

THE MISTAKEN EMPHASIS ON ORGANIC BRAIN DAMAGE IN  
CAPITAL HABEAS LITIGATION

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## INTRODUCTION

The increasing use of evidence about how the human brain works is sure to be one of the defining features of practicing law in the twenty-first century.<sup>1</sup> One area of litigation where the use of neuroscience evidence has already become fairly widespread is death penalty litigation, particularly federal habeas proceedings.<sup>2</sup> Habeas petitioners now frequently claim that they have diminished culpability because they suffer from “organic brain damage.”<sup>3</sup> Yet the petitioners raising such claims, and the courts resolving them, have not been particularly precise in defining exactly what “organic brain damage” means.<sup>4</sup> A review of the medical literature, however, reveals that this lack of precision is understandable. The truth is that “organic brain damage” is not a specific, recognized condition. Instead, it is an umbrella term that habeas petitioners use to cover a broad range of brain pathologies. The continued uninformed use of the term leads to its imprecise use in the sentencing context.

This Note proceeds in three parts. First, it offers an explanation for why evidence of organic brain damage has become so popular in capital litigation and identifies how litigants and courts have used the term without recognizing its imprecise nature.<sup>5</sup> Second, it reviews the relevant neuroscience literature to demonstrate that neuroscience evidence still presents serious unresolved shortcomings in the courtroom context.<sup>6</sup> Finally, it argues that there is only a tenuous relationship between brain function assessments and criminal responsibility.<sup>7</sup>

## I. THE GROWING IMPORTANCE OF BRAIN EVIDENCE AND THE LACK OF CLARITY ABOUT WHAT “ORGANIC BRAIN DAMAGE” MEANS

Evidence of organic brain damage has become incredibly important in capital cases.<sup>8</sup> There are two general explanations for why that is the case. First, the emphasis on aggravating and mitigating evidence has created a

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1. See generally Stephen J. Morse, Session 1: Neuroscience, Brain, and Behavior VI: Neuroscience and the Law, THE PRESIDENT’S COUNCIL ON BIOETHICS (Sept. 9, 2004), <https://bioethicsarchive.georgetown.edu/pcbe/transcripts/sep04/session1.html>; Jeffrey Rosen, *The Brain on the Stand*, N.Y. TIMES (Mar. 11, 2007) <http://www.nytimes.com/2007/03/11/magazine/11Neurolaw.t.html>.

2. See *infra* Part I.C.

3. See *id.*

4. See *id.*

5. See *infra* Part I.

6. See *infra* Part II.

7. See *infra* Part III.

8. See Rosen, *supra* note 1 (statement of Daniel Martell) (“Some sort of organic brain defense has become de rigueur in any sort of capital defense . . .”).

system of incentives that encourages capital defendants—both at trial and in habeas proceedings—to seek evidence that they have brain abnormalities that contributed to their crime. Second, the two leading Supreme Court cases to reverse death sentences based on ineffective assistance of counsel have emphasized counsel’s failure to find and present evidence in the mitigation phase that the defendant had diminished mental functioning. As a result, habeas petitioners are increasingly presenting evidence that they suffer from organic brain damage. Yet petitioners presenting such claims, and the courts reviewing them, often do not demonstrate a nuanced understanding of what the term “organic brain damage” means in a particular petitioner’s case.

#### A. *Aggravating and Mitigating Factors*

The first factor incentivizing habeas petitioners to obtain and present evidence that they suffer from brain abnormalities is the impact such evidence has on the balance between the aggravating and mitigating circumstances that determine the appropriateness of a death sentence. The Supreme Court’s death penalty jurisprudence in the late twentieth and early twenty-first centuries has limited capital punishment “to those offenders who commit a narrow category of the most serious crimes and whose extreme culpability makes them the most deserving of execution.”<sup>9</sup> In 1972, the Court effectively placed a moratorium on the death penalty<sup>10</sup> in *Furman v. Georgia*.<sup>11</sup> It ended that moratorium four years later when it decided *Gregg v. Georgia*, which held that the Constitution permits capital punishment so long as “the sentencing authority is apprised of the information relevant to the imposition of sentence and provided with standards to guide its use of the information.”<sup>12</sup>

In the wake of *Gregg*, legislators and the courts have set about defining a number of aggravating circumstances and mitigating factors for juries and judges to consider in determining whether a particular murder warrants a death sentence.<sup>13</sup> Aggravating factors are circumstances that “distinguish one particular killing as worse, and therefore, eligible for a sentence of

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9. Kennedy v. Louisiana, 554 U.S. 407, 420 (2008) (quoting Roper v. Simmons, 543 U.S. 551, 568 (2005) (internal quotation marks omitted)).

10. Janet C. Hoeffel, *The Jurisprudence of Death and Youth: Now the Twain Should Meet*, 46 TEX. TECH L. REV. 29, 63 (2013).

11. 408 U.S. 238, 239–40 (1972) (per curiam).

12. *Gregg v. Georgia*, 428 U.S. 153, 195 (1976) (opinion of Stewart, J.); see also *Eddings v. Oklahoma*, 455 U.S. 104, 111 (1982) (reading this principle as the holding of *Gregg*).

13. William J. Bowers & Benjamin D. Steiner, *Death by Default: An Empirical Demonstration of False and Forced Choices in Capital Sentencing*, 77 TEX. L. REV. 605, 613–21 (1999).

death, from the thousands of others each year that are not eligible.”<sup>14</sup> An aggravating factor may be used in imposing the death penalty so long as it (1) “genuinely narrow[s] the class of persons eligible for the death penalty” and (2) “reasonably justif[ies] the imposition of a more severe sentence on the defendant compared to others found guilty of murder.”<sup>15</sup> Examples include the commission of multiple murders, the killing of a law enforcement officer, and killing for financial gain.<sup>16</sup> Conversely, mitigating factors are circumstances that “provide[] reasons why the defendant should not be sentenced to death.”<sup>17</sup> They are not as narrowly defined as aggravating factors because the Eighth Amendment requires the jury or judge making the sentencing decision to consider “any aspect of a defendant’s character or record and any of the circumstances of the offense that the defendant proffers as a basis for a sentence less than death.”<sup>18</sup> Of course, this does not mean that defendants may present evidence that, as a matter of law, is completely irrelevant.<sup>19</sup> Examples of mitigating factors include lack of a prior criminal record, an abusive childhood, an underlying mental disorder, youth, and expressed remorse for the crime.<sup>20</sup>

The post-*Furman* approach to sentencing in death penalty cases has provided defendants with a greater incentive to present evidence that they suffer from some form of brain damage or impairment.<sup>21</sup> There are two general reasons why. First, the requirement that a death sentence cannot be imposed without aggravating factors incentivizes prosecutors, who do not want to lose face by seeking the death penalty and failing to secure it, to seek the death penalty only in those cases where the murder is especially deserving of one.<sup>22</sup> And though the aggravating-factor requirement does not eliminate all arbitrariness in the decision to seek the death penalty, there is empirical evidence suggesting that the greater the number of aggravating factors that fit a given crime, the greater the likelihood that prosecutors will

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14. LINDA E. CARTER, ELLEN S. KREITZBERG & SCOTT W. HOWE, UNDERSTANDING CAPITAL PUNISHMENT LAW 96 (2d ed. 2008).

15. *Zant v. Stephens*, 462 U.S. 862, 877 (1983).

16. CARTER, KREITZBERG, & HOWE, *supra* note 14, at 96.

17. *Id.* at 131.

18. *Skipper v. South Carolina*, 476 U.S. 1, 4 (1986) (quoting *Eddings v. Oklahoma*, 455 U.S. 104, 110 (1982) (internal quotation marks omitted)).

19. *See Lockett v. Ohio*, 438 U.S. 586, 605 n.12 (1978) (“Nothing in this opinion limits the traditional authority of a court to exclude, as irrelevant, evidence not bearing on the defendant’s character, prior record, or the circumstances of his offense.”); *e.g.*, *United States v. Brown*, 441 F.3d 1330, 1351–52 (11th Cir. 2006) (holding that testimony from the victim’s family members expressing their personal opinion *opposing* the imposition of the death penalty was immaterial).

20. CARTER, KREITZBERG, & HOWE, *supra* note 14, at 131.

21. The increasing availability and development of such evidence is obviously also an incentive. *See Rosen*, *supra* note 1.

22. *Cf.* CARTER, KREITZBERG, & HOWE, *supra* note 14, at 96 n.5 (explaining that the aggravating-factors requirement helps to separate the “worst of the worst” from run-of-the-mill murders).

seek the death penalty.<sup>23</sup> When the aggravating factors are strong, defendants have an incentive to counter with the strongest mitigating evidence available to them. And expert testimony that the defendant has a brain condition that mitigates his culpability is much more likely to sway the jury than other factors such as the defendant's lack of prior convictions or difficult childhood.<sup>24</sup>

Second, courts reviewing death sentences on appeal have acknowledged that several aggravating factors—those that speak to the heinous nature of the murder as well as to the cold and calculated manner of the killing—carry great weight in assessing the balance between aggravating and mitigating factors.<sup>25</sup> If the jury finds those aggravating factors when deciding on a death sentence, the inmate seeking to overturn his death sentence in federal habeas proceedings will need to be able to counter the weight those factors carry.<sup>26</sup> This gives habeas petitioners a strong incentive to try and show that the jury did not get a chance to hear mitigating evidence that directly counteracts those strong aggravating factors—namely, evidence that the petitioner suffered from some sort of brain abnormality.<sup>27</sup> As the next section will show, the Supreme Court's

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23. This conclusion is supported by a study conducted by the *Atlanta Journal-Constitution* analyzing murder cases in Georgia from 1995 through 2004. See *An AJC Special Report*, ATL. J. CONST., Sept. 23, 2007, <http://www.myajc.com/news/news/state-regional/matter-life-and-death-death-still-arbitrary/nk5kF/>. It found that, “[o]f the 132 murderers who made up the worst 10 percent of cases” as measured by Georgia’s ten statutory aggravating factors, “[p]rosecutors sought death in 103 of those cases.” *Id.* The rate at which prosecutors sought the death penalty in those cases (approximately 78%) was about three times higher than the rate at which prosecutors sought the death penalty in all death-eligible cases (approximately 25%). See *id.*

24. See Stephen P. Garvey, *Aggravation and Mitigation in Capital Cases: What Do Jurors Think?*, 98 COLUM. L. REV. 1538, 1555 (1998) (finding that the most effective mitigating factor in the eyes of jurors in death penalty cases was that the “killing was committed under influence of extreme mental or emotional disturbance”).

25. See, e.g., *Buzia v. State*, 926 So. 2d 1203, 1216 (Fla. 2006) (holding that Florida’s heinous-atrocious-or-cruel and the cold-calculated-and-premeditated-manner statutory aggravators “are ‘two of the most serious aggravators set out in the statutory sentencing scheme’” (quoting *Larkins v. State*, 739 So. 2d 90, 95 (Fla.1999))).

26. For example, a habeas petitioner claiming ineffective assistance of counsel in the penalty phase of his trial must show that, but for counsel’s mistakes, there is a reasonable probability that he would have received a different sentence. See, e.g., *Porter v. McCollum*, 558 U.S. 30, 40–41 (2009). The court assesses that probability by “consider[ing] ‘the totality of the available mitigation evidence—both that adduced at trial, and the evidence adduced in the habeas proceeding—and reweigh[ing] it against the evidence in aggravation.’” *Id.* at 41 (quoting *Williams v. Taylor*, 529 U.S. 362, 397–98 (2000) (internal quotation marks omitted)).

27. Of course, that attempt may not always be successful. See, e.g., *Lynch v. State*, 2 So. 3d 47, 77 (Fla. 2008) (holding that there was not a reasonable probability that the expert testimony from mental health experts presented at the defendant’s state post-conviction proceeding would have changed the outcome of the penalty phase of his capital trial, because none of the experts had explained how their various diagnoses could be squared with the facts that the defendant “thoroughly planned and carried out his memorialized intent to murder [the victim] and then demonstrated critical impulse control by refusing to commit suicide [which had been part of his original plan]”); see also *Smith v. Gibson*, 197 F.3d 454, 463 (10th Cir. 1999) (“[T]his court has, ‘on numerous occasions determined that . . . evidence of low I.Q. and/or organic brain damage’ ‘does not outweigh evidence supporting . . .

decisions overturning death sentences have encouraged habeas petitioners to claim that their brain abnormalities amount to “brain damage” or “organic brain damage.”

### B. *Ineffective Assistance of Counsel*

The second factor incentivizing habeas petitioners to bring claims based on newly-discovered evidence of organic brain damage is the fact that failure to present evidence of “brain damage” in the sentencing phase is the only ground that the Supreme Court has regularly recognized as a basis for a successful ineffective assistance of counsel claim. Such claims are ultimately grounded in the Sixth Amendment, which guarantees criminal defendants “the right . . . to have the Assistance of Counsel for [their] defence.”<sup>28</sup> The Supreme Court has given that right substantive content by holding that the right to counsel guarantees not only representation by an attorney, but also “the effective assistance of competent counsel.”<sup>29</sup>

The Supreme Court established the standard for ineffective assistance of counsel in *Strickland v. Washington*.<sup>30</sup> In denying the habeas petitioner’s claim, the *Strickland* Court established two principles that have been particularly important in litigation involving neuroscience evidence. First, the Court fashioned the now familiar two-part test for establishing ineffective assistance. Courts will not reverse a conviction or vacate a sentence unless (1) “counsel’s performance was deficient,” and (2) “the deficient performance prejudiced the defense.”<sup>31</sup> That standard applies to a federal habeas petitioner’s claim that he received ineffective assistance of counsel during his trial in state court.<sup>32</sup> Second, *Strickland* confirmed that trial counsel has a “duty to investigate” that requires a “thorough investigation of law and facts relevant to plausible options” for the defendant.<sup>33</sup> These two principles have combined to create extensive litigation, particularly in federal habeas proceedings, over trial counsel’s failure to find and present mitigating evidence of the defendant’s brain abnormalities (or mental-health problems).<sup>34</sup> Indeed, four Supreme Court

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multiple aggravating circumstances . . . .” (quoting *Foster v. Ward*, 182 F.3d 1177, 1189 (10th Cir. 1999))).

28. U.S. CONST. amend. VI.

29. *McMann v. Richardson*, 397 U.S. 759, 771 (1970).

30. 466 U.S. 668, 675–83 (1984).

31. *Id.* at 687.

32. *Id.* at 697.

33. *Id.* at 690.

34. Over the past thirty years, ineffective assistance of counsel claims have grown to become the most frequently raised form of claim in federal habeas proceedings. See Tom Zimpleman, *The Ineffective Assistance of Counsel Era*, 63 S.C. L. REV. 425, 433–39 (2011) (summarizing the empirical

cases since *Strickland* have held that trial counsel's failure to find evidence of the defendant's diminished mental capacity justified reversing the defendant's death sentence.

The first to do so was *Wiggins v. Smith*.<sup>35</sup> The Supreme Court held that the habeas petitioner had been denied effective assistance of counsel based on his trial attorneys' failure to: (1) find and present evidence of the "severe privation and abuse" Wiggins suffered as a child, which included "physical torment" and "sexual molestation"; and (2) present expert testimony explaining that Wiggins had "diminished mental capacities," which were characterized as borderline mental retardation.<sup>36</sup>

*Wiggins* created two footholds for habeas petitioners looking to challenge their death sentences based on ineffective assistance of counsel. Initially, it identified a basis for withholding the heavy deference given to trial counsel's "strategic choices."<sup>37</sup> That deference ordinarily precludes vacating a sentence based on trial counsel's failure to present certain mitigation evidence.<sup>38</sup> But the *Wiggins* Court explained that, where the claimed deficiency is counsel's failure to find and present certain mitigating evidence, courts owe no deference to counsel's decision unless counsel's failure to find that mitigating evidence was the result of a "reasoned strategic judgment" to limit the scope of counsel's investigation.<sup>39</sup> The *Wiggins* Court also cemented the power of brain evidence in assessing the prejudice to the defense, explaining that the unrepresented mitigation evidence was "powerful" and "relevant to assessing a defendant's moral culpability."<sup>40</sup>

The second case, *Rompilla v. Beard*, introduced the notion that "organic brain damage" is an important form of mitigation evidence.<sup>41</sup> The Court held that the habeas petitioner had been denied effective assistance of counsel based on his trial attorney's failure to find and present evidence that he: (1) "suffers from organic brain damage, an extreme mental

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data). And the uptick in federal court decisions addressing ineffective assistance of counsel claims based on a failure to find evidence of "organic brain damage" suggests that they have grown in popularity as well. See *infra* note 52.

35. 539 U.S. 510, 535–38 (2003).

36. *Id.* at 535.

37. See *Strickland v. Washington*, 466 U.S. 668, 690 (1984) (explaining that "strategic choices made after thorough investigation of law and facts relevant to plausible options are virtually unchallengeable").

38. See, e.g., *United States v. Luciano*, 158 F.3d 655, 660 (2d Cir. 1998) ("The decision not to call a particular witness is typically a question of trial strategy that appellate courts are ill-suited to second-guess.").

39. See 539 U.S. at 526.

40. *Id.* at 534–35.

41. 545 U.S. 374, 377–78 (2005); see also *Commonwealth v. Rompilla*, 983 A.2d 1207, 1208 (Pa. 2009).

disturbance significantly impairing several of his cognitive functions”;<sup>42</sup> (2) was neglected as a child by an alcoholic mother who “was missing from home frequently for a period of one or several weeks at a time”;<sup>43</sup> and (3) had an IQ “in the mentally retarded range.”<sup>44</sup> The Court therefore reversed the death sentence.<sup>45</sup>

Finally, two per curiam decisions by the Supreme Court have reaffirmed the impact that evidence of brain damage has when evaluating ineffective assistance of counsel claims. In *Porter v. McCollum*, the habeas petitioner was sentenced to death after he broke into the home of his ex-girlfriend and murdered her and her new boyfriend.<sup>46</sup> Though counsel had argued in the penalty phase that the petitioner “has other handicaps that weren’t apparent during the trial” and was not “mentally healthy,” he did not present any expert testimony about the petitioner’s mental health.<sup>47</sup> The Supreme Court unanimously held that the petitioner had been denied effective assistance of counsel based on his trial attorney’s failure to find and present mitigating evidence of “(1) Porter’s heroic military service in two of the most critical—and horrific—battles of the Korean War, (2) his struggles to regain normality upon his return from war, (3) his childhood history of physical abuse, and (4) his brain abnormality, difficulty reading and writing, and limited schooling.”<sup>48</sup> The Court therefore reversed his death sentence.<sup>49</sup> And in *Sears v. Upton*, the Court held that the habeas petitioner was denied effective assistance of counsel because his trial attorney failed to find and present mitigating evidence that he “performs at or below the bottom first percentile in several measures of cognitive functioning and reasoning” due to “frontal lobe brain damage Sears suffered as a child, as well as drug and alcohol abuse in his teens.”<sup>50</sup> Both *Porter* and *Sears* reinforce the lesson from *Wiggins* and *Rompilla*: Showing that trial counsel failed to find and present evidence that the defendant suffered from some kind of brain damage (or a similar brain abnormality) is a habeas petitioner’s best chance of reversing his death sentence.

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42. *Rompilla*, 545 U.S. at 392 (quoting *Rompilla v. Horn*, 355 F.3d 233, 244 (3d Cir. 2004) (Sloviter, J., dissenting)).

43. *Id.* at 393 (quoting Lodging to Application).

44. *Id.*

45. *Id.*

46. 558 U.S. 30, 31–33 (2009).

47. *Id.* at 32 (quoting Trial Transcript).

48. *Id.* at 41.

49. *Id.* at 31.

50. 561 U.S. 945, 945–46 (2010).



C. *The Use of Evidence of “Organic Brain Damage” in Capital Habeas Litigation*

The Supreme Court’s decisions have sent the signal that mental-health evidence—particularly evidence of brain damage—is strongly persuasive when arguing that trial counsel was ineffective.<sup>51</sup> And it appears that both habeas petitioners and the federal courts have heard the message. The number of federal habeas petitions raising claims of organic brain damage has more than doubled since the Supreme Court decided *Wiggins*.<sup>52</sup> And empirical evidence suggests that habeas petitions claiming that trial counsel failed to find and present mitigating evidence of organic brain damage or other mental health issues account for a large number of successful habeas petitions.<sup>53</sup>

While evidence of brain damage has increased in importance, there has not been a commensurate increase in the level of nuance with which litigators bring such claims or the courts handle them. For example, in a federal habeas appeal recently decided by the Eleventh Circuit, the experts who testified on behalf of the habeas petitioner in his state post-conviction proceeding provided at least three different diagnoses of the prisoner’s alleged neurological or psychological impairments.<sup>54</sup> Yet post-conviction counsel lumped all of the diagnoses together in arguing that the petitioner’s trial counsel had failed to present evidence of his “brain damage” or his “organic brain damages.”<sup>55</sup> This lack of differentiation led the court to

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51. See Ellen G. Koenig, Note, *A Fair Trial: When the Constitution Requires Attorneys to Investigate Their Clients’ Brains*, 41 *FORDHAM URB. L.J.* 177, 183, 222 (2013) (collecting seventy-nine cases in the last twenty years that have held that trial counsel was ineffective for failing to present neuroscience evidence, 64% of which were decided after *Wiggins*).

52. *Wiggins* was decided on June 26, 2003. See *Wiggins v. Smith*, 539 U.S. 510, 510 (2003). A Westlaw search conducted on February 24, 2016, in the All Federal Cases database, using the Boolean terms “habeas & (‘organic brain damage’) /p (strickland OR ineffective)”, yielded 292 opinions: 206 were decided after *Wiggins*, 85 were decided before *Wiggins*, and one was decided on the same day as *Wiggins*. A search conducted the same day using the same terms, except substituting “brain damage” for “organic brain damage,” yielded 567 cases: 425 after *Wiggins*, 141 before *Wiggins*, and one the same day as *Wiggins*.

53. A study of the success rate of federal habeas litigation, which examined habeas petitions filed in district courts from 2000 to 2002 and decided before December 2006, found that of the 267 petitions filed by state prisoners on death row, only thirty-three were granted. See NANCY J. KING, FRED L. CHEESMAN II & BRIAN J. OSTRUM, FINAL TECHNICAL REPORT: HABEAS LITIGATION IN U.S. DISTRICT COURTS, 28 (2007), <https://www.ncjrs.gov/pdffiles1/nij/grants/219559.pdf>. And of those thirty-three successful petitions, eight of them—24%—were granted based on claims that trial counsel failed to find and present mitigating mental health evidence. *Id.* at 117–24.

54. See *Lynch v. Sec’y, Fla. Dep’t of Corr.*, 776 F.3d 1209, 1221–22 (11th Cir. 2015) The prisoner’s experts had diagnosed him with (1) “psychoaffective disorder,” (2) “right hemisphere brain damage and psychosis,” and (3) “mild brain impairment and possible psychosis.” *Id.* at 1222 n.10.

55. See Appellee Cross-Appellant’s Brief at 35, 45–47, 50, 52, *Lynch v. Sec’y, Fla. Dep’t of Corr.*, 776 F.3d 1209 (11th Cir. 2015), No. 12-15188, 2015 WL 108623.

address the various diagnoses “us[ing] ‘brain impairment’ as an umbrella term.”<sup>56</sup>

Indeed, courts in federal habeas litigation frequently deal with claims of organic brain damage without ever defining the petitioner’s malady any more specifically.<sup>57</sup> Even in cases where a single expert’s diagnosis is at issue, courts rarely define the particular condition that qualifies as “organic brain damage.”<sup>58</sup> The same applies to the level of detail courts use when describing the particular tests or diagnostic tools that experts use in diagnosing an individual with “organic brain damage.”<sup>59</sup> In another decision from the Eleventh Circuit, the court discussed an affidavit from a “board-certified clinical neuropsychologist” attesting that the petitioner “suffers from organic brain damage to the frontal lobes,” but did not identify what particular tests or diagnostic methods the expert used to reach that conclusion.<sup>60</sup> In sum, litigants and courts are using the term organic brain damage without any particular precision as to what the term means or how it is being assessed.

## II. BRAIN FUNCTION ASSESSMENTS AND THEIR IMPLICATIONS

The failure of habeas litigants and courts to take a more nuanced approach to the rising number of claims based on allegations of organic brain damage is problematic. To begin with, the current medical literature does not recognize “organic brain damage” as a specific condition. So continuing to use the label as if it were a distinct medical condition presents the risk that habeas petitioners with different conditions are treated as if they were the same. Furthermore, the two general methods used by expert witnesses in cases where the habeas petitioner claims to have organic brain damage—neuroimaging and neurofunctional assessments—both have serious shortcomings, especially when used as means to assess an individual’s culpability for a particular criminal act.<sup>61</sup>

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56. See *Lynch*, 776 F.3d at 1222 n.10.

57. See, e.g., *Dickens v. Ryan*, 740 F.3d 1302, 1309, 1317, 1319, 1329 (9th Cir. 2014); *Sneed v. Johnson*, 600 F.3d 607, 610 (6th Cir. 2010); *Knighton v. Mullin*, 293 F.3d 1165, 1179 (10th Cir. 2002).

58. See, e.g., *Seidel v. Merkle*, No. C-94-1621-SI, 1997 WL 168541, at \*1 (N.D. Cal. Mar. 27, 1997).

59. See, e.g., *Stokley v. Ryan*, 659 F.3d 802, 812 (9th Cir. 2011); *Fautenberry v. Mitchell*, 515 F.3d 614, 648 (6th Cir. 2008); *Smith v. Trammell*, No. CIV-09-293-D, 2014 WL 4627225, at \*25 (W.D. Okla. Sept. 16, 2014).

60. *Ferrell v. Hall*, 640 F.3d 1199, 1213 (11th Cir. 2011).

61. See *Koenig*, *supra* note 51, at 195–99 (identifying the two major sources of mitigating evidence of brain abnormalities as being “brain scans” (i.e., neuroimaging) and neuroscience evaluations that test psychological function (i.e., neurofunctional assessments)). This Note uses the term “neurofunctional assessment” as a catchall for the various non-imaging methods that test for brain damage or impairment by assessing functions associated with certain brain structures or pathways. See *infra* notes 97–101 and accompanying text.

### A. *The Absence of “Organic Brain Damage” in the Medical Literature*

Despite habeas petitioners’ frequent invocation of the term as if it were a discrete medical condition, the current medical literature does not recognize “organic brain damage” as such. Searches of the MEDLINE database for “organic brain damage,” “organic brain dysfunction,” “organic brain disorder,” and “organic brain injury” yielded no current medical literature supporting the use of these blanket terms to describe a consistent set of symptoms.<sup>62</sup> The term “organic brain damage” was found only in decades-old articles, where it was used merely as a blanket term to describe a set of consistent symptoms and not as a concrete diagnosis.<sup>63</sup>

The term could conceivably refer, non-exclusively, to a number of pathologies with a great breadth of symptoms among them, such as anoxic, traumatic, congenital, or degenerative brain injuries.<sup>64</sup> The uncertain application of the term in the medical literature to describe such a broad range of pathologies—with appreciably different symptoms and effects—counsels against allowing it to develop into a new trend in a highly impactful area of jurisprudence.

### B. *Neuroimaging as Evidence of Brain Damage*

When habeas petitioners present claims based on organic brain damage, they often rely on neuroimaging interpreted by an expert witness.<sup>65</sup> Neuroimaging evidence consists of images depicting the results of various noninvasive brain scan technologies.<sup>66</sup> Hence neuroimaging is sometimes called a “brain scan.”<sup>67</sup>

There are currently seven commonly used technologies for producing neuroimaging evidence.<sup>68</sup> Most of the technologies produce either structural or functional images of the brain.<sup>69</sup> Structural images depict the specific anatomy (the structure) of the soft tissue that makes up an

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62. MEDLINE is a bibliographic database of life sciences and biomedical information that compiles content from academic journals covering medicine, healthcare, pharmacy, and biology, among other areas.

63. See, e.g., Arthur J. Gallese, Jr., *Spiral Aftereffect as a Test of Organic Brain Damage*, 12 J. CLINICAL PSYCHOL. 254, 254 (1956); Jack R. Haynes & S.B. Sells, *Assessment of Organic Brain Damage by Psychological Tests*, 60 PSYCHOL. BULL. 316, 316–17 (1963); Erika Fromm et al., *Hypnotic Simulation of Organic Brain Damage*, 69 J. ABNORMAL & SOC. PSYCHOL. 482, 482 (1964).

64. Email from Jesse Chen, M.D., to author (Jan. 18, 2015, 9:12 EST) (on file with author).

65. See Koenig, *supra* note 51, at 194–95.

66. See *id.* at 194–98; Abram S. Barth, Note, *A Double-Edged Sword: The Role of Neuroimaging in Federal Capital Sentencing*, 33 AM. J.L. & MED. 501, 503 (2007).

67. See Koenig, *supra* note 51, at 194–95.

68. See Walter Sinnott-Armstrong et al., *Brain Images as Legal Evidence*, 5 EPISTEME 359, 359–61 (2008).

69. *Id.*

individual's brain, while functional images depict the activity (the function) of an individual's brain over a period of time.<sup>70</sup> Structural imaging technologies include: computerized tomography (CT) scanning, diffusion tensor imaging (DTI), and magnetic resonance imaging (MRI).<sup>71</sup> Functional imaging technologies include: positron emission tomography (PET), single-photon emission computerized tomography (SPECT), and magnetoencephalography (MEG).<sup>72</sup> The one technology that falls into both categories is functional magnetic resonance imaging (fMRI), which produces both structural and functional images.<sup>73</sup>

Exploring the strengths and limitations of all seven technologies is beyond the scope of this Note. Instead, this Note will focus on functional MRI, which has become the most widely used form of neuroimaging due to its ability to produce both structural and functional images, its high-resolution images, and its relatively low cost.<sup>74</sup> Functional MRI scans measure fluctuations in the ratio of oxygenated to deoxygenated blood in the brain because blood-flow strength relates to neural activity.<sup>75</sup> During the scanning, the individual may be asked to perform tasks that require the use of different brain processes, resulting in images that show the difference in signals generated during the completion of the different tasks.<sup>76</sup> The highly-processed images create a visual representation of differentials in brain activity between contrasting states, as measured by magnetic resonance.<sup>77</sup>

Functional MRI has been a powerful tool in general scientific research.<sup>78</sup> In particular, researchers have used fMRI to link development and activity levels in particular areas of an individual's brain with various personality or behavior abnormalities.<sup>79</sup> For example, studies using fMRI have identified a link between particular areas of the brain and Antisocial

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70. *Id.*

71. *See id.*; Barth, *supra* note 66, at 503; Staci A. Gruber & Deborah A. Yurgelun-Todd, *Neurobiology and the Law: A Role in Juvenile Justice?*, 3 OHIO ST. J. CRIM. L. 321, 325 (2006); Koenig, *supra* note 51, at 197–98; O. Carter Snead, *Neuroimaging and the “Complexity” of Capital Punishment*, 82 N.Y.U. L. REV. 1265, 1281 (2007).

72. *See* Snead, *supra* note 71, at 1282–83.

73. *See* Barth, *supra* note 66, at 503.

74. *See* Koenig, *supra* note 51, at 197–98; Sinnott-Armstrong et al., *supra* note 68, at 359; Snead, *supra* note 71, at 1284–85.

75. *See* Sinnott-Armstrong et al., *supra* note 68, at 361. In contrast, PET and SPECT scans merely measure cerebral blood flow generally, without differentiating oxygenated and deoxygenated blood. *See* Snead, *supra* note 71, at 1284–85.

76. *See* Sinnott-Armstrong et al., *supra* note 68, at 361.

77. *Id.*

78. *See, e.g.,* Snead, *supra* note 71, at 1284.

79. *See* Sinnott-Armstrong et al., *supra* note 68.

Personality Disorder (APD).<sup>80</sup> APD is commonly associated with violent criminal activity and has been diagnosed in high percentages of male criminals within prison systems in various countries.<sup>81</sup> Studies have found a correlation between APD and a reduced amplitude of low-frequency fluctuations in the right orbitofrontal cortex, the left temporal pole, the right inferior temporal gyrus, and the left cerebellum posterior lobe compared to normal controls.<sup>82</sup> Studies have also found general links between APD and prefrontal and temporal cortex impairments, as well as abnormal amygdala activity compared to normal controls.<sup>83</sup> Further, studies have found reduced activity in the prefrontal and temporal lobes of individuals with psychopathy, while showing structural and functional abnormalities in common with individuals with APD.<sup>84</sup>

While fMRI has proved extremely valuable for general scientific research, it has not been as valuable in linking a particular individual's neural activity and that individual's past behavior.<sup>85</sup> In fact, neuroscientists have not yet established—even as a general matter—a definitive “causal relationship between specific brain functionality and criminal behavior.”<sup>86</sup> Simply put, “many brain regions are involved in a wide variety of functions, and this considerably complicates any effort to directly connect a particular and unusual brain feature with a particular past behavior.”<sup>87</sup> Neuroscience is even further away from the kind of connection that is most relevant for criminal law: being able to draw sure conclusions about whether a particular individual's brain abnormalities were a contributing factor in a specific crime the individual committed.<sup>88</sup>

Using neuroimaging evidence in the criminal litigation context requires an understanding of the precise nature of the data and its probative value in establishing criminal responsibility.<sup>89</sup> This value is frustrated by a number

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80. Huasheng Liu et al., *Changes in Low-Frequency Fluctuations in Patients with Antisocial Personality Disorder Revealed by Resting-State Functional MRI*, 9 PLOS ONE 1 (2014) (investigating the underlying neural mechanisms of Antisocial Personality Disorder, a personality disorder characterized by repeat violent criminal behavior).

81. *Id.* at 1 (citing Seena Fazel & John Danesh, *Serious Mental Disorder In 23000 Prisoners: A Systematic Review Of 62 Surveys*, 359 LANCET 545 (2002)).

82. *Id.*

83. *Id.*

84. *Id.* at 1–2.

85. See Sinnott-Armstrong et al., *supra* note 68, at 362–67; see also Nicole A. Vincent, *Neuroimaging and Responsibility Assessments*, 4 NEUROETHICS 35, 42 (2011).

86. See, e.g., Adam Teitcher, Note, *Weaving Functional Brain Imaging into the Tapestry of Evidence: A Case for Functional Neuroimaging in Federal Criminal Courts*, 80 FORDHAM L. REV. 355, 363 (2011).

87. Walter Glannon, *The Limitations and Potential of Neuroimaging in the Criminal Law*, 18 J. ETHICS 153, 155 (2014) (quoting Owen D. Jones et al., *Neuroscientists in Court*, 14 NATURE REV. NEUROSCIENCE 730 (2013)).

88. See Sinnott-Armstrong et al., *supra* note 68, at 362–67; see also Vincent, *supra* note 85.

89. See Sinnott-Armstrong et al., *supra* note 68, at 362–67.

of problems that arise when neuroimaging is used in the courtroom. First, difficulties arise when trying to establish normality based on brain function images. Because studies based on functional brain images generally present data averages across a group, they fail to show the naturally-occurring wide range of variations between individual profiles.<sup>90</sup> Determining abnormality can thus be elusive. Second, even if studies can determine abnormal functional patterns, the low base rate (rarity) of individuals within the population who have criminal-responsibility-affecting functional abnormalities will result in a high incidence of false positives—a low predictive value.<sup>91</sup> A defendant who receives such a false positive is unlikely to submit to repeat testing that would improve on the predictive value of neuroimaging research.<sup>92</sup> Third, even the establishment of a functional brain abnormality in a particular defendant does not equal a valid prediction that the defendant will actually commit a violent crime.<sup>93</sup> Fourth, even neuroimaging evidence of a brain abnormality that is correlated with violent criminal behavior (such as prefrontal and temporal cortex impairments associated with APD)<sup>94</sup> does not prove causation, except in rare circumstances.<sup>95</sup> An example of such an exception could be a tumor—detectable through MRI—that affects executive-function-performing parts of the brain and thus impairs a defendant's control over his actions.<sup>96</sup>

### C. *Neurofunctional Assessments as Evidence of Brain Damage*

The other major source of evidence for habeas petitioners claiming to suffer from organic brain damage are assessments that measure brain function as a means to identify brain damage. These include neuropsychological assessments, neurological evaluations, and neuropsychiatric evaluations.<sup>97</sup> Neuropsychological assessments examine an individual's cognitive, emotional, and executive functions.<sup>98</sup> The subject takes a battery of tests aimed at assessing psychological functions known to

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90. *Id.* at 362.

91. *Id.* at 364.

92. *Id.*

93. *Id.*

94. *See supra* Part II.A.

95. *See* Sinnott-Armstrong et al., *supra* note 68, at 365 (describing the unusual case of a convicted child pornographer whose criminal behavior was shown to commence, stop, resume, and then finally cease with the growth, surgical removal, recurrence, and final successful removal of a large tumor in his brain).

96. Glannon, *supra* note 87, at 155.

97. John H. Blume & Emily C. Paavola, *Life, Death, and Neuroimaging: The Advantages and Disadvantages of the Defense's Use of Neuroimages in Capital Cases—Lessons from the Front*, 62 MERCER L. REV. 909, 911–12 (2011).

98. *Id.* at 911.

be linked with particular brain structures or pathways, such as attention and concentration, visual perception and reasoning, self-regulation and motor ability, and emotional status.<sup>99</sup> Meanwhile, neurological evaluations involve a clinical assessment, usually conducted by a neurologist or neurosurgeon, that examines “the parts and functions of the body that are most often impacted by a problem in the central nervous system.”<sup>100</sup> And neuropsychiatric evaluations use a variety of tests aimed at assessing cognitive and mental functioning.<sup>101</sup>

Like neuroimaging, behavioral assessments present significant problems when presented as evidence of whether a defendant can make sound moral judgments. First, behavioral assessments provide ample opportunity for a lack of truthfulness. Assessments conducted directly with the defendant take the form of tests. Given the benefits of obtaining evidence that indicates diminished cognitive and volitional capacities, defendants have immense incentive to give those answers which will be most advantageous in court.<sup>102</sup> The same incentive to lie applies to third parties providing biographical information. Second, it may not be possible to devise an objectively verifiable assessment that reveals whether a defendant is incapable of controlling himself or merely refuses to do so.<sup>103</sup>

### III. ANALYZING CLAIMS OF ORGANIC BRAIN DAMAGE IN FEDERAL HABEAS LITIGATION

Given the imprecise nature of “organic brain damage” and the limitations of neuroimaging and neurofunctional evaluations as means of explaining an individual’s criminal conduct, courts should be cautious when assessing habeas claims based on allegations of organic brain damage. As explained above, habeas petitioners usually present evidence of organic brain damage as support for ineffective-assistance-of-counsel claims—faulting their trial counsel for not finding and presenting evidence of their organic brain damage as mitigation evidence in the penalty phase of their trials.<sup>104</sup> A nuanced approach to such claims must demand precision from the habeas petitioner in at least three facets of the ineffective-assistance claim.

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99. *See id.*

100. *Id.* at 912.

101. *Id.*

102. Vincent, *supra* note 85, at 37.

103. *Id.* (citing Bernadette McSherry, *Criminal Responsibility, ‘Fleeting’ States of Mental Impairment, and the Power of Self-Control*, 27 INT’L J. L. & PSYCHIATRY 445, 224 (2004)).

104. *See, e.g.*, Appellee Cross-Appellant’s Brief at 35, 45–47, 50, 52, Lynch v. Sec’y, Fla. Dep’t of Corr., 776 F.3d 1209 (11th Cir. 2015) (No. 12-15188), 2015 WL 108623.

The first facet concerns whether trial counsel's investigation of potential mitigation evidence qualifies as deficient performance.<sup>105</sup> After *Wiggins* and *Rompilla*, a habeas petitioner can establish deficient performance by demonstrating that his trial attorney did not have a reasonable strategic justification for not investigating whether the defendant had a brain abnormality that mitigated his culpability.<sup>106</sup> But when a habeas petitioner brings such a claim, courts should be mindful that there are many potential methods of assessing whether an individual suffers from brain damage, and a trial attorney's failure to pursue a particular form of expert evidence is not necessarily a strategic blunder.

As discussed earlier, there are at least ten generally accepted methods of assessing whether an individual suffers from a brain abnormality, including seven forms of neuroimaging and three forms of neurofunctional assessment.<sup>107</sup> Furthermore, some of the methods are far more costly than others. For example, some neurofunctional assessments can cost three or four thousand dollars, and fMRIs can cost "a few thousand dollars" as well.<sup>108</sup> On the other hand, a CT scan is usually less than \$700.<sup>109</sup> Because a habeas petitioner bears the burden of establishing deficient performance and *Strickland* requires courts to presume counsel acted reasonably,<sup>110</sup> courts must make sure that the habeas petitioner has eliminated any presumptive basis for counsel's failure to acquire the expert evidence that the habeas petitioner presents on appeal.

The second facet of ineffective-assistance-of-counsel claims that warrants close attention concerns the prejudice determination.<sup>111</sup> Courts must be careful not to allow a general label, like "organic brain damage," to have an undue impact on their assessment of whether the petitioner was prejudiced by counsel's failure to present certain expert evidence at the mitigation phase of the trial. Often, habeas petitioners will criticize trial counsel for failing to present neuroscience evidence showing that the defendant had brain damage, even though trial counsel did present testimony showing that the defendant suffered from a psychological disorder.<sup>112</sup>

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105. See *Strickland v. Washington*, 466 U.S. 668, 687–88 (1984) (requiring that an ineffective assistance of counsel claim "show that counsel's performance was deficient" which means "that counsel's representation fell below an objective standard of reasonableness").

106. See *Rompilla v. Beard*, 545 U.S. 374, 380–90 (2005); *Wiggins v. Smith*, 539 U.S. 510, 521–34 (2003).

107. See *supra* notes 68–73, 97–101 and accompanying text.

108. Koenig, *supra* note 51, at 196, 199.

109. See *id.* at 197.

110. See *Strickland*, 466 U.S. at 690.

111. See *id.* at 687, 694–96.

112. See, e.g., *Lynch v. Sec'y, Fla. Dep't of Corr.*, 776 F.3d 1209, 1221–22 (11th Cir. 2015) (assessing a claim that criticized trial counsel for presenting expert testimony concluding that the



The third facet of ineffective-assistance-of-counsel claims that warrants close attention also involves the prejudice determination.<sup>113</sup> Given how general the conclusions based on neuroimaging and neurofunctional assessments are, courts should be cautious about allowing new evidence to have an outsized impact on their prejudice determinations. When a habeas petitioner brings an ineffective-assistance-of-counsel claim challenging counsel's performance in the mitigation phase of the trial, the petitioner is challenging the death sentence, not the conviction.<sup>114</sup> This alters a court's prejudice analysis slightly so that the court "consider[s] 'the totality of the available mitigation evidence—both that adduced at trial, and the evidence adduced in the habeas proceeding'—and 'reweig[hs] it against the evidence in aggravation.'"<sup>115</sup>

As neuroimaging technology continues to develop and potentially broaden in applicability, there are competing views on whether it possesses the necessary credibility for use in the courtroom.<sup>116</sup> Most importantly, brain function images lack probative value of the defendant's mental state at the time of the crime. Additionally, there is evidence that neuroscience evidence may be unfairly prejudicial to the defendant.<sup>117</sup> Continued research is necessary to improve our understanding of what roles different neural pathways play in brain abnormalities associated with crime.<sup>118</sup> Deepening knowledge of the relationships between brain function and behavior will be instrumental in establishing any potential probative value neuroimaging may have in the courtroom.

#### A. *The Relationship Between Neuroimaging and Behavior*

Despite its perceived promise of deep insights into the neural basis of criminality, neuroscience is still in its youth and requires significant progress before it can provide proof of criminal responsibility in the courtroom. Critics of functional neuroimaging emphasize that the data provided by fMRI and other brain scan technologies is not dispositive.<sup>119</sup>

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defendant suffered from a "schizoaffective disorder" but not presenting any testimony concluding that he had brain damage).

113. See *Strickland*, 466 U.S. at 690.

114. See *id.* at 695; see also *Johnson v. United States*, 860 F. Supp. 2d 663, 762 (N.D. Iowa 2012).

115. *Porter v. McCollum*, 558 U.S. 30, 41 (2009) (quoting *Williams v. Taylor*, 529 U.S. 362, 397–98 (2000)).

116. See generally *supra* Part II.

117. 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 (2010) (arguing that fMRI should not be admitted at this time as evidence of a defendant's past mental state).

118. See Betsy J. Grey, *Neuroscience, PTSD, and Sentencing Mitigation*, 34 CARDOZO L. REV. 53, 92 (2012).

119. Teitcher, *supra* note 86.

Neuroscientists still await the establishment of a causal relationship between specific neural pathways as well as their abnormalities and violent criminal behavior.<sup>120</sup> The presence of abnormalities alone does not provide sufficient insight into a defendant's behavior—"many brain regions are involved in a wide variety of functions, and this considerably complicates any effort to directly connect a particular and unusual brain feature with a particular past behavior."<sup>121</sup> Further, it is possible for an abnormality to have been present within a defendant's brain for some time and to have had no effect on the defendant's ability to control himself at the time of the crime.<sup>122</sup> There is a need for clarification regarding what kinds of neuroscience evidence are actually related to a defendant's capacity to understand the consequences of his actions. The current state of the law does not clearly distinguish between types of neuroscience evidence that are and are not relevant to this capacity. This results in a lack of clarity on what evidence is appropriate for use in the mitigation phase of sentencing. In some cases, questions have arisen regarding whether evidence of organic brain damage should go toward a classification as mental disorder or mental defect. A defendant's mental defect could underlie an inability to understand or appreciate the consequences of his actions.<sup>123</sup>

With the increasing use of both neuroscience evidence and mitigating factors like organic brain damage comes an increased need for expert witnesses who have a sophisticated understanding of neuroscience and its limitations.<sup>124</sup> Differences between the neuroscience and legal fields should inform how neuroscience evidence is presented to the layperson, so as to avoid confusion and inappropriate inferences.<sup>125</sup> For example, uninformed jurors and judges could easily interpret fMRI images as analogous to an x-ray showing activity within the brain.<sup>126</sup> The reality is much more complex—the seemingly innocent simplification ignores the very important fact that the images actually represent a processed statistical analysis of data taken from blood flow within the brain.<sup>127</sup> This fact has implications for how statistically outlying data should be treated.

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120. *Id.*

121. Glannon, *supra* note 87, at 155 (quoting Owen D. Jones et al., *Neuroscientists in Court*, 14 NATURE REV. NEUROSCIENCE 730, 734 (2013)).

122. *Id.*

123. *See* *Briscoe v. Scribner*, No. CIV S-04-2175 FCD GGH P, 2010 WL 1525695 (E.D. Cal. Apr. 15, 2010) (using this proposition at trial).

124. *See* Jones et al., *supra* note 87, at 732–33.

125. *Id.* at 733.

126. *Id.*

127. *Id.*

*B. The Impact of Neuroscience Evidence on the Jury*

Concerns over the use of neuroscience in the courtroom extend beyond its unestablished probative value. As evidenced by the need for competent expert witnesses to help maintain standards and aid in the interpretation of brain scan images, there is a significant risk that those unfamiliar with neuroscience may misinterpret the data they see. Jurors may mistakenly assign the images a probative value that they lack. The visual allure of images may impart scientific credibility beyond that seen with verbal evidence alone.<sup>128</sup> Empirical studies suggest that the images may have a more persuasive impact on jurors than is warranted.<sup>129</sup> Jurors in one study indicated that they had changed their minds about the defendant's criminal liability after being shown brain function images.<sup>130</sup> They mistakenly understood that evidence of a brain abnormality meant that the defendant was unable to control his violent impulses.<sup>131</sup>

Although evidence of potential juror bias has elicited concern among lawyers and judges, few studies have been conducted to date, and the effect of neuroscience on juries is still unclear.<sup>132</sup> A 2014 study suggested that showing jurors neuroimages increases the chances of a desirable outcome for defendants with certain kinds of brain abnormalities.<sup>133</sup> For example, defendants diagnosed with psychopathy benefited from the use of neuroimages—they were deemed less responsible and were less frequently sentenced to death.<sup>134</sup> However, schizophrenic defendants faced increased judgments of responsibility when neuroimages were presented.<sup>135</sup> In contrast, neuroscience evidence presented without accompanying images decreased both judgments of responsibility and death sentences.<sup>136</sup> Furthermore, jurors appeared to favor defense counsel's arguments when the defense presented an expert witness who showed them neuroimages.<sup>137</sup> Juror responses to images of brain scans thus remain complex and uncertain. As a result, judges must carefully weigh the probative value of

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128. Tanneika Minott, *Born This Way: How Neuroimaging Will Impact Jury Deliberations*, 12 DUKE L. & TECH. REV. 219, 226 (2014).

129. See Glannon, *supra* note 87, at 157–58.

130. *Id.*

131. *Id.*

132. Adam B. Schneiderman, *No Such Thing as a Sure Thing: Neuroscience, the Insanity Defense, and Sentencing Mitigation*, 26 THE JURY EXPERT 1, 12 (2014) (finding that “the impact [on juries] of neuroscientific evidence and neuroimages in particular is complex and remains unclear”).

133. *Id.* (citing findings from Michael J. Saks et al., *The Impact of Neuroimages in the Sentencing Phase of Capital Trials*, 11 J. EMPIRICAL LEGAL STUD. 105 (2014)).

134. Saks et al., *supra* note 133.

135. *Id.*

136. *Id.*

137. *Id.*

neuroimaging evidence against potential juror bias when deciding whether to admit images.

#### CONCLUSION

Technological advances in neuroscience have brought the unprecedented ability to noninvasively study the brain's functions. Brain imaging presents enormous potential with regard to evidence in criminal litigation. Capital defendants claim with increasing frequency that they have diminished culpability as a result of "organic brain damage." The use of the catchall concept of organic brain damage as a mitigating factor in sentencing is inappropriate as long as courts fail to provide a concrete definition for the term and establish standards for neuroimaging evidence supporting brain abnormalities. At this point in its development, neuroimaging lacks the refinements necessary to establish a probative relationship between brain function images and criminal responsibility. Courts should be extremely careful in allowing the use of "organic brain damage" as a mitigating factor in capital litigation and in admitting both neuroscience and behavioral evidence in support of it.

*Aleksandra Matuszewski\**

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\* J.D. Candidate, the University of Alabama School of Law (May 2016).