or to permit defendant's father to pay for genetic testing because the evidence of a genetic link to violence was not reliably

courts considered the link between genes and violence too attenuated, and either refused to order genetic testing of the defendant,⁵³ or refused to submit the genetic issue to the jury.⁵⁴

Defendants with arguments based on stronger science have not fared any better. More sophisticated studies of genetic anomalies and their link to crime were at issue in *Mobley v. State*,⁵⁵ where the defense attempted to obtain expert testimony to mitigate the sentence of a capital murder defendant on the basis of monoamine oxidase A (MAOA) abnormality, an attempt that was also unsuccessful. The defendant came from a family that had been troubled with serious behavioral problems for generations, and the defense sought to obtain genetic testing information for use as mitigation evidence.⁵⁶ The defendant appealed his murder conviction based on the trial court's failure to order genetic testing.⁵⁷ The Georgia Supreme Court rejected the appeal, finding that the scientific basis for a genetic link was too uncertain.⁵⁸ New counsel, filing a habeas petition, claimed that Mobley's former counsel was ineffective for, among other reasons, declining an offer from Mobley's father to pay for the testing.⁵⁹ While the habeas court vacated, the Georgia Supreme Court reversed, reinstated the sentence on appeal, and denied reconsideration, explaining that no expert testimony would be needed because Mobley's father's cousin's testimony about the family history of behavioral problems was sufficient to present the "genetics theory."⁶⁰ Such an approach—declining to hear expert causation testimony because lay testimony had been presented—would be difficult to imagine in a civil case.

Not only do the courts foreclose the possibility of expert testimony by refusing requests for testing, but they are largely unsympathetic to claims that expert testimony should have been developed, but was not. Claims of ineffective assistance of counsel for failure to develop the genetic aspect of the defense have rarely been successful. *Landrigan v. Stewart*⁶¹ involved an ineffective assistance claim, for failing to present mitigating evidence during the penalty phase of the petitioner's capital murder trial. Unlike Mobley, there was no family history testimony. The petitioner had refused to permit his birth mother or ex-wife to testify about the family history of alcoholism and behavioral problems. The petitioner contended, however, that he would have cooperated had the attorneys presented expert evidence that his "biological background made him what he is."⁶²

established); *State v. Roberts*, 544 P.2d 754, 758–59 (Wash. App. Div. 3 1976) (affirming trial court's denial of genetic testing because of the uncertain causal connection between XYY and criminal conduct).

53. *Yukl*, 372 N.Y.S.2d at 317–20; *Roberts*, 544 P.2d at 758–79.

54. *Tanner*, 91 Cal. Rptr. at 659–60; *Millard*, 261 A.2d at 232.

55. *See Turpin v. Mobley*, 502 S.E.2d 458, 465–66 (Ga. 1998) (finding no ineffective assistance of counsel in failing to accept defendant's father's offer to pay for genetic testing for MAOA deficiency analysis after the trial court refused to pay for it).

56. *See Deborah W. Denno, Revisiting the Legal Link between Genetics and Crime*, 69 L. & Contemp. Probs. 209, 209 (2006) (discussing *Mobley v. State*, 455 S.E.2d 61 (Ga. 1995)).

57. *Mobley*, 455 S.E.2d 61.

58. *Id.* at 66.

59. *Turpin*, 502 S.E.2d at 463.

60. *Id.* at 465–66.

61. 272 F.3d 1221, 1224 (9th Cir. 2001).

62. *Id.* at 1228.

The available evidence that his counsel could have developed and presented at Landrigan's sentencing hearing, but did not, include the facts that Landrigan had an organic brain disorder, had been abandoned by his alcoholic mother at six months, and adopted by alcoholic parents; his biological father was on death row in Arkansas for capital murder; and that Landrigan's substance abuse began at an early age. The en banc appeals court noted that, subsequent to Landrigan's conviction and sentencing, an expert had done a thorough neuropsychosocial evaluation, concluding that Landrigan's genetic makeup, in utero exposure to teratogenic substances, early maternal rejection, and his troubled interactions with his adoptive family resulted in disordered behavior that was beyond his control and left him unable to function in a society that expects individuals to operate in an organized and adaptive manner.⁶³ Defense counsel had failed to uncover any of this information, despite the obligation to conduct a thorough background investigation.

The United States Supreme Court reversed, upholding the district court's determination that even with an evidentiary hearing, Landrigan would not be entitled to habeas relief.⁶⁴ Characterizing the omitted mitigation evidence as "weak," the Court determined that Landrigan had waived his right to mitigating evidence by his refusal to permit his mother or ex-wife to testify about the family history. Because the expert testimony "would not have changed the result," the failure to present it was not ineffective assistance.⁶⁵

Justice Stevens, joined by Justices Souter, Ginsburg, and Breyer, dissented, explaining that it was not until years after his conviction that Landrigan even learned about his organic brain disease, that the reason he did not know about it was that his counsel had failed to investigate, and that for the majority to assume he would have refused to permit such testimony was "pure guesswork."⁶⁶ Curiously, both majority and dissent in *Landrigan* agreed that counsel's assistance was ineffective. They disagreed about whether the failure to present the omitted testimony would have made any difference. The majority thought not. The dissent thought that there was enough of a question to put it to the jury.

Both *Mobley* and *Landrigan* illustrate two erroneous judicial assumptions about expert testimony concerning genetic influences. First, as noted previously, the Court assumed that family history is redundant of expert testimony, which is (or should be) incorrect. There can be many reasons for a family history of violence. If some of them are biological, rather than lifestyle choices, that evidence should be presented to the jury. Second, the Court assumed that the jury would be confused about the evidence, that it would hurt as much as help the defendant. While judges may be correct that reasonable people may differ about what inferences to draw, that does not mean that they should take the question from the jury. It is precisely when reasonable people can differ that the voice of the community is most important. Whether this evidence hurts or helps is a quintessential jury question, and the evidence should be presented for their

63. *Landrigan v. Schriro*, 441 F.3d 638, 645 (9th Cir. 2001), *rev'd*, 127 S. Ct. 1933 (2007).

64. *Schriro v. Landrigan*, 127 S. Ct. 1933.

65. *Id.* at 1943–44.

66. *Id.* at 1944 (Stevens, Souter, Ginsburg & Breyer, JJ., dissenting).

determination.

B. *Negating Mens Rea*

Very seldom is genetic testimony proffered at the guilt stage of the trial. Even when it is, it has been met with some skepticism by juries. For example, in *State v. Payne*,⁶⁷ a molecular neurobiologist testified about the relationship of neurotransmitters (specifically serotonin) to impulsive violence.⁶⁸ The jury was instructed on the elements of first degree (premeditated) murder, second degree (knowingly killing another), and voluntary manslaughter (killing under the influence of passion under adequate provocation) as well as on diminished capacity.⁶⁹ Rather than convict on first degree murder, with which the defendant had been charged, the jury convicted the defendant of second degree murder, in what might be seen as a compromise verdict.⁷⁰ The trial court also considered the expert testimony in mitigation at sentencing, but found it a slight factor that did not excuse or mitigate the defendant's conduct, because it did not consider the crimes he committed to be impulsive.⁷¹ On appeal, the court upheld the conviction. Nonetheless, one might view the expert testimony as having had some impact on the jury.

Judges are also skeptical about the inferences that can be drawn from genetic testimony. In one case where the appellate court acknowledged that there had been plain error in the jury instructions on voluntary intoxication to negate mens rea, the court nonetheless held it to be harmless. Although the issue had been adequately raised by an addiction expert testifying about the effects of methamphetamine, including the genetic basis for addiction, the court nonetheless held that "no reasonable juror could have concluded, based on the entire record in this case . . . that [defendant] did not deliberately intend to kill [the victim]."⁷²

Just as genetic testimony is used in civil cases to demonstrate exposure through biomarkers that would not occur in the absence of exposure, connecting genetic anomalies to mental conditions appears to be a prevalent use of genetic testimony.⁷³ For example, in *State v. Sexton*,⁷⁴ the defendant proffered genetic testimony to negate mens rea, that his drug use had triggered a latent pre-existing mental illness.⁷⁵ The defendant had voluntarily ingested L.S.D. two or three weeks before killing a complete stranger

67. 2002 WL 31624813 (Tenn. Crim. App. Nov. 20, 2002) (upholding conviction for second degree murder).

68. *Id.* at **4–6.

69. *Id.* at *13.

70. *Id.* at *1.

71. *Id.* at **17–18.

72. *Malone v. State*, 168 P.3d 185, 203 (Okla. Crim. App. 2007).

73. See e.g. *State v. Hughbanks*, 792 N.E.2d 1081, 1103 (Ohio 2003) (genetic tendency to mental illness); *Davis v. State*, 2004 WL 253396 (Tenn. Crim. App. Feb. 11, 2004) (involving expert defense testimony of a genetic predisposition for mental illness); *Cauthern v. State*, 145 S.W.3d 571, 588 (Tenn. Crim. App. 2004) (defense psychiatrist attempting to present evidence of a genetic predisposition to impulsive behavior); *State v. Maraschiello*, 88 S.W.3d 586, 599 (Tenn. Crim. App. 2000) (defense trial testimony that defendant probably had a genetic predisposition to delusional disorders).

74. 904 A.2d 1092 (Vt. 2006) (upholding defendant's right to present evidence regarding diminished capacity but not insanity).

75. *Id.* at 1096.

who happened to be riding by his house on her bicycle, because, as defendant told the expert, he was feeling an urge to "kill people and 'gather their souls.'" ⁷⁶ The court-appointed psychiatrist concluded that the defendant was insane at the time of the offense, resulting from a substance-induced psychosis or from a previously undiagnosed schizophrenic disorder triggered by the substance abuse. ⁷⁷ Because of the prohibition on using voluntary substance abuse as an excuse, the Vermont Supreme Court held that the defendant was entitled to present this evidence as relevant to mens rea, but could not assert an insanity defense on its basis. ⁷⁸

C. Mitigation Testimony

Most capital cases to date that have attempted to bring in genetics testimony have done so in the context of mitigation during the penalty phase of the case, rather than at the guilt phase of the proceedings. ⁷⁹ One reason for the predominance of genetic testimony at the sentencing stage is that the admissibility standards for mitigating evidence during sentencing are fairly generous. In a capital case, any relevant evidence is admissible in mitigation. ⁸⁰ Although *Daubert* explained that to be relevant, scientific evidence must demonstrate its validity, ⁸¹ in most of the recent cases involving genetics testimony, if the defense presents such evidence in mitigation, the court has found it admissible. ⁸² As a practical matter, even when freely admissible, genetic predisposition testimony does not appear to help the defendants much. ⁸³ The proffered testimony is rarely scrutinized for scientific validity, and is generally admitted. It rarely does the defendant much good, either, since the jury has already convicted for a heinous crime. ⁸⁴

Part of the reason that genetic testimony in mitigation is so ineffective may stem from the way it is presented. Rather than a molecular neurobiologist, like the expert who

76. *Id.* at 1095.

77. *Id.* at 1096.

78. *Id.* at 1099–1111.

79. See Denno, *supra* n. 55, at 221 (observing that "genetics evidence is submitted primarily as a mitigating factor in death penalty cases rather than as a defense relating to the defendant's level of culpability at the trial court level" and noting that admissibility criteria for mitigating evidence are more flexible than those used in defenses).

80. *Penry v. Lynaugh*, 492 U.S. 302, 327–28 (1989) (explaining that "the jury must be allowed to consider and give effect to mitigating evidence relevant to a defendant's character or record or the circumstances of the offense"); *Hitchcock v. Dugger*, 481 U.S. 393, 394, 399 (1987); *Lockett v. Ohio*, 438 U.S. 586, 604 (1978) (plurality opinion).

81. *Daubert v. Merrill Dow Pharms., Inc.*, 509 U.S. 579, 592–93 (1993).

82. See e.g. *Manning*, 885 So. 2d at 1096–97 (defendant's expert testified during sentencing that the defendant's alcohol problems and low mental ability may have stemmed from a genetic predisposition); *Hughbanks*, 792 N.E.2d at 1104 (finding mitigating factors, including psychiatrist's testimony about inherent mental disease, outweighed by aggravating factors).

83. See e.g. *Stevens v. State*, 770 N.E.2d 739, 754–55 (Ind. 2002) (defendant sentenced to death despite mitigating evidence about defendant's genetic predisposition); *Hughbanks*, 792 N.E.2d at 1104 (genetic testimony regarding inherent mental disorder was outweighed by aggravating circumstances); but see *Arausa v. State*, 2003 WL 21803322 at *2 (Tex. App. Aug. 6, 2003) (finding that trial court had not erred in declining to appoint defense psychiatrist to develop mitigating evidence on genetic predisposition to violence among victims of abuse).

84. See Erica Beecher-Monas, *Evaluating Scientific Evidence: An Interdisciplinary Framework for Intellectual Due Process* 150 (Cambridge U. Press 2007) (discussing the impact of cognitive dissonance on jury sentencing decisions).

testified in *Payne*, most genetic testimony in mitigation is proffered by psychologists who have no particular expertise in genetics, and who testify in only the most general way about how the family history of violence demonstrates a genetic predisposition in the defendant.⁸⁵

IV. WHAT *SHOULD* COURTS DO WITH GENETIC INFORMATION?

What is the relevance of a biological predisposition to acts of violence or sexual violence? Does a genetic link to crime negate mens rea, constitute evidence for a legal defense, like insanity, or is it a mitigating circumstance calling for more lenient sentencing?⁸⁶ Does it mean that the defendant is not responsible, is less responsible, or does it affect responsibility at all? Or is the *Landrigan* majority correct that genetic testimony will only confirm the jury's inclination to see the defendant as "bad to the bone?" These are important questions, and implicate important constitutional concerns, like the right of the defendant to present a complete defense, and the necessity of the jury's hearing all the information it needs to make a reasoned moral judgment about imposing the death penalty.

Legal liability for crime is a two-part inquiry: actus reus (the voluntary act) and mens rea (the intent necessary to commit the act).⁸⁷ The question of whether expert testimony can help the jury decide whether the defendant had the requisite intent is extremely controversial.⁸⁸ There are various defenses (insanity is one) that may implicate genetic information. In addition, the sentence of the convicted defendant will depend on the assessment of personal culpability. Genetic testimony has been proffered as relevant to all of these elements.⁸⁹ Because the standards for relevance are broad (any tendency to make a fact of consequence to the issues at trial more or less probable), expert genetics testimony has rarely been excluded on this basis. Instead, the courts use other circumventing gambits to keep it from the jury.

Not all genetic testimony should be admitted. Quite a bit of expert testimony proffered in criminal cases is conclusory and unscientific. The courts ought to keep such testimony out. After the Supreme Court's *Daubert* trilogy of cases, the question for admissibility of scientific evidence now is whether the testimony has met the standards

85. See e.g. *Berryman v. Ayers*, 2007 WL 1992049 at *87 (E.D. Cal. July 10, 2007) (denying evidentiary hearing where new evidence was proffered by clinical psychologist and psychiatrist who proposed to testify to a predisposition to alcoholism and this was cumulative of family testimony); *Manning*, 885 So. 2d at 1097 (psychological testimony that capital defendant was a slow learner with genetic propensity to alcoholism); *Hughbanks*, 792 N.E.2d at 1101 (mitigation testimony at capital sentencing proffered by psychiatrist testifying that the defendant had a genetic tendency toward schizophrenia, based on his father's paranoid schizophrenia).

86. See Nita A. Farahany & James E. Coleman, Jr., *Genetics and Responsibility: To Know the Criminal from the Crime*, 69 L. & Contemp. Probs. 115, 146 (2006) (distinguishing between mens rea, defined as mental culpability, and the excuse of insanity). The authors opine that "[a] successful claim that a defendant lacks the capacity to form mens rea is hard to imagine." *Id.* at 125.

87. See Wayne R. LaFare, *Criminal Law* § 5.1, 239 (4th ed., West 2003). Some crimes do not require proof of mens rea, but those crimes are not those in which genetic testimony is typically involved.

88. See e.g. *Clark*, 548 U.S. 735 (finding exclusion of expert testimony regarding mens rea constitutional).

89. See Farahany & Coleman, *supra* n. 85, at 163 (concluding that "genetic predisposition evidence is irrelevant to both liability and the defenses of justifications and excuses, it should have little role in the negation or mitigation of a defendant's criminal liability"). Notably, Professors Farahany and Coleman do not contend that proffered expert testimony has been widely excluded on the basis of relevance. Rather, they argue that the criminal law currently "has no place . . . for behavioral genetics evidence." *Id.* at 149.

and methods of science.⁹⁰ If anything, there should be more judicial screening of the expert testimony proffered in these cases. Most of the genetics testimony cases involved psychologists testifying that because there was a family history of bad behavior, and that because the defendant exhibited some of the symptoms associated with a particular anomaly, there might be a chromosomal anomaly to account for it.⁹¹ Leaving such testimony to psychologists untrained in genetics, and without specific testing that could be expected to link the anomaly to the defendant may be a tactical problem for these defenses. The courts ought to demand more. As the Supreme Court explained in *Daubert*, the requirement that expert testimony be helpful to the jury, “supported by appropriate validation—i.e., ‘good grounds,’ based on what is known,” is a condition of relevance.⁹²

But the kind of sophisticated evaluation involved in a case like *Landrigan*,⁹³ where the neuropsychological evaluation discussed the convergence of factors including the defendant’s “genetic makeup, in utero exposure to teratogenic substances, early maternal rejection, and his troubled interactions with his adoptive family” is difficult to dismiss as unscientific. Indeed, rather than any lack of science, the Supreme Court in *Landrigan* was concerned that the testimony would not have helped the defendant. This is a major controversy, but it is not the province of the judge. Precisely because reasonable minds can differ on the inference to be drawn from evidence of a genetic predisposition it should be submitted to the jury.

Similarly, in *Payne*,⁹⁴ where a molecular neurobiologist testified about the role of serotonin in impulsive violence, and had tested the defendant’s serotonin levels as compared to a control group, and found them very low, the evidence appeared to be well supported.⁹⁵ It would probably have met *Daubert* standards, had there been an inquiry. And it had at least some effect on jury deliberations, since they declined to convict on premeditation. That such well supported testimony is rare is all the more reason for courts to demand more exacting standards from the scientists that testify before them.

A. *How Valid is the Science?*

There has been considerable research on the biology of violence, and the complex way environmental stimuli affect gene expression. Despite strong evidence that the

90. See *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 141 (1999) (extending the validity inquiry to technical as well as scientific evidence); *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 142 (1997) (citing *Daubert*, 509 U.S. at 589); *Daubert*, 509 U.S. at 579 (requiring judicial gatekeeping for scientific validity).

91. See e.g. *Hall*, 160 S.W.3d at 33 (school psychologist testified that the defendant’s physical appearance and other characteristics indicated a genetic disorder “such as XXY, Klinefelter Syndrome, YYX, Extra Y Chromosome; or Fragile X Syndrome”).

92. *Daubert*, 509 U.S. at 590. Even in state courts that have eschewed the *Daubert* standard in favor of the old general acceptance rule, and in proceedings where the rules of evidence do not apply, there is an increased concern with scientific validity. See e.g. *Lee v. Health & Hum. Servs.*, 2005 WL 1125672 (Fed. Cl. Apr. 8, 2005) (finding testimony of genetic predisposition to fibromyalgia admissible under *Daubert*, although acknowledging that the Federal Rules of Evidence do not apply to proceedings under the National Vaccine Compensation Program).

93. *Landrigan*, 441 F.3d at 645.

94. 2002 WL 31624813.

95. *Id.* at *13.

cycle of violence is repeated across generations,⁹⁶ it is highly unlikely that anyone will discover a “crime gene.” The interaction between genes and the environment is too complex for such a simplistic view. That does not mean, however, that biology is irrelevant in assessing criminality.⁹⁷

Scandinavian twin and adoption studies are often cited as favoring a genetic role in crime.⁹⁸ Studies of petty criminals in Sweden and Denmark, for example, observed that the biological parents of petty criminals—but not the adoptive parents—had increased rates of criminality over the population base rate.⁹⁹ Notably, these studies did not examine violent crime specifically, but included any criminal infractions as an outcome.¹⁰⁰ None of the adoption studies have reported an association between violent convictions and parental background, although one study showed that it was more likely in identical (monozygotic) twins than in fraternal (dizygotic) twins, for both twins to have violent convictions.¹⁰¹

XXX chromosome abnormality studies, once cited as evidence of “crime genes,” have been shown at most to have an indirect link to (property, but not violent) crime through mental retardation.¹⁰² The MAOA studies are more promising. In these, alleles of specific genes have been identified and linked with propensities to violence.¹⁰³

96. See e.g. Natl. Inst. of Just., *The Cycle of Violence Revisited*, <http://www.ncjrs.org/pdffiles/cyclepre.pdf> (Feb. 1996) (studying 1,575 subjects over a 26-year period, and concluding that abused and neglected children were twice as likely to be arrested as juveniles as children without such a history, and more likely to be arrested for a violent offense).

97. See Debra Niehoff, *The Biology of Violence: How Understanding the Brain, Behavior, and Environment Can Break the Vicious Circle of Aggression* 238 (Free Press 1999). For a critical perspective, see Kenneth A. Taylor, *On the Explanatory Limits of Behavioral Genetics*, in *Genetics and Criminal Behavior*, *supra* n. 13, at 117–39.

98. See e.g. Terrie E. Moffitt & Avshalom Caspi, *Evidence from Behavioral Genetics for Environmental Contributions to Antisocial Conduct*, in *Crime and Schizophrenia: Causes and Cures* 45, 47 (Adrian Raine ed., Nova Sci. 2006) (noting that genes influence 40–50% of the variation in antisocial behavior, and citing twin and adoption studies); Niehoff, *supra* n. 96, at 238 (noting that “twin and adoption data favor a role for genetic influences” in crime but cautioning that “when concordance rates for violent crimes were extracted from the Scandinavian data, none of the studies made a very convincing case for an appreciable genetic influence on violence”). Cf. Matt Ridley, *The Agile Gene: How Nature Turns on Nature* 83 (Harper Collins 2003) (noting that twin studies demonstrate that “personality is about as heritable as body weight”).

99. Niehoff, *supra* n. 96, at 238. The theory underlying these studies was that if the environment was the important variable rather than inheritance, the rate should have been higher with both sets of parents.

100. *Id.*

101. C. R. Cloninger & I. I. Gottesman, *Genetic and Environmental Factors in Antisocial Behavior Disorders*, in *The Causes of Crime: New Biological Approaches* 92, 96–101 (S. A. Mednick et al. eds., Cambridge U. Press 1987).

102. See Owen D. Jones, *Behavioral Genetics and Crime*, in *Context*, 69 L. & Contemp. Probs. 81, 91 n. 36, 91–92 (2006) (noting that the XXX abnormality has at most an indirect link to these individuals’ increased risk of arrest and conviction).

103. MAOA regulates neurotransmitters such as serotonin, dopamine, and epinephrine, and has been associated with psychopathy, childhood hyperactivity, childhood aggression, impulsivity, and substance abuse. See Grant T. Harris et al., *The Construct of Psychopathy*, 28 Crime & Just. 197, 224 (2001) (acknowledging that “findings on all of these laboratory-based theories of psychopathy often seem somewhat ephemeral”). See also Avshalom Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 Sci. 851, 851–53 (2002) (studying 442 men in New Zealand for differences in MAOA activity alleles and correlating these differences with maltreatment in childhood and subsequent violent behavior). The results demonstrated that the high activity form of the gene did not manifest in violent propensities even if the men had been mistreated as boys, while those with the low-active form of the gene, who had been mistreated, committed four times as many rapes, assaults, and robberies as the average. *Id.*

1. The Role of Neurotransmitters in Violence

Violence most certainly has biochemical correlates. Abnormal neurotransmitter levels, serotonin in particular, long have been associated with violent crime.¹⁰⁴ Serotonin is a neurotransmitter that regulates inhibitory mechanisms of the central nervous system, and is thought to mediate conditions ranging from depression to impulsive violence.¹⁰⁵ Specific genes that appear to regulate neurotransmitter activity have been identified, as has the gene for tryptophan, a precursor of serotonin.¹⁰⁶ Monoamine oxidase A and B are enzymes that metabolize these neurotransmitters, and abnormal levels of these enzymes have been linked to risky and impulsive behaviors.¹⁰⁷ However, there is some evidence that serotonin levels affect mental function generally, so that rather than a propensity for violence, affected people simply lack constructive outlets for aggression, and are less adept at concealing it.¹⁰⁸

2. Stress Responses

People have different levels of stress response, and while some of this difference may be due to genetic differences among individuals, the differences may also be environmentally determined, may be due to traumatic brain injury, or may be a combination. Moreover, gene expression may be “turned on” or “off” by life events. For example, a gene called 5-HTT is associated with depression, suicide, and aggression.¹⁰⁹ But unless people carrying the predisposing allele are exposed to life events that they perceive as stressful, they are no more likely to become depressed than people with the healthy allele.

Environmental factors play a role in altering gene expression. For example, the expression of a gene in rats that makes them fearful and jumpy can be altered by how regularly the mother licks and grooms the pups.¹¹⁰ If mom licks and grooms frequently, the “jumpy” gene expression is changed so that the rat grows up to be calm and curious. This and other examples of developmental “plasticity” show that a given genotype can develop in different ways depending on the environment. Moreover, any neural deficits (developmental or traumatic) seem to become more pronounced when infants are deprived of care or nutrition. It may well be that “neuropsychological impairments

104. See Graham A. Rogeness et al., *Neurochemistry and Child and Adolescent Psychiatry*, 31 J. Am. Acad. Child & Adolescent Psych. 765, 775–77 (1992).

105. Wasserman & Wachbroit, *supra* n. 13, at 10 (discussing the Brunner 1993 study of MAOA).

106. See Richard A. Glennon & Malgorzata Dukat, *Serotonin Receptor Subtypes*, in *Psychopharmacology: The Fourth Generation of Progress* 415, 421 (Floyd E. Bloom & David J. Kupfer eds., Raven Press 1995) (discussing the role of serotonin and the serotonergic genes); Baker, *supra* n. 45, at 34 (noting that several genes associated with neurotransmitters have been identified in quantitative trait loci studies); Frederic Sandou et al., *Enhanced Aggressive Behavior in Mice Lacking 5-HT1B Receptor*, 265 Sci. 1875, 1875–78 (1994) (observing aggressive behavior in mice).

107. J. C. Shih et al., *Monoamine Oxidase: From Genes to Behavior*, 22 Annual Rev. Neuroscience 197, 209–10 (1999).

108. See Evan Balaban et al., *Mean Genes and the Biology of Aggression: A Critical Review of Recent Animal and Human Research*, 11 J. Neurogenetics 1, 18 (1996).

109. See Avshalom Caspi, *Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene*, 301 Sci. 386, 386 (2003).

110. E.W. Fish et al., *Epigenetic Programming of Stress Responses Through Variations in Maternal Care*, 1036 Annals N.Y. Acad. Sci. 167, 167 (2004).

disrupt normal development and increase vulnerability" to poor social environments.¹¹¹ That is, the environment can elicit markedly different traits from the same DNA.

Depression, violent aggression, and antisocial personality disorder have all been linked to problems with the stress response.¹¹² And while some violent criminals have lowered central nervous system (CNS) and autonomic nervous system (ANS) arousal, others have heightened arousal.¹¹³ Brain, body, genes, hormones, and hormone receptors are intimately interconnected and responsive to environmental conditions. Although each of these disorders has a different pattern of expression, all are associated with abnormal endocrine feedback, norepinephrine and serotonin functions, and altered glucocorticoid levels.¹¹⁴ This suggests that inappropriately violent behavior may sometimes involve a stress response disorder. People who appear to lack a conscience may actually lack the biological machinery necessary to warn them that they are heading for disaster.¹¹⁵ Studies of recidivistic violent offenders, adults with antisocial personality disorder, and antisocial adolescents have all documented statistically significant reductions in levels of cortisol, which is the main circulating stress hormone.¹¹⁶ Testosterone levels also appear to sensitize males to the social environment, affecting levels of aggression.¹¹⁷

111. James C. Howell & J. David Hawkins, *Prevention of Youth Violence*, 24 Crime & Just. 263, 268 (1998) (citing Terrie E. Moffitt, *Adolescence-Limited and Life-Course-Persistent Antisocial Behavior: A Developmental Taxonomy*, 100 Psychol. Rev. 674, 674-701 (1993)).

112. Niehoff, *supra* n. 96, at 183.

113. *Id.* at 181. The normal human CNS displays immediate, short-term, instinctive reflexive activity as a first line of defense to real or perceived threats. Measures of antisocial behavior in fifteen year-old males have been correlated with reduced autonomic nervous system activation. See Adrian Raine et al., *Autonomic Nervous System Factors Underlying Disinhibited, Antisocial, and Violent Behavior: Biosocial Perspectives and Treatment Implications*, 794 Annals N.Y. Acad. Sci. 46, 48 (1996) (reviewing nine-year prospective study of crime development and noting that it is the "first study providing evidence for underarousal in an antisocial population in all three psychophysiological response systems"). Further studies showed that measures of underarousal of the CNS and ANS taken at fifteen years of age were related to criminality status assessed at twenty-four years of age. Adrian Raine et al., *Relationships between Central and Autonomic Measures of Arousal at Age 15 Years and Criminality at Age 24 Years*, 47 Archives Gen. Psych. 1003, 1003 (1990). Lowered levels of arousal were accompanied by decreased activation of the reticular activating system (RAS), which is the part of the brain that controls sleep/wake cycles and arousal, and lowered hypothalamic-modulated stress responses. See *id.* Generally speaking, the hypothalamus, along with the RAS, helps regulate the body's physiological response to stress, often referred to as "fight or flight." Robert M. Sapolsky, *Stress, the Aging Brain, and the Mechanisms of Neuron Death* 3-9 (MIT Press 1992). For a more detailed discussion of the stress response, see Edgar Garcia-Rill & Erica Beecher-Monas, *Gatekeeping Stress: The Science and Admissibility of Post-Traumatic Stress Disorder*, 24 UALR L. Rev. 9, 12-14 (2001) (noting that in order to initiate "fight-or-flight" responses, the RAS projects into many regions, simultaneously alerting the cortex to the event, priming the motor system to be able to fight or flee, and to the hypothalamus, where the arousal response triggers the stress response).

114. *Id.*

115. See Niehoff, *supra* n. 96, at 181 (stating that "[n]o warning bell of anxiety or disgust sounds when . . . [antisocial individuals are] about to commit an atrocity").

116. *Id.* For additional information on the studies cited by Niehoff, see also Bo Bergman & Bo Brismar, *Hormone Levels and Personality Traits in Abusive and Suicidal Male Alcoholics*, 18 Alcoholism: Clinical & Experimental Research 311, 311-15 (1994); Keith McBurnett et al., *Anxiety, Inhibition, and Conduct Disorder in Children: II. Relation to Salivary Cortisol*, 30 J. Am. Acad. Child & Adolescent Psych. 192 (1991); Matti Virkkunen et al., *CSF Biochemistries, Glucose Metabolism, and Diurnal Activity Rhythms in Alcoholic, Violent Offenders, Fire Setters, and Healthy Volunteers*, 51 Archives Gen. Psych. 20, 20-27 (1994); Matti Virkkunen, *Urinary Free Cortisol Secretion in Habitually Violent Offenders*, 72 Acta Psychiatrica Scandinavica 40, 40 (1985).

117. See Frederick S. vom Saal, *Models of Early Hormonal Effects on Intrasex Aggression in Mice, in Hormones and Aggressive Behavior* 197, 198 (Bruce B. Svare ed., Plenum Press 1983) (observing that even

The cerebral cortex also plays a role in mediating stress responses, controlling, through inhibition, impulses from the older, primordial parts of the brain.¹¹⁸ Humans have much greater amounts of cerebral cortex than primates and apes, which allow us to respond more selectively to our environment. All primates exhibit violent aggression in response to threat, including human infants as young as eighteen months.¹¹⁹ Most children learn to decrease the use of physical aggression over time as they learn more successful social strategies.¹²⁰ Some children learn better than others, and some social environments are more conducive to learning alternative strategies.¹²¹

Moreover, if the cortex is damaged, or suffers from decreased blood flow, or metabolism, known as "hypofrontality," the cortex loses some of its inhibitory power, releasing "primordial" behaviors.¹²² Damage, decreased uptake of glucose, and reduced blood flow or reduced function, have all been observed in the frontal cortex of violent individuals and murderers.¹²³ Instinctive behaviors, including exaggerated "fight or flight" responses to misperceived threats, may result in violent or exaggerated behavior in an attempt to attack or flee. Hypofrontality is evident in such disorders as schizophrenia, posttraumatic stress disorder, and depression.¹²⁴

Events or conditions placing people with these disorders under undue stress could easily lead to exaggerated responses, such as striking out due to over-perceived threats. Is this mens rea or mitigation evidence? Quite possibly, and qualified experts should be permitted to explain these factors to the jury.

3. Sex Hormones

Testosterone plays a major role in sexuality and aggression.¹²⁵ Although most violence is perpetrated by young men against other young men struggling over dominance for reproductive resources, violent tendencies can develop at any time: In utero,¹²⁶ in early infancy,¹²⁷ or even after the onset of puberty.¹²⁸ Although male

intauterine positioning between sibling males and females affects levels of post-birth aggression); Renee J. Primus & Carol K. Kellogg, *Gonadal Hormones During Puberty Organize Environment-Related Social Interaction in the Male Rat*, 24 *Hormones & Behavior* 311, 320 (1990) ("The presence of testosterone [in rats] . . . appears to be critical for the expression of environment-related changes [in social interaction].").

118. See generally Frank N. Dempster, *The Rise and Fall of the Inhibitory Mechanism: Toward a Unified Theory of Cognitive Development and Aging*, 12 *Dev. Rev.* 45 (1992).

119. See Richard E. Tremblay, *Prevention of Chronic Physical Aggression: An Epigenetic Perspective*, in *Crime and Schizophrenia*, supra n. 97, at 267, 274.

120. *Id.* at 274–75.

121. *Id.* at 281.

122. See e.g. Erica Beecher-Monas & Edgar Garcia Rill, *Genetic Predictions of Future Dangerousness: Is There a Blueprint for Violence?* 69 *L. & Contemp. Probs.* 301 (2006).

123. Raine et al., *supra* n. 112; see also Hugo D. Critchley et al., *Prefrontal and Medial Temporal Correlates of Repetitive Violence to Self and Others*, 47 *Biological Psych.* 928, 931–33 (2000); J. Grafman et al., *Frontal Lobe Injuries, Violence, and Aggression: A Report of the Vietnam Head Injury Study*, 46 *Am. Acad. Neurology* 1231, 1231 (1996).

124. See e.g. Edgar Garcia-Rill, *Disorders of the Reticular Activating System*, 49 *Med. Hypotheses* 379, 381–82 (1997) (reviewing study that showed hypofrontality in schizophrenic patients).

125. See Florence Thibaut, *Perspectives on Treatment Interventions in Paraphilias*, in *Handbook of Medical Psychiatry* 909–18 (J.C. Soares & S. Gershon eds., Marcel Dekker 2003) (reviewing literature).

126. See e.g. Constance Holden, *The Violence of the Lambs*, 289 *Sci.* 580 (2000) (noting that "[r]esearchers are increasingly coming to view violence as the end result of multiple risk factors that may include a biological vulnerability—either genetic or created in the prenatal environment—that can be brought out or reinforced by

mammals have higher levels of physical violence than females, and although the violence follows the testosterone curve, its role remains unclear.¹²⁹ A number of studies link high testosterone levels in adults with violent behavior.¹³⁰ In childhood aggression, however, low testosterone levels appear to be linked to violent aggression.¹³¹ Testosterone levels are at least partly heritable.

Although pedophilia is not well studied, some researchers believe that neurological deficits, chromosome aberration, or early childhood abuse or sexual experience may imprint desire onto inappropriate outlets.¹³² Some potentially important anomalies have been observed in pedophiles. Right temporal lobe hypometabolism has been identified in adult pedophiles¹³³ as have lower baseline cortisol levels,¹³⁴ and monoaminergic dysfunction.¹³⁵ The best available evidence suggests some disruption in the development of the neurosystem.¹³⁶ While exposure to physical violence in childhood predisposes adults to violence, it is unclear whether exposure to sexual abuse in childhood has the same effect on sexual offending.¹³⁷ Approximately thirty percent of all adult sex offenders were sexually abused as children.

While both rape and child molestation are categorized as sexual violence under most sexual offending statutes, rape appears to be more of an aggressive and dominance strategy—a weapon, rather than an appetite.¹³⁸ Testosterone blocking drugs appear to

the social environment”).

127. Frans B. M. de Waal, *Primates—A Natural Heritage of Conflict Resolution*, 289 Sci. 586, 588 (2000) (discussing the violence between two infant monkeys after displaying aggression and biting each other).

128. E.g. Niehoff, *supra* n. 96, at 159.

129. See Thibaut, *supra* n. 124, at 910.

130. See e.g. J.A. Harris, *Review and Methodological Considerations in Research on Testosterone and Aggression*, 4 *Aggression & Violent Behavior* 273, 273–91 (1999) (reviewing studies).

131. See generally Richard E. Tremblay et al., *Testosterone, Physical Aggression, Dominance, and Physical Development in Early Adolescence*, 11 *Intl. J. Behavioral Dev.* 753 (1998).

132. See F. S. Berlin et al., *A Five-Year Plus Follow-Up Survey of Criminal Recidivism within a Treated Cohort of 406 Pedophiles*, 12 *Am. J. Forensic Psychol.* 5 (1991); Fred S. Berlin & Edgar Kraut, *Pedophilia Diagnostic Concepts, Treatment and Ethical Considerations*, 7 *Am. J. Forensic Psychol.* 13 (1986).

133. See generally Mario F. Mendez et al., *Pedophilia and Temporal Lobe Disturbances*, 12 *J. Neuropsychiatry & Clinical Neuroscience* 71, 71–76 (2000) (suggesting that “[a] predisposition to pedophilia may be unmasked by hypersexuality from brain disease”).

134. See generally Michael Maes et al., *Lower Baseline Plasma Cortisol and Prolactin Together with Increased Body Temperature and Higher mCPP-Induced Cortisol Responses in Men with Pedophilia*, 24 *Neuropsychopharmacology* 37, 37–44 (2001) (studying hormonal and serotonergic alterations in pedophiles).

135. See Martin P. Kafka, *The Monoamine Hypothesis for the Pathophysiology of Paraphilic Disorders: An Update*, 989 *Annals N.Y. Acad. Sci.* 86, 91 (2003) (reviewing data on the role of monoamine neuromodulators in paraphiliacs and concluding that “a specific role for these neuromodulators in the paraphilic condition remains neither proven nor rejected”).

136. F.S. Berlin & E. Krout, *Pedophilia: Diagnosis Concepts, Treatment, and Ethical Considerations*, 7 *Am. J. Forensic Psych.* 13, 20 (1986) (observing that “the best available evidence suggests that most sexual deviance and paraphilia is caused by very early biomedical events leading to neurodevelopmental disruption”).

137. Compare e.g. John Monahan et al., *Rethinking Risk Assessment: The MacArthur Study of Mental Disorder and Violence* 54–55 (Oxford U. Press 2001) (noting that “[a]lthough prior physical abuse as a child . . . [is] associated with postdischarge violence, prior sexual abuse was not”) with Ctr. Sex Offender Mgt., *Understanding Juvenile Sexual Offending Behavior: Emerging Research, Treatment Approaches and Management Practices*, <http://www.csom.org/pubs/juvbrf10.html> (Dec. 1999) (reporting that twenty to fifty percent of sexually abusive youth had a history of physical abuse, while forty to eighty percent had a history of sexual abuse).

138. See Niehoff, *supra* n. 96, at 164 (claiming rape is motivated by aggression); Grant T. Harris et al., *Appraisal and Management of Risk in Sexual Aggressors: Implications for Criminal Justice Policy*, 4 *Psychol. Pub. Policy & L.* 73, 85 (1998) (“[A]lthough rapists are more dangerous [than child molesters] (i.e., more likely

chill sexual behavior but not anti-social aggression.¹³⁹ There is no evidence that sex offenders have either increased androgen levels or receptor activity.¹⁴⁰

B. *Do Genes Determine Behavior?*

None of this evidence suggests that genes determine behavior. Behavior (including violent aggression and sexual deviance) results from interacting factors including genes, social circumstances, economic, cultural, and developmental factors.¹⁴¹ But it works the other way around as well: Brains and behavior also switch genes on and off.¹⁴² Brain diseases also affect behavior, and some diseases may be the result of errant genes, while others are the result of developmental problems or environmental insults.¹⁴³

When there is evidence of such a genetic anomaly, it is information important to understanding the behavior of an individual. To the extent that the intent behind an individual's actions is relevant (that is, in a case not involving strict criminal liability), if such information is available, and the expert can demonstrate its scientific basis, it should be presented to the fact finder. If the circumstances under which a person acted are relevant to a jury's moral deliberations in deciding about whether to impose the death penalty, and if those circumstances include genetic influences, then, as long as the expert can demonstrate a scientific basis, that information should also be before the jury. Similarly, if there is a genetic correlate for mental retardation, schizophrenia,¹⁴⁴ or other mental disease, that would be useful information in determining whether the defendant was malingering.

In terms of prevention, any attempt to prevent violent behavior will implicate many forms of science, including knowledge about ways in which altering specific factors can alter the behavioral outcome. Genetic traits cannot be changed (at least not easily), but the environment, or the underlying mechanisms of response to environmental stimuli, can be.¹⁴⁵ The monoamine oxidase A gene is a case in point. People with the genetic anomaly (which is a recessive gene located on the X chromosome, so primarily men—who have only one X chromosome—are affected) have a higher incidence of

to exhibit violent recidivism) overall, much of that violence does not appear to be sexually motivated.”).

139. Niehoff, *supra* n. 96, at 164; *but see* Raine et al., *supra* n. 112, at 94 (citing studies showing that “administration of anti-androgens and progesterone derivatives have some effect in lowering violence and sexual aggression”).

140. See Anu S. Aromaki et al., *Testosterone, Sexuality and Antisocial Personality in Rapists and Child Molesters: A Pilot Study*, 110 *Psych. Research* 239, 239–47 (2002).

141. See Robert Wachtbroit, *Understanding the Genetics-of-Violence Controversy*, in *Genetics & Criminal Behavior* 25, 33 (David Wasserman & Robert Wachtbroit eds., Cambridge U. Press 2001) (discussing the fallacies of reductionism).

142. See Matt Ridley, *Genome: The Autobiography of a Species in 23 Chapters* 152–56 (Harper Collins 1999) (noting that “it is the body that switches on genes when it needs them, often in response to a more or less cerebral or even conscious, reaction to external events” and giving as examples the increase in cortisol levels of a young male baboon joining a new group and establishing its rank in the social pecking order; and experiments showing an increase in stress hormones with a loss of control in British civil servants).

143. See Dean Mobbs et al., *Law, Responsibility, and the Brain*, 5 *PLoS Biology* 693 (Apr. 2007) (available at <http://ssrn.com/abstract=982487>) (noting that brain diseases reduce the ability to act freely).

144. A recent study demonstrated genetic links to schizophrenia. See e.g. Benedict Carey, *Study Ties Genetic Variations to Schizophrenia*, www.nytimes.com/2008/03/28/science/28gene.html (Mar. 28, 2008).

145. See Baker et al., *supra* n. 45, at 34 (explaining that modifying the environment can change patterns of response).

violent behavior, but only if they were abused as children.¹⁴⁶ Theoretically, one could alter the potential for behavioral problems in a child afflicted with this anomaly by adding monamine oxidase A to the child's system, but one could also change it by preventing parental abuse. An example of this kind of interaction is the huge success of neonatal screening for phenylketonalanine (PKU), a genetic anomaly that results in mental retardation. PKU testing of newborn children is now routine hospital procedure. Simply modifying the infant's diet (by omitting foods containing phenylalanine) "cures" the condition, preventing the gene's expression.

This is not only true for the nervous system. All traits, from biological traits like hair and height to complex psychological traits like intelligence, are caused by interdependent interactions of genes, development, and the environment. Genetic factors may instigate the process, but at every point in the building and tuning process of creating and maintaining an individual, the environment plays a hand.

C. *Let the Jury Decide the Weight*

Once the judge has decided on the relevance and reliability of the scientific testimony, it ought to go to the jury. Yet, the question of whether evidence of genetic influences helps or hurts defendants is one with which the courts continue to struggle. In *Schriro v. Landrigan*,¹⁴⁷ Justice Thomas, writing for the majority, opined that "assuring the court that genetics made [Landrigan] the way he is could not have been very helpful," and "the mitigating evidence he seeks to introduce would not have changed the result."¹⁴⁸ Although a number of pre-*Landrigan* cases had upheld ineffective assistance claims for failing to present genetic evidence,¹⁴⁹ the more prevalent result has been the refusal to find ineffective assistance on that basis, generally because the court felt that the unrepresented testimony would not have made a difference in the outcome.¹⁵⁰

146. See Avshalom Caspi et al., *supra* n. 102, at 851–53 (studying 442 men in New Zealand for differences in MAOA activity alleles and correlating these differences with maltreatment in childhood and subsequent violent behavior). The results demonstrated that the high activity form of the gene did not manifest in violent propensities even if the men had been mistreated as boys, while those with the low-active form of the gene, who had been mistreated, committed four times as many rapes, assaults, and robberies as the average. *Id.* A second study by the Caspi group in 2003 also reported on a gene-environment interaction, this time in the promoter region of the serotonin transporter gene. Caspi, *supra* n. 108, at 386–89 (reporting on the interaction between the short allele of the serotonin transporter gene and stressful environment).

147. 127 S. Ct. 1933.

148. *Id.* at 1944. This echoes the reservations of the dissent in the Ninth Circuit, which thought that "the mitigating value of any proven genetic predisposition for violence would not have outweighed its aggravating tendency to suggest Landrigan was undeterrable and, even from prison, would present a future danger to society." *Landrigan*, 441 F.3d at 651 (Bea, J., dissenting).

149. See e.g. *Hendricks v. Calderon*, 864 F. Supp. 929, 947 (N.D. Cal. 1994), *cert. denied*, 517 U.S. 1111 (granting habeas relief); *Fudge v. State*, 120 S.W.3d 600, 602–03 (Ark. 2003) (remanding based on ineffective assistance claims because counsel had failed to present evidence of defendant's genetic or learned propensity for violence).

150. *Johnson v. Quartermain*, 483 F.3d 278, 288 (5th Cir. 2007) (denying certificate of appealability for ineffective assistance despite affidavit from mitigation specialist regarding unrepresented evidence of genetic predisposition to substance abuse, mental illness, and childhood abuse because "even if considered, the affidavit would provide no grounds for relief because in the context of [defendant's] extensive history of extreme and brutal violence" it would make little difference); *Cauthern*, 145 S.W.3d at 578, 628–29 (failure to present evidence of genetic predisposition to impulsive behavior did not amount to ineffective assistance because such testimony would have made no difference in the outcome); *U.S. v. Curtis*, 44 Mil. J. 106 (Armed Forces App. 1996) (no ineffective assistance for failure to present evidence of genetic predisposition to

This is a spurious quandary, because whether the testimony of a genetic predisposition to violence would help or hurt the defendant's case is a quintessential jury question. A primary function of the jury is to bring the voice of the community to bear on conflicting and contradictory evidence, and to draw inferences from the evidence to determine the probable course of events. The jury is composed of individuals with a variety of backgrounds and experiences, is independent of the government, and is a one-time actor in the justice system.

One of the key features of justice—at least in the United States—is the jury.¹⁵¹ The jury system provides a structure for citizen participation and brings the voice of the community into the process of legal decision-making.¹⁵² It is integral to the separation of powers doctrine.¹⁵³ In giving citizens the right and obligation to participate in the judicial system, the Framers believed that they were implementing democracy by interposing “the commonsense judgment of a group of laymen” between the accuser and the accused.¹⁵⁴ The jury was expected to bring community values to what might otherwise become arbitrary decision-making.¹⁵⁵

The jury's role as fact finder is thus supplemented and informed by its role as the moral voice of the community. It is precisely in making difficult factual and moral decisions that the jury is most valuable. Pre-empting the presentation of expert testimony regarding genetic influences or other biological information that may bear on either the guilt or sentencing phase of the proceedings ought to be based on something more than judicial instinct. In order to keep relevant and reliable information from the jury, courts should at least have a principled basis, rather than the current ad-hoc approach.

V. CONCLUSION

Whether the evidence of genetic predisposition to violence (or sexual violence) has a sound scientific basis depends of course, on the proffered testimony. The courts appear to have been correct about the unreliability of XY testimony for proving a predisposition to violence. The MAOA link, however, appears to rest on a more solid foundation. In *Mobley*, where the defense sought genetic testing for MAOA deficiency, the scientific basis at the time was a single study of a Dutch family linking the genetic anomaly to behavioral problems.¹⁵⁶ The court thought that the theory of a MAOA

alcoholism).

151. Rita J. Simon, *The Jury: Its Role in American Society* 6–7 (Lexington Bks. 1980) (describing the role of popular participation in the administration of justice).

152. See Charles W. Wolfram, *The Constitutional History of the Seventh Amendment*, 57 Minn. L. Rev. 639, 653–56, 654 n. 47 (1973) (discussing the British circumvention of the colonists' right to trial by jury as a significant cause of the American Revolution).

153. See *id.* at 662–71 (discussing the jury as a popular check on the three branches of government).

154. See *Williams v. Fla.*, 399 U.S. 78, 100 (1978) (noting the “community participation and shared responsibility that results from the [jury's] determination of guilt or innocence”). For an argument that the decline of jury trials represents a significant erosion of democracy, see Jason Mazzone, *The Justice and the Jury*, 72 Brook. L. Rev. 35, 55–56 (2006) (discussing the jury jurisprudence of Justice Blackmun and noting that in federal court, juries resolve only about four percent of criminal cases).

155. See Bernard Schwartz, *The Bill of Rights: A Documentary History* vol. 1, 3–16 (Chelsea H. 1971) (discussing the function of the jury as a bulwark against tyranny).

156. *Mobley*, 455 S.E.2d at 65. The Brunner study was the basis for the defense request. See Hans G.

deficiency link to crime was premature,¹⁵⁷ and at the time, it may have been. But there are more sophisticated studies available now, and the courts will not be as justified in dismissing the science as premature.

There are several conclusions to be drawn from the use of genetic testimony in criminal trials. First, the scientific basis for such testimony is often extremely shaky. Such unfounded testimony ought to be excluded. Second, there is some evidence about genetic links to crime that is more firmly founded, such as MAOA, and if the defense can establish the scientific basis for the expert testimony, the defense should be able to present this evidence to the jury as a mitigating factor, along with all the typical background factors (like childhood abuse) that go to mitigation. Failure to explore this option should be considered ineffective assistance. Third, whether the jury actually finds genetic testimony mitigating or aggravating should be left to the jury. Excluding testimony about genetic predisposition based on the judge's assessment that it would be aggravating prevents the defendant from providing a full defense, and removes community assessment from an important problem.

Brunner et al., *Abnormal Behavior Associated with a Point Mutation in the Structural Gene for Monoamine Oxidase A*, 262 Sci. 578 (1993).

157. *Mobley*, 455 S.E.2d at 66.