



# Neuroscience nuance: dissecting the relevance of neuroscience in adjudicating criminal culpability

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## INTRODUCTION

The usefulness of neuroscience in determining the blameworthiness of a particular criminal defendant is highly contested.<sup>1</sup> On one side are those who argue that neuroscience is virtually irrelevant in criminal court or useful only as corroborating evidence of legally relevant impairment proven through other means.<sup>2</sup> On the other

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<sup>1</sup> This article does not address the use of neuroscience to assess competence or treatment issues, nor issues of criminal policy. The MacArthur Foundation Research Network on Law and Neuroscience, <http://lawneuro.org/> has been especially focused on the latter issue. See eg Iris Vilares et al., *Predicting the Knowledge-Recklessness Distinction in the Human Brain*, P NATL. ACAD. SCI. USA EARLY EDITION, [https://papers.ssrn.com/sol3/papers.cfm?abstract\\_id=2922210](https://papers.ssrn.com/sol3/papers.cfm?abstract_id=2922210) (finding neural evidence of a detectable difference between knowing and recklessness mental states, a finding that might bolster that distinction in criminal cases); cf. *United States v. Hendrickson*, 25 F.Supp.3d 1166, 1172, 1179 (N.D. Iowa 2014) (deciding, based in part on neuroscience, that addiction can be a basis for mitigation at sentencing). In these latter instances, neuroscience is relied upon to support a change in the law's stance to entire categories of people, not a specific individual.

<sup>2</sup> Uri Maoz & Gideon Yaffe, *What Does Recent Neuroscience Tell Us about Criminal Responsibility*, 3 J. L. & BIOSCI. 120, 124 (2016) ('very little is known about the brain that is of significance for understanding criminal

side are those who contend that neuroscience will soon show, if it has not already shown, that most criminals have little or no control over their behavior.<sup>3</sup> In between are those who recognize the tenuous nature of neuroscientific claims in criminal cases but nonetheless suggest that they can in some cases provide independent evidence relevant to excuse or mitigation.<sup>4</sup>

This article takes issue with all three stances. The first two sets of claims are overstated, and the last type of claim is insufficiently nuanced. Neuroscience does have something to offer court determinations of criminal liability and punishment, but it is far from upending the criminal law's basic premise that most choices to commit crime are blameworthy. At the same time, those who agree with this intermediate position have not done a particularly good job at clarifying how they think neuroscience can help. Any such clarification needs to recognize that there are many different types of neuroscience evidence and many different types of legal claims that it might address.

This article explores more precisely the types of neuroscience evidence that might be presented and when that evidence is material under accepted legal doctrine. It concludes that, even on the assumption that the data presented are accurate, some commonly proffered neuroscientific evidence is immaterial or only weakly material, not only at trial but also at sentencing. At the same time, it recognizes that certain

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responsibility'); SALLY SATEL & SCOTT O LILJENFELD, *THE SEDUCTIVE APPEAL OF MINDLESS NEUROSCIENCE* 445 (2013) (neuroscience 'cannot distinguish between those who *could not* control themselves from those who *did not* control themselves, nor from those in between who managed to wrestle their impulses to the ground'); Michael S. Pardo & Dennis S. Patterson, *Philosophical Foundations of Law and Neuroscience*, 2010 U. ILL. L. REV. 1211, 1231–35 (arguing that claims about neuroscience's relevance to criminal law 'confuse properties of people with properties of brains'); Stephen J. Morse, *Brain Overclaim Syndrome and Criminal Responsibility: A Diagnostic Note*, 3 OHIO ST. J. CRIM. L. 397, 405 (2006) (stating 'a perfect correlation between brain states and the behavioral criteria for responsibility . . . is a fantasy based on present knowledge and probably always will be when we are considering complex human actions', but also noting, *Id.* at 400–1, that occasionally neuroscience can bolster a particular conclusion when 'behavioral evidence is in doubt').

<sup>3</sup> DAVID M. EAGLEMAN, *INCOGNITO* 151 (2011) (relying on neuroscience findings in concluding that 'blameworthiness is the wrong question'); Joshua Greene & Jonathan Cohen, *For the Law, Neuroscience Changes Nothing and Everything*, 359 PHIL. TRANS. ROY. SOC'Y LOND. B: BIOL. SCI. 1775–76, 1781 (2004) (stating that all criminals 'are victims of neural circumstances' and speculating that as science progresses 'more and more people will develop moral intuitions that are at odds with our current social practices'); Robert M. Sapolsky, *The Frontal Cortex and the Criminal Justice System*, 359 PHIL. TRANS. ROY. SOC'Y LOND. B: BIOL. SCI. 1787, 1794 (2004) (stating that neurological research suggests that human agents are like 'cars', 'punishment' of which makes no sense).

<sup>4</sup> See Elizabeth O. Bennet, *Neuroscience and Criminal Law: Have We Been Getting It Wrong for Centuries and Where Do We Go From Here?*, 85 FORDHAM L. REV. 437, 449 & 451 (2016) ('Clearly, brain scans may be used in sentencing proceedings to identify and support claims of lesser culpability due to circumstances beyond the control of the offender that could have a mitigating effect on the sentence', and suggesting that neuroscience will 'likely' lead to expansion of the excuses); Dean Mobbs et al., *Law, Responsibility and the Brain*, 5 PLOS BIOL. 693, 696 (2007) ('there are many instances where brain disease can lead to anti-social behaviour, and these inevitably pose important complications for moral and legal systems that tend to divide responsibility for actions into dichotomous alternatives—guilty versus not guilty—instead of seeing responsibility as existing along a continuum'); Richard Redding, *The Brain-Disordered Defendant: Neuroscience and Legal Insanity in the Twenty-First Century*, 56 AM. U. L. REV. 51, 53 (2006) (arguing for 'a return to control tests for insanity' that 'comports with modern neuroscience research on the role of brain dysfunction in impulsive criminal behavior'); Laura Reider, *Toward a New Test for the Insanity Defense: Incorporating the Discoveries of Neuroscience into Legal and Moral Theories*, 48 UCLA L. REV. 289, 331 (1998) ('the empirical sciences suggest that this subset of offenders [certain offenders with brain lesions] is not capable of rational choice once the emotional and biological cues that normally guide individuals in the decision-making process are absent').

types of neuroscience evidence can be very useful in criminal adjudication, especially at sentencing.

The article begins in Part I, like many articles in this vein do, by describing the criminal law doctrines that are most likely to trigger use of neuroscience. The description of this familiar territory is brief, meant only to emphasize that, at present, these doctrines are narrowly defined. At trial, they tend to minimize the relevance of, and sometimes preclude, neuroscience evidence. At sentencing, the scope of inquiry is broader and thus neuroscience is more likely to be useful. At the same time, however, if the risk of reoffending is a legitimate sentencing consideration, as is the case in many jurisdictions, such evidence is also often relevant to the prosecution's case.

Part II identifies five types of neuroscience evidence and describes how they relate to the law defining criminal liability and criminal punishment described in Part I. The five types, in roughly ascending order of usefulness, are as follows: (1) *Evidence of abnormality*: Evidence showing that the defendant has neurological impairment (eg brain imaging showing that the defendant has frontal lobe disorder, or FLD); (2) *Cause-of-an-effect evidence*: Evidence showing that the defendant's neurological impairment is common in criminals or others who behave in an antisocial manner (eg research showing that many criminals have FLD); (3) *Effect-of-a-cause evidence*: Evidence tending to show that the defendant's neurological impairment predisposed him or her to commit the crime (eg research showing that people with FLD are more likely to commit crime than those without FLD); (4) *Individualized neuropsychological findings compared against known performance baselines*: Psychoneurological testing results showing that the defendant has behavioral impairments that are legally relevant (eg testing showing that the defendant, say one with FLD, is highly impulsive or unable to conceptualize); and (5) *Individualized neuropsychological findings compared against known performance baselines*: Evidence showing that the defendant's impairments are similar to impairments the law has recognized as exculpatory or mitigating (eg evidence that the defendant's FLD is similar in legally relevant respects to the brain of a 14 year old).

The admissibility of neuroscience evidence should depend on which of these five categories is at issue. All five types of evidence may have what has been called 'rhetorical relevance', that is, the potential for swaying a judge or jury in the defendant's (or prosecution's) favor.<sup>5</sup> But a close analysis of each type of evidence makes clear that *genuine* relevance establishes a narrower threshold. More specifically, the first two types of neuroscience evidence should often be considered immaterial at trial; whether they should be admissible at sentencing depends on the law of the jurisdiction. The other three types of neuroscience evidence are more likely to be relevant at both trial and sentencing, with the final category of evidence probably being the most persuasive. But these latter types of evidence are also much more difficult to produce than the first two.

As the foregoing summary suggests, this article focuses entirely on what evidence scholars call relevance or materiality; it avoids complications that arise when courts also consider the validity of the science presented. Thus, this article will assume the accuracy of expert testimony to the effect that a defendant has, for example, FLD, that research

<sup>5</sup> Stephen J. Morse, *Brain Overclaim Redux*, 31 LAW & INEQ.: J. THEORY & PRACT. 509, 528 (2013) (noting that some reformers 'are so eager to achieve their desired result that they will either uncritically over-claim both the validity and legal relevance of the neuroscience, or they will simply grasp at rhetorical relevance if they believe that it helps their cause').

indicates a certain proportion of prisoners have FLD, or that a defendant has particular types of volitional impairment. This assumption is a big one, since some types of testimony about neuroscience are of questionable scientific pedigree.<sup>6</sup> But, over time, many of these flaws in producing neuroscience evidence could well be overcome. Further, this assumption allows attention to be focused on the more important issue, at least from the legal perspective, of when neuroscience evidence is relevant to criminal liability and punishment.

### CRIMINAL LAW DOCTRINE<sup>7</sup>

Suppose the following: John Doe, a 55-year-old farmer, is charged with murder.<sup>8</sup> There is no doubt Doe killed the person the prosecution alleges he killed; in fact Doe admits he shot the victim as she crossed one of his fields. Doe also says that at the time of the killing he was angry at the victim, who he believed had routinely trespassed on his property and scoffed at him when Doe protested. But Doe admits he did not act in self-defense. Although Doe has no criminal record, he and his family tell defense counsel that he has a history of ‘blowing up’ at people. In the hopes of finding something that will mitigate her client’s culpability, the lawyer goes to a neurologist, who scans the client’s brain and finds evidence of what the expert calls ‘frontal lobe disorder’.

The lawyer is excited by these results. But should she be? Does or should the criminal law consider this type of neuroscience evidence useful in deciding whether to convict or how much to punish an individual? If so, how can the lawyer shape the neuroscience evidence to make the best case possible at trial or sentencing? Should the attorney worry about the possibility that this evidence might be more useful to the prosecution?

Consider first the use of neuroscience to mitigate or nullify criminal responsibility at trial. At this stage of the process, neuroscience is most likely to be relevant to four defensive arguments: a claim that the defendant’s act was ‘involuntary’, a claim that the defendant lacked the mens rea (or mental state) for the offense, and a claim that the defendant was insane, due either to cognitive or volitional impairment. All of these doctrines have a limited scope.

The involuntary act argument is seldom applicable in a criminal case. It requires proof of a lack of conscious control over the body, as might occur with an epileptic seizure or sleepwalking.<sup>9</sup> Impulsive action, of the type often claimed in connection with criminal conduct, is not involuntary in this sense, because a connection between brain

<sup>6</sup> For a sampling of many of the problems, see Adina L. Roskies & Kimberly Farbota, *Scientific Issues*, in MODERN SCIENTIFIC EVIDENCE §§ 20:26, 20:49 (David L. Faigman et al. ed., 2016–2017.) (describing ‘artifacts’ that introduce erroneous readings, the difficulty in distinguishing ‘signal’ from ‘noise’, the error that can be introduced by pooling the multiple scans needed to obtain accurate results, problems associated with variability in individual brain functioning, and the difficulty in attributing particular functions or behaviors to any one area of the brain). See also OWEN JONES, JEFFREY SCHALL & FRANCIS SHEN, LAW AND NEUROSCIENCE ch. 9 (Limits and Cautions) (2014) (discussing these and other measurement issues).

<sup>7</sup> The foregoing discussion focuses entirely on United States doctrine. Discussion of the criminal law and rules of evidence in other countries is beyond the scope of this article.

<sup>8</sup> This hypothetical is based loosely on *People v. Chiesa*, 2005 WL 3113464, where the defendant unsuccessfully contended that brain damage, as demonstrated by CAT, PET, and SPECT scans showing damage to his prefrontal cortex, temporal lobes, and cerebellum, ‘left him unable to control his emotions and sent him into uncontrollable rages’. *Id.* at \*4.

<sup>9</sup> See WAYNE R. LAFAVE, CRIMINAL LAW 323 (5th ed. 2010).

and behavior still exists. Doe's admission that he consciously took aim and shot the victim precludes this defense in his case.

The second possible legal defense focuses on whether the defendant had the mens rea for the crime. For instance, first-degree murder requires proof of premeditation, while second-degree murder requires intent to kill, and manslaughter is usually associated with an intentional killing in the heat of passion or a non-intentional but reckless killing.<sup>10</sup> Most people, even those with flagrant psychosis (think of Andrea Yates, the woman who drowned her five children<sup>11</sup>), have the intent to engage in the conduct associated with the crime and to cause its result. Those who do not usually are at least reckless with respect to harm they cause, which often means they are guilty of some offense, albeit perhaps not as serious as one that requires intent.<sup>12</sup> Doe might have a partial mens rea defense. Given his admission that he intended to harm the victim, he cannot escape some liability. But he might be able to argue that his impairment meant he lacked the capacity to premeditate the killing, and thus at most is guilty of second-degree murder. He might even be able to argue that he was experiencing what the Model Penal Code calls 'extreme mental or emotional disturbance' (EMED) at the time of the crime, which if proven could, in some jurisdictions, allow him to end up with a manslaughter rather than murder conviction.<sup>13</sup> To succeed with this defense, however, he would have to show his anger is a 'reasonable' response to the victim's actions.<sup>14</sup> Furthermore, this argument is available only when the charge is homicide; EMED mitigation is not available in non-homicide cases.

The third and fourth possible defenses both have to do with insanity. The first question under any insanity test is whether the defendant had a mental disease or defect at the time of the crime.<sup>15</sup> Although the typical mental disorder associated with insanity is psychosis,<sup>16</sup> this predicate has generally been given broad scope, with a typical definition based on the D.C. Circuit Court of Appeals formulation stating that 'a mental disease or defect includes any abnormal condition of the mind which substantially

<sup>10</sup> See *id.* at 809 (first-degree murder), 816 (second-degree murder), 819 (manslaughter).

<sup>11</sup> *Woman Not Guilty in Retrial in the Deaths of Her Five Children*, NEW YORK TIMES, July 27, 2006 (reporting that Yates was found not guilty by reason of insanity, despite successfully carrying out a plan to drown her children, because 'she suffered from severe postpartum psychosis and, in a delusional state, believed that Satan was inside her and that killing the youngsters would save them from hell').

<sup>12</sup> Under the Model Penal Code for instance, recklessness is the default mens rea. See MODEL PENAL CODE, § 2.02(3) (1962).

<sup>13</sup> The Model Penal Code defines manslaughter as a homicide that is 'committed recklessly' or that 'is committed under the influence of extreme mental or emotional disturbance for which there is a reasonable explanation or excuse'. *Id.* at § 210.3(1). About 12 states have adopted this formulation. See Paul H. Robinson, *Murder Mitigation in Fifty-Two American Jurisdictions: A Case Study in Doctrinal Interrelation Analysis*, 47 TEX. TECH. L. REV. 19, 24 (2014).

<sup>14</sup> See *supra* note 13.

<sup>15</sup> LAFAVE, *supra* note 9, at 399 (describing the requirement and noting that '[t]here has never been a clear and comprehensive determination of what type of mental disease or defect is required' for insanity test purposes).

<sup>16</sup> See GARY MELTON ET AL., PSYCHOLOGICAL EVALUATIONS FOR THE COURTS: A HANDBOOK FOR MENTAL HEALTH PROFESSIONALS AND LAWYERS 203–4 (3d ed. 2007) (reporting research indicating that most insanity pleas are based on psychosis and most people found insane have psychosis).

affects mental or emotional processes and substantially impairs behavior controls'.<sup>17</sup> The evidence of FLD in Doe's case might well get the defense past that hurdle.

However, proving the mental defect predicate is not enough. The second question in an insanity case addresses the kind of impairment the mental defect caused. One formulation of this issue focuses on cognitive impairment, usually described as a substantial inability to know or appreciate the wrongfulness of the criminal act, and a second type of insanity test looks at volitional impairment, usually described as an irresistible impulse or a significantly impaired ability to conform behavior to the requirements of the law.<sup>18</sup> Because the impairment under either prong must be serious, even people with psychosis are often convicted under these defenses.<sup>19</sup> Doe may have thought the victim was in the wrong, but he also likely appreciated that he should not have killed her.<sup>20</sup> At best, then, he might have a volitional insanity defense. But that defense usually requires proof of a very strong compulsion,<sup>21</sup> and in any event less than a third of the states recognize it.<sup>22</sup>

This brief list exhausts the defenses available to most criminal defendants who assert they have a neurological impairment that mitigates their criminal liability. It should be apparent that in most cases neuroscience is not likely to be useful in proving these defenses, even if the court allows it to be introduced. In some cases where there is already strong evidence of automatic behavior, lack of intent, or severe cognitive or volitional impairment, such evidence might provide helpful corroboration of a defense. But in the typical criminal case, like Doe's, the defendant acts voluntarily, with intent, and with an understanding of the distinctions society makes between right and wrong. That leaves as the only viable defense in the typical neuroscience case either an EMED claim or an insanity claim based on volitional impairment. But, again, very few states recognize either defense.

Once a person has been convicted and sentencing is the issue, the law's strictures on evidence are more relaxed,<sup>23</sup> but not necessarily significantly. For instance, under the federal sentencing guidelines applicable in non-capital cases, a 'downward departure' based on mitigating mental condition is generally permissible only if the condition is 'present to an unusual degree' that 'distinguish[es] the case from the typical case[]',<sup>24</sup> and only if the condition 'contributed substantially to the commission of the

<sup>17</sup> *McDonald v. United States*, 312 F.2d 847, 851 (D.C. Cir. 1962). See also LAFAYE, *supra* note 9, at 399 ('it would seem that any mental abnormality, be it psychosis, neurosis, organic brain disorder, or congenital intellectual deficiency . . . will suffice if it has caused the consequences described in the second part of the test').

<sup>18</sup> For a description of the various cognitive and volitional formulations, see CHRISTOPHER SLOBOGIN ET AL., *LAW AND THE MENTAL HEALTH SYSTEM: CIVIL AND CRIMINAL ASPECTS* 629–34 (6th ed. 2014).

<sup>19</sup> MELTON ET AL., *supra* note 16, at 233 (reporting research indicating that only about a quarter of insanity pleas succeed at trial).

<sup>20</sup> See Redding, *supra* note 4, at 53 (2006) ('frontally-damaged individuals typically do not lack understanding, they lack behavioral control').

<sup>21</sup> MELTON ET AL., *supra* note 16, at 216–17 (describing approaches to the insanity defense consistent with caselaw that popularizes the volitional impairment test by requiring a showing that the defendant would have committed the crime even had there been a policeperson at the scene); *United States v. Kunak*, 17 C.M.R. 346, 357–58 (C.M.A. 1954) (describing the 'policeman at the elbow test').

<sup>22</sup> Robinson, *supra* note 13, at 33 (table).

<sup>23</sup> For instance, in most jurisdictions the usual rules of evidence do not apply. See eg *United States v. Fields*, 584 F.3d 313, 342 (5th Cir. 2007).

<sup>24</sup> 18 U.S.C., FEDERAL SENTENCING GUIDELINES, § 5H1.3 (Mental and Emotional Conditions) (2011).



offense'.<sup>25</sup> Further the guidelines provide that a downward departure should generally not be granted at all if, as is possible in Doe's case, the offense (or the defendant's criminal history) indicates 'a need to incarcerate the defendant to protect the public'.<sup>26</sup> Some states are more open to mitigating evidence at sentencing, but others follow the federal government's lead.<sup>27</sup>

The situation is different at capital sentencing. There, the Supreme Court has held that, given the heightened concern about reliability when the death penalty is a possibility, virtually any evidence about the offender's character should be admitted.<sup>28</sup> That stance may explain why the available data indicate that neuroscience evidence is presented as often in death penalty cases as in all other types of cases combined.<sup>29</sup> At the same time, in many states prosecutors are allowed to argue in support of the death penalty that the defendant is dangerous,<sup>30</sup> which a capital sentencing jury might well conclude is the case with an offender whose ability to control conduct is impaired. This so-called double-edged sword problem (which as indicated above, also afflicts non-capital sentencing) could lead defense attorneys to hesitate about introducing neuroscience evidence even when it appears to be strongly mitigating.<sup>31</sup>

Thus, at both trial and sentencing, several obstacles to the presentation of neuroscience evidence exist, even assuming the science itself is impeccable. That does not mean that neuroscience is usually excluded. Even when they doubt its relevance, judges may decide to allow introduction of evidence to avoid a possible appellate issue. Nonetheless, the narrowness of the criminal law's doctrines probably affects outcomes. The available data (which, admittedly, are spotty) indicate that neuroscience rarely has an impact outside of capital cases, and even there the impact appears to be minimal.<sup>32</sup>

<sup>25</sup> *Id.*, § SK2.13 (Diminished Capacity).

<sup>26</sup> *Id.* Note that, since *United States v. Booker*, 543 U.S. 220 (2005), the guidelines have been 'voluntary', so that judges are now more likely to reduce sentences even in cases that do not meet these criteria.

<sup>27</sup> See generally John F. Pfaff, *The Continued Vitality of Structured Sentencing Following Blakeley: The Effectiveness of Voluntary Guidelines*, 54 UCLA L. REV. 234, 295 (2006) (noting that, since most state judges are elected, downward departures may be less common than in federal court).

<sup>28</sup> See eg *Woodson v. North Carolina*, 428 U.S. 280, 304 (1978) ('in capital cases the fundamental respect for humanity underlying the Eighth Amendment . . . requires consideration of the character and record of the individual offender and the circumstances of the particular offense as a constitutionally indispensable part of the process of inflicting the penalty of death').

<sup>29</sup> Deborah W. Denno, *The Myth of the Double-Edged Sword: An Empirical Study of Neuroscience Evidence in Criminal Cases*, 56 B.C. L. REV. 493, 501–2 (2015) (reporting that two-thirds of the 553 cases in which the defense presented evidence on neuroscience involved capital charges); Nita A. Farahany, *Neuroscience and Behavioral Genetics in U.S. Criminal Law: A Behavioral Analysis*, 2 J. L. & BIOSCI. 485, 492 (2016) (finding that approximately 40% of cases in which neuroscience or other biogenetic information was introduced were capital cases).

<sup>30</sup> Carla Edmondson, *Nothing is Certain but Death: Why Future Dangerousness Mandates Abolition of the Death Penalty*, 20 LEWIS & CLARK L. REV. 857 (2016) (reporting that 6 death penalty states make dangerousness a statutory aggravating factor, another 11 allow prosecutors to argue dangerousness as a non-statutory aggravator, and another 2 allow the prosecutor to introduce evidence of dangerousness in rebuttal to mitigating evidence).

<sup>31</sup> See discussion of this issue, *infra* text accompanying notes 77–82.

<sup>32</sup> The only study that looks at actual success rates attributable to mitigating neuroscience claims comes from Terry Maroney, who found that even in juvenile cases, where neuroscience could be expected to have its biggest impact, 'courts tend to regard even scientifically sound claims as legally irrelevant'. Terry Maroney, *The False Promise of Adolescent Brain Science in Juvenile Justice*, 85 NOTRE DAME L. REV. 89, 94 (2009) (also stating, based on a survey of both appellate and trial level decisions, that 'despite projections, adolescent brain science has had, is likely to have, and should have only moderate impact in the courts'). See also Denno, *supra* note

Understanding why requires a closer look at the types of neuroscience evidence that might exist and how they relate to the substantive criminal law.

### FIVE TYPES OF NEUROSCIENCE EVIDENCE<sup>33</sup>

Under current law, neuroscience will probably not be useful at criminal trials or non-capital sentencing unless it can be associated with very significant cognitive or volitional impairment, and even at capital sentencing its usefulness may be a mixed bag. Yet such evidence undoubtedly will still be proffered, especially by defense attorneys. Courts, lawyers, and experts considering whether to admit, present, or testify about neurological findings should consider carefully the specific types of neuroscience evidence at issue, because not all of it is of equal value. There are at least five such categories, discussed here in ascending order of usefulness.

#### Evidence of Abnormality

The simplest, and perhaps the most common, type of neuroscience evidence proffered at trial or sentencing purports to show that the defendant's brain is different from a 'healthy' or 'normal' brain. For instance, as in Doe's case, the defendant might be diagnosed with frontal lobe disorder or a traumatic brain injury (TBI) based on the results of a brain scan obtained through structural magnetic resonance imaging (sMRI).<sup>34</sup> More specifically, the results might show, for example, both a 'reduced volume' in the frontal lobe (the area of the brain associated with executive functions such as planning, decision making, and regulation of impulses) and a 'reduced volume' in the left side of the limbic system (associated with regulation of emotions, in particular mediation of perception and action), the combination of which suggests greater impul-

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29, at 507–8 (finding a success rate of 25% for claims that counsel was ineffective due to a failure to make neuroscience-related arguments; although the author points out that this is a relatively high success rate for IAC claims, these claims typically ask only for resentencing, not dismissal, so even in the 'successful' cases the ultimate impact of neuroscience evidence is unclear). Similar findings come from Farahany, *supra* note 29, who found that between 20% and 30% of defendants who presented neuroscience evidence 'enjoyed some success on appeal', most on ineffective assistance grounds, *Id.* at 507. Farahany also states that 'while neurobiological evidence may bolster a finding of mental illness or impairment, the legal standard for insanity may still remain an insurmountable hurdle for most defendants', *Id.* at 500, and that with respect to intent, 'judges and juries tend to credit the circumstantial evidence [of premeditation] over the neurobiological'. *Id.* at 503. Cf. Paul Catley & Lisa Claydon, *The Use of Neuroscientific Evidence in the Courtroom by Those Accused of Criminal Offenses in England and Wales*, 2 J.L. & BIOSCI. 510, 519–20 (2015) (finding that, in 'most cases' in which appellants raised neuroscience claims, convictions were affirmed, and that neuroscience substantially contributed to success at sentencing in about 10% of the cases).

<sup>33</sup> The term 'neuroscience', as used in this article, is meant to apply broadly to evidence based on brain scans, including fMRI, EEG, CT, and PET scans, as well as evidence that links brain circuitry or functioning to certain types of behavior. While there may be more 'neuro' than 'science' in the instances reported here, this usage is consistent with how courts, and scholars who have looked at the courts' decisions, refer to the subject. See generally Matthew Ginther, *Neuroscience or Neurospeculation? Peer Commentary on Four Articles Examining the Prevalence of Neuroscience in Criminal Cases Around the World*, 3 J.L. & BIOSCI. 324 (2016) (making this point).

<sup>34</sup> For a summary of studies on the effect of frontal lobe damage and traumatic brain injury on executive functioning, see Sydney Goss, *Frontal Lobe Traumatic Brain Injuries and Executive Dysfunctioning*, S. Ill. U. Carbondale Research Papers, Paper 348 (2013), [http://opensiuc.lib.siu.edu/cgi/viewcontent.cgi?article=1439&context=gs\\_rp](http://opensiuc.lib.siu.edu/cgi/viewcontent.cgi?article=1439&context=gs_rp). For a description of sMRI (often abbreviated to MRI), see Roskies & Farbota, *supra* note 6, at § 20:26.



sivity.<sup>35</sup> Or a positron emission tomography (PET) or functional magnetic resonance imaging (fMRI)<sup>36</sup> might show that the individual has ‘low-resting metabolism in the amygdala and high-resting metabolism in cortical regions’, suggesting that the person is ‘vulnerable to loss of control when challenged because the amygdala, which issues the fight-or-flight signal, will be activated while the cortex, or “thinking brain,” becomes hypoactivated’.<sup>37</sup> Ideally, this evidence would be accompanied by a behavioral history of episodic temper tantrums or similar evidence of dyscontrol, as manifested in Doe’s case.

On its face, this type of evidence may seem very relevant to criminal liability. But in fact it is only weakly so. The problem with such evidence is that it fails to make the causative link between the neurological impairment and the crime. The fact that a person’s brain structure or functioning is abnormal does not mean that the abnormality contributed significantly or at all to a specific act such as a criminal offense. For one thing, a brain scan after a crime may well misrepresent what the brain was like at the time of the crime.<sup>38</sup> More importantly, determining whether a given neural defect, even if stable, contributed to a particular crime will rarely be possible,<sup>39</sup> at least in the absence of group or individual data focused on that issue (the third and fourth categories of evidence described below).<sup>40</sup> Many people who have a ‘reduced volume in the frontal lobe’ or ‘low-resting metabolism in the amygdala’, just like many people who are intellectually disabled or have TBI, never commit crime or come close to doing so, even when they manifest irrational or impulsive behavior on a routine basis.<sup>41</sup>

In some cases, the circumstantial behavioral evidence may be sufficient to overcome this problem. For instance, in one famous case, a Virginia schoolteacher who at age 37

<sup>35</sup> Ruben Gur et al., *A Perspective on the Potential Role of Neuroscience in the Court*, 84 FORDHAM L. REV. 547, 564 (2016) (describing this as a possible finding).

<sup>36</sup> For descriptions of PET and fMRI technology, see Roskies & Farbota, *supra* note 6, at §§ 20:24 & 20:28.

<sup>37</sup> Gur et al., *supra* note 35, at 565 (describing this as a possible finding).

<sup>38</sup> See Roskies & Farbota, *supra* note 6, at § 29:49 text at n.2 (‘If a defendant is scanned months or years after the act in question, and the scan detects an abnormality, it is not a simple matter to conclude with confidence that the same abnormality was present at the time in question or, even if it was, that it would have meaningfully affected behavior.’). See also Helen Mayberg, *Does Neuroscience Give Us New Insights into Criminal Responsibility?*, in *A JUDGE’S GUIDE TO NEUROSCIENCE: A CONCISE INTRODUCTION* 37, 37–39 (Andrew S. Mansfield ed., 2010) (stating the principal difficulty in trying to make inferences about criminal responsibility from brain lesions and changes in the brain is that scans are not sufficiently contemporaneous with the crime).

<sup>39</sup> SATEL & LILIENTHAL, *supra* note 2, at 150 (‘Neuroscientists cannot yet forge tight causal links between brain data and behavior.’). Most courts have agreed. See *eg* *States v. Montgomery*, 635 F.3d 1074, 1090 (8th Cir. 2011) (finding that results of PET scan had no causal relationship with defendant’s criminal behavior); *Jackson v. Calderon*, 211 F.3d 1148, 1165 (9th Cir. 2000) (PET scans did not show defendant ‘was unable to premeditate or form a specific intent at the time of the shooting’); *Coe v. Carpenter*, 2016 WL 1274599 at \*67 (W.D. Tenn. 2016) (‘current brain imaging methods cannot readily determine whether a defendant knew right from wrong or maintained criminal intent or mens rea at the time of the criminal act’); *United States v. Mezvinsky*, 206 F.Supp.2d 661, 672 (E.D. Pa. 2002) (noting the time lag between testing and the crime); *United States v. Freeman*, 2015 WL 175879 at \*5 (M.D. Fla. 2015) (same).

<sup>40</sup> In other work, I have argued that criminal defendants ought to be able to tell their ‘stories’ through psychiatric testimony, even if it is speculative. See *eg* CHRISTOPHER SLOBOGIN, *PROVING THE UNPROVABLE: THE ROLE OF LAW, SCIENCE AND SPECULATION IN ADJUDICATING CULPABILITY AND DANGEROUSNESS* 50–56 (2007). However, in these cases the expert testimony relies, or at least should rely, almost entirely on concrete evidence of cognitive or behavioral impairment in the individual in question at the time of the crime or the time of the evaluation, rather than on the individual’s ‘condition’ (*eg*, schizophrenia).

<sup>41</sup> Mayberg, *supra* note 38, at 39 (noting that while there are many studies linking brain abnormalities to behavior disorders, most persons with brain lesions do not act criminally and many criminals do not have brain lesions).

suddenly developed an interest in child pornography and eventually sexually propositioned his stepdaughter (as well as the female staff of the treatment program to which he was sentenced!) was found to have a brain tumor; after surgery, his impulsive sexualized conduct abated. When, about a year later, he began engaging in highly sexualized behavior again, doctors found that the tumor had regrown; complete removal of the tumor ended the antisocial behavior.<sup>42</sup> Somewhat less persuasive, but similarly dramatic, was the neurological evidence in the case of Charles Whitman, the sniper who killed several people at the University of Texas and upon autopsy was found to have a brain tumor encroaching on his amygdala.<sup>43</sup> But cases like these are very unusual; few defendants can present the seemingly straightforward connection between neurology and behavior that these cases provide.

In short, evidence of abnormality, on its own, is rarely material at trial to prove impairment at the time of the offense or as proof of what may have caused any impairment that the defendant asserts. In cases similar to the Virginia schoolteacher's and Whitman's, it may be relevant to an EMED argument in a homicide trial or to a volitional insanity defense in those states that recognize that doctrine. But the typical evidence of neurological abnormality provides an insufficient basis for making the necessary link to the crime. For that reason, it may not even be relevant at sentencing, at least outside the death penalty setting.<sup>44</sup> Indeed, as indicated earlier, in federal court such evidence is explicitly irrelevant at ordinary sentencing proceedings when it is also suggestive of high risk (presumably not an issue in the schoolteacher's case once the tumor was removed, but arguably a serious one if the evidence shows, as in the initial example above, that the person is 'vulnerable to loss of control when challenged').

### Cause of an Effect Research

Perhaps, however, if evidence of a defendant's abnormality can be supplemented with research showing a heightened occurrence of *antisocial or criminal behavior* among similarly impaired individuals, the defense might fare better. Defense attorneys could proffer two types of studies along these lines. The first type, discussed here, addresses what statisticians call the cause (in our case, brain abnormality) of an effect (in our case, violence).<sup>45</sup> An early example of cause-of-an-effect research in the neurology domain, replicated many times since, is a study conducted by Bryant and his colleagues.<sup>46</sup> Bryant looked at 110 inmates, 55 of whom committed violent crime and 55 of whom committed non-violent crime.<sup>47</sup> He found that 73% of the prisoners convicted of violent crime had FLD, while only 28% of those who committed non-violent crime had

<sup>42</sup> An account of this case is found at *Doctors Say Pedophile Lost Urge After Tumor Removed*, USA TODAY, June 28, 2003, [http://usatoday30.usatoday.com/news/health/2003-07-28-pedophile-tumor\\_x.htm](http://usatoday30.usatoday.com/news/health/2003-07-28-pedophile-tumor_x.htm).

<sup>43</sup> An account of this case is found in David Eagleman, *The Brain on Trial*, Atlantic (July-Aug. 2011) <http://www.theatlantic.com/magazine/archive/2011/07/the-brain-on-trial/308520/> (2011).

<sup>44</sup> See FAIGMAN ET AL, *supra* note 6, § 20:11 (concluding that '[a] review of case law suggests that these kinds of assertions [regarding brain injury] are often admitted without challenge in the penalty phase of a capital trial...').

<sup>45</sup> See generally A. Philip Dawid, *Statistical Causality from a Decision-Theoretic Perspective*, 2 ANN. REV. STAT. & APPL. 273 (2014).

<sup>46</sup> Ernest T. Bryant et al., *Neuropsychological Deficits, Learning Disabilities and Violent Behavior*, 52 J. CONSULT. & CLIN. PSYCHOL. 323 (1984).

<sup>47</sup> *Id.* at 323–24.

FLD.<sup>48</sup> Put another way, the Bryant study suggests that, among those apprehended for committing a violent crime, there is a 73% chance that the perpetrator has frontal lobe damage (FLD). Other researchers have obtained similar results.<sup>49</sup>

This type of evidence sounds highly exculpatory, at least in a jurisdiction with a volitional impairment defense or where premeditation is an issue. But, looked at closely, this kind of research tells us only that a high proportion of violent people have FLD. It does not indicate the probability that people who have FLD will be violent, which is the central question the law wants answered. Unfortunately, when defense attorneys present nomothetic (group-based) research, it is most likely to be this type of cause-of-an effect data. In related contexts, courts have rightly held that such evidence is not germane to whether defendants engaged in the kind of uncontrollable, unintentional, or highly impulsive conduct that is the focus of exculpatory doctrines.<sup>50</sup> Because this kind of research says virtually nothing about how the asserted abnormality contributed to crime, it is no more useful than evidence of abnormality by itself.

### Effect of a Cause Research

Another type of study, which compares the prevalence of criminal behavior among those with neurological impairment to those who do not have such impairment, comes closer to answering the causation question, because rather than addressing the cause of an effect, it addresses the effect (eg violence) of a cause (eg FLD). For instance, some research indicates that, all else being equal, the prevalence rate for violence among people with FLD is about 10% higher than for those who are not lobe damaged, data suggesting that FLD predisposes people to commit crime.<sup>51</sup> Studies also indicate that maltreated children, who tend to have smaller cortical volume, exhibit higher rates of adult maladaptive conduct than controls.<sup>52</sup>

In contrast to mere evidence of abnormality, this type of evidence is useful in addressing causation, because it suggests that, in a certain percentage of cases, but for the brain dysfunction the crime would not have occurred. But this type of data must be put in context. Without knowing the base rate for violence by people who do not have the relevant neurological abnormality, crime prevalence information is of little help to legal factfinders. If it turns out, for instance, that only about 2% of the general

<sup>48</sup> *Id.*

<sup>49</sup> See Katherine Harmon, *Brain Injury 7 Times Greater Among U.S. Prisoners*, SCIENTIFIC AMERICAN, Feb. 4, 2012, [www.scientificamerican.com/article/traumatic-brain-injury-prison/](http://www.scientificamerican.com/article/traumatic-brain-injury-prison/); John Matthew Fabian, *Forensic Neuropsychological Assessment and Death Penalty Litigation*, THE CHAMPION 33 (2009) (reporting a study finding that 61% of habitually violent offenders had a history of brain injuries); Richard E. Redding, *The Brain-Disordered Defendant: Neuroscience and Legal Insanity in the Twenty-First Century*, 56 AM. U. L. REV. 51, 56 (2006) (reporting a study finding brain dysfunction in 94% of homicide offenders).

<sup>50</sup> See *eg* *People v. Yuki*, 364372 N.Y.S.2d 313, 318–19 (1974) (rejecting an argument based on research showing that large numbers of prisoners have an extra Y chromosome, because ‘the experts merely suggested that aggressive behavior may be one manifestation of the XYY syndrome [and] could not confirm that all XYY individuals are involuntarily aggressive; in fact, some identified XYY individuals have not exhibited such tendencies’).

<sup>51</sup> C. Brower & Bruce H. Price, *Neuropsychiatry of Frontal Lobe Dysfunction in Violent and Criminal Behaviour: A Critical Review*, 71 J. NEUROL. NEUROSURG. & PSYCHIATRY 720, 725 (2001) (‘Based on results reviewed here, a reasonable conjecture for the increased risk of violence associated with clinically significant focal frontal lobe injury might be 10% over the base rate for a given population.’).

<sup>52</sup> See Lois A. Weithorn, *Developmental Neuroscience, Children’s Relationships with Primary Caregivers and Child Protection Policy Reform*, 63 HASTINGS L.J. 1487, 1508–9 (2012).

population commits violent crime (a probable overestimate),<sup>53</sup> the statistics described above indicating a higher rate of violence among those with FLD merely establish that 2.2% of people with FLD commit violent crime, which does not suggest a high level of impulsivity (or of cognitive impairment). More to the point, those statistics are unlikely to be influential with a judge or jury.

Some studies point to much higher prevalence rates associated with genetic traits. Perhaps most famous in this regard is a neurogenetic study carried out by Avshalom Caspi and his colleagues examining 442 men in New Zealand. Caspi found that, by age 26, roughly 85% of the men within this group who had been abused as children and had low serotonin levels (low MAOA activity alleles) committed rape, robbery, or assault, four times the average of the overall group.<sup>54</sup> This evidence suggests that just two characteristics—one environmental and one genetic—strongly predispose people to commit serious crime.

The main argument that even this evidence should not be exculpatory or have significant mitigating effect has been most forcefully put by Stephen Morse:

Whether a predisposing factor is produced by a mental disorder or by some other ‘normal’ or ‘abnormal’ cause makes no difference to whether the agent is responsible. A cause is just a cause, and causation per se is not an excuse, whether the causation is ‘normal’ or ‘abnormal.’ If causation were an excuse, no one would be responsible for any conduct, because all behavior is caused by multiple variables not within the agent’s control.<sup>55</sup>

In this view, Caspi’s study would be immaterial at trial, and perhaps of low value to the defense at sentencing as well, even assuming that dangerousness is not an offsetting consideration.

In essence, Morse’s stance is that, when gauging criminal responsibility, all causes are the same. As a doctrinal matter, however, that is not correct. The insanity defense gives exculpatory effect to a particular cause: ‘mental disease or defect’. The EMED formulation gives mitigating effect to ‘unusual ... mental disturbance’ and the federal sentencing guidelines allow downward departures for ‘mental conditions’. While perhaps based more on folk psychology and intuition than pristine philosophical tenets,<sup>56</sup> legal doctrine has long been based on a ‘medical model’,<sup>57</sup> differentiating between people who act precipitously because of ‘mental disorder’ and people who do so because they are drunk, have a strong libido, had a bad day at work, or picked on the victim because the victim reminded them of an abusive father. Studies like Caspi’s provide evidence of the distinctive cause the law seeks with its exculpatory and mitigating doctrines.

<sup>53</sup> In 2012, for instance, the FBI reported 1214,462 violent crimes, see FEDERAL BUREAU OF INVESTIGATION CRIME REPORTS, <https://ucr.fbi.gov/crime-in-the-u.s/2012/crime-in-the-u.s.-2012/violent-crime/violent-crime/>, in an adult population of over 250,000,000.

<sup>54</sup> Avshalom Caspi et al., *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 SCIENCE 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).

<sup>55</sup> Stephen J. Morse, *Uncontrollable Urges and Irrational People*, 88 VA. L. REV. 1025, 1040 (2002).

<sup>56</sup> Morse himself often resorts to folk psychology in support of his claims. See eg Stephen J. Morse, *Criminal Law and Common Sense: An Essay on the Perils and Promise of Neuroscience*, 99 MARQUETTE L. REV. 39, 50–51 (2015) (describing the congruence between the law’s approach to responsibility and ‘folk psychology’).

<sup>57</sup> See generally, Jules B. Gerard, *The Usefulness of the Medical Model to the Legal System*, 39 RUTGERS L. REV. 377, 398–403, 418 (1987).

Of course, such proof does not end the matter; all of the exculpatory and mitigating legal doctrines also have a behavioral component. Even if the defense presents strong evidence that the crime is caused by neurological abnormality, the prosecutor will often be able to point to evidence of planning, which undercuts an EMED or volitional insanity defense. The prosecutor might also emphasize that even in the Caspi cohort 15% of those studied did not commit crime, suggesting control is possible. At sentencing, the prosecutor confronted with the Caspi study might add that the same two traits that mitigate also aggravate, given their link to violent behavior.

Finally, the prosecutor can undermine effect-of-cause studies by pointing to difficulties in generalizing from a nomothetic study to the case at hand.<sup>58</sup> For instance, if prevalence research about FLD defines FLD narrowly, to require evidence of a serious defect, but the FLD of someone like Doe is minimal, it is only tangential to the defendant's case. Similarly, if most of the crimes committed by the Caspi cohort were assaults, that study may not be considered particularly germane in a murder trial.

### Individualized Neuropsychological Findings Compared Against Known Performance Baselines

A fourth type of expert testimony—one that might be combined with the third type in an effort to show the criminogenic effects of neural abnormality—would begin not with neurological findings, but with behavioral findings produced by neuropsychological testing. For instance, a neuroscientist or neuropsychologist evaluating someone like Doe might administer standardized tests that measure various domains of self-control, such as the Go/NoGo, Stop-Signal, and Card Sorting tasks, and then compare the defendant's results to baseline results for the general population.<sup>59</sup> In this way, the expert might be able to say something meaningful about a particular defendant's capacity to control conduct, and perhaps as well the defendant's ability to plan, form intent, and be cognizant of risks, issues relevant to mens rea inquiries. This type of information differs from simple evidence about abnormality and its usual effects (the first two types of evidence discussed) because it provides insight into the particular defendant's functioning, rather than the type of functioning that *might* accompany a particular brain anomaly and is *not* specific to the individual.

Several obstacles confront this approach, however. One concern that need not give us pause is the fact that, because these performance tasks involve asking the subject to choose to respond to stimuli, malingering is possible.<sup>60</sup> Fortunately, methods can be devised to overcome this problem.<sup>61</sup> In any event, as noted earlier, this article assumes that testing results validly show what they purport to show.

<sup>58</sup> On the group-to-individual 'fit' problem, see David L. Faigman, John Monahan & Christopher Slobogin, *Group to Individual (G2i) Inference in Scientific Testimony*, 81 U. CHI. L. REV. 417, 441–43 (2014).

<sup>59</sup> For a description of these tasks and limitations on their ability to discern impulsivity, see Joshua W. Buckholtz, Varlerie Reyna & Christopher Slobogin, *A Neuro-Legal Lingua Franca: Bridging Law and Neuroscience on the Issue of Self-Control*, 5 MENTAL HEALTH L. & POL'Y J. 1, 13–14, 17, 19–21 (2016) (noting the imperfect fit between performance on tasks and particular cognitive constructs, the lack of consistent results between performances on similar tasks, and generalizability issues).

<sup>60</sup> See *Id.* at 14 (noting that subjects involved in the Go/NoGo task 'are instructed to press a button each time they see a consonant, and to avoid pressing the button each time they see a vowel', an easily manipulated task).

<sup>61</sup> Gur et al, *supra* note 35, at 562 ('Often, these tests include measures of "effort" in which easy tasks are disguised as difficult, and someone who tries malingering a deficit will fail them').

Of greater concern is the difficulty of obtaining baseline data for all the relevant demographic groups needed to make useful comparisons with the defendant. Findings from neuropsychological testing, or any neurological test for that matter, can vary significantly based on gender, age, education, and other variables. Without this baseline information, it can be difficult to interpret specific results.<sup>62</sup> Another concern, already noted in connection with other types of research, is that, even if the baseline measurements can be obtained, a reading about relative impulsivity at one point in time does not necessarily say anything about impulsivity at the time and in the setting of the crime. Relatedly, much more collaboration between scientists and policymakers must take place before the assertion can be made with any degree of confidence that the results on a Go-No Go test or any similar test provide useful information about the particular impairment-at-the-time-of-the-crime question the law asks.<sup>63</sup>

Finally, of course, science cannot answer the normative question of how far below average a defendant would have to register on a particular performance task to be considered legally impaired. Should the cut-off associated with non-responsibility or mitigation be set at the bottom 25th percentile of the reference group or the bottom first percentile, at two standard deviations from the mean or three? Evaluators at the University of Pennsylvania use an algorithm purporting to translate the results of neuropsychological tests into images showing brain regions that are ‘dysfunctional’, as defined by the blue ribbon panel of experts who helped develop the algorithm.<sup>64</sup> This type of evaluation technique can standardize results and reduce bias in interpreting those results. Ultimately, however, the law, not experts, must determine how much ‘dysfunction’ is required for legal purposes.<sup>65</sup>

### Individualized Neuroscience Findings Compared Against Known Legal Baselines

In some areas, the courts have already made this determination. In *Atkins v Virginia*,<sup>66</sup> the Supreme Court exempted people with intellectual disability from the death penalty, and in a series of other cases the Court exempted juveniles from the death penalty and mandatory life sentences.<sup>67</sup> These decisions stand for the proposition that the individuals in these two groups are ‘categorically less culpable than the average criminal’.<sup>68</sup> If an expert could compare, in a legally meaningful way, the structure or functioning

<sup>62</sup> Buckholtz et al., *supra* note 59, at 21 (‘there exist no large, demographically representative datasets of the kind required for standardization of measures. This means that, as of now, even the best experimental tasks are not useful as tests for determining capacity in any individual defendant.’) (emphasis in original).

<sup>63</sup> *Id.* at 29 (‘if the law wishes, or intends to permit, the use of cognitive and neuro-scientific evidence to make inferences about legally germane aspects of mind and brain, legal policymakers must work with scientists to ensure that their constructs map on to scientific valid data’).

<sup>64</sup> Gur et al., *supra* note 35, at 562–63.

<sup>65</sup> Even in defining intellectual disability for purposes of the exemption from the death penalty, the one situation in which the Court has deferred heavily to the mental health professions in delineating responsibility criteria, the states are granted some ‘flexibility’. See *Moore v. Texas*, 137 S. Ct. 1039, 1052 (2017).

<sup>66</sup> 536 U.S. 304 (2002).

<sup>67</sup> *Roper v. Simmons*, 543 U.S. 551 (2005) (holding that execution of juveniles under 18 violates the Eighth Amendment); *Graham v. Florida*, 560 U.S. 48 (2010) (holding that the Eighth Amendment bars life without parole sentences on juveniles under 18 who are not convicted of homicide); *Miller v. Alabama*, 564 U.S. 460 (2012) (holding that Eighth Amendment bars mandatory life sentences without parole for juveniles under 18 convicted of any crime).

<sup>68</sup> *Roper*, 543 U.S., at 567 (‘today our society views juveniles, in the words *Atkins* used respecting the mentally retarded, as “categorically less culpable than the average criminal,”’ citing *Atkins*, 536 U.S. at 316).



of a defendant's brain with the average analogous results for juveniles or people with intellectual disability, the testimony could be considered highly relevant, at least at sentencing.

This use of neuroscience has been called 'scientific stare decisis'—the notion that scientifically similar groups ought to be treated the same for legal purposes.<sup>69</sup> If a 30-year-old's brain looks like or functions the same way as a juvenile's brain in relevant respects, the law ought to evaluate that adult's culpability in the same way it does a juvenile's. The Supreme Court has obliquely recognized this idea in *Sears v Upton*,<sup>70</sup> where it reversed a death sentence of a person who performed at or below the bottom first percentile in several measures of cognitive functioning and reasoning because of FLD. The Court explained that, even though the defendant did not necessarily suffer these impairments before the age of 18 (which is required under the exemption established in *Atkins*), he should nonetheless be exempted from the death penalty because 'regardless of the cause of his brain damage[,] he was among the most impaired individuals in the population'.<sup>71</sup>

The primary obstacle here is the scientific and legal challenge of identifying the neurological characteristics that should be used to make these comparisons. While the measures of cognitive functioning that impressed the Court in *Sears* might allow juxtaposing people whose impairments developed at different points of their lives, neurological data may not be similarly commensurate. In particular, reliably comparing the brain of an adult with that of a juvenile in legally relevant domains may be very difficult, given developmental differences.

However, there is some hope in this regard. In a series of preliminary studies, Kiehl and his colleagues developed a measure of 'brain age' using 19 partitions of gray matter, the volume and density of which change systematically across individuals in a manner consistent with aging processes.<sup>72</sup> After obtaining brain age MRI data from a sample of 1300 individuals, ranging in chronological age from 12 to 65, the researchers studied an independent sample for which they had recidivism data. They found that 'the *brain-age* measures outperformed *chronological age* in calculating how likely an individual was to be reincarcerated'.<sup>73</sup> In other words, criminality was more closely linked to the maturity of the brain than to the age of the person.

While this research focused on risk rather than culpability, Kiehl's group also found that the same nine brain areas—found in the anterior temporal lobes, the amygdala, and the orbital frontal cortex—were 'reasonable targets for assessing the relationship between antisocial behavior and neural function'.<sup>74</sup> This finding supports the proposition that if a particular adult's neural function can be shown to be similar to that of the average juvenile, mitigation at sentencing may be the appropriate response. Once again,

<sup>69</sup> Christopher Slobogin, *Scientizing Culpability: The Implications of Hall v. Florida and the Possibility of a 'Scientific Stare Decisis'*, 23 WM. & MARY BILL RTS. J. 415, 424–28 (2014).

<sup>70</sup> 561 U.S. 945 (2010).

<sup>71</sup> *Id.* at 949 (internal quotations removed).

<sup>72</sup> Kent A. Kiehl et al., *Age of Gray Matters: Neuroprediction of Recidivism* (The Mind Research Network, Working Paper, 2016), cited in Lyn M. Gaudet et al., *Can Neuroscience Help Predict Future Antisocial Behavior*, 85 FORDHAM L. REV. 503, 520 n. 124 (2016).

<sup>73</sup> Gaudet et al., *supra* note 72, at 520 (emphasis in original).

<sup>74</sup> *Id.* at 521.

however, because the same evidence may also indicate higher recidivism risk, advocates will need to think carefully about how to frame such neurological evidence.

## CONCLUSION

This brief survey of the extent to which neuroscience evidence might be relevant under current criminal law doctrine suggests that courts and juries could properly be dismissive of much of it simply on materiality grounds, before even beginning to evaluate its validity. In particular, simple evidence of neuroabnormalities and research about the cause of effects is of minimal relevance. At the same time, courts should give careful consideration to and welcome good prevalence research addressing the criminogenic effects of neurological aberration like the defendant's, individualized neuropsychological tests results about legally relevant impairments that are gauged on relevant reference groups, and comparisons of the defendant's brain scans with scans of populations, like juveniles, that are legally entitled to mitigation. Unfortunately, the latter three types of evidence are much more difficult to produce than the first two.

Of course, another way to increase the relevance of neuroscience is to modify legal doctrine. An incremental change, suggested by Deborah Denno, is to recognize an intermediate defense between involuntariness and full culpability that permits more lenient treatment when a person acts intentionally but with impaired consciousness.<sup>75</sup> A similar result might be achieved by construing either or both prongs of the insanity defense more broadly,<sup>76</sup> and by extending the EMED defense to all crimes.

The double-edged sword dilemma that arises when neuroscience evidence meant to be mitigating can simultaneously be characterized as indicative of risk might also be addressed. The claim has been made that prosecutors seldom exploit evidence of neurological impairment, even when it suggests that the defendant represents a high risk of reoffending.<sup>77</sup> But, anecdotally, defense attorneys involved in capital sentencing proceedings are clearly concerned about that possibility,<sup>78</sup> and prosecutors and courts have been known to use analogous mitigation evidence as a sword.<sup>79</sup> One response

<sup>75</sup> Deborah Denno, *Crime and Consciousness: Science and Involuntary Acts*, 87 MINN. L. REV. 269, 274 (2002) (arguing for a new category of criminal liability labeled 'semi-voluntary acts').

<sup>76</sup> See eg Reider, *supra* note 4 (arguing for an expansion of the insanity test to accommodate neurological findings); Redding, *supra* note 4 (same).

<sup>77</sup> Denno, *supra* note 29, at 498–99 (interpreting the results of a study of cases in which neuroscience evidence was introduced as proof of 'a criminal justice system that is surprisingly willing to accept and comprehend both the strengths and limitations of neuroscience evidence in ways that clearly discredit the myth of the double-edged sword'). It is worth noting that this study looked only at cases that were appealed, and that it did not have access to transcripts, which would have revealed prosecutorial efforts at exploiting the double-edge sword. Cf. Tennard v. Dretke, 542 U.S. 274, 289 (2004) (noting that 'the prosecutor's comments pressed exactly the most problematic interpretation of the special issues, suggesting that Tennard's low IQ was irrelevant in mitigation, but relevant to the question whether he posed a future danger').

<sup>78</sup> Richard Bonnie, *Mental Illness, Diminished Responsibility and the Death Penalty: A New Frontier*, 41 HUM. RTS. 21, 21 (2017) ('[M]any defense attorneys worry, and research has shown, that evidence of mental illness will amount to a "double-edged sword" . . .'); Jennifer Chandler, *The Use of Neuroscientific Evidence in Canadian Criminal Proceedings*, 2 J. L. & BIOSCL. 550, 569 (2015) (finding evidence of double-edged sword difficulties in Canada).

<sup>79</sup> See Edmondson, *supra* note 30, at 893 ('Many states allow the prosecution to argue that a defendant poses a future danger, but place few, if any, limitations or boundaries on those arguments. Even when a court does concede that an argument was improper, only on rare occasions does the court find the defendant was actually prejudiced by that error.')

to this concern is to ban government references to the dangerousness associated with neurological conditions, a response the Supreme Court has contemplated when the mitigating evidence is based on ‘mental illness’.<sup>80</sup> Another counter is to require an instruction reminding the jury that the only alternative to the death penalty is life without parole, alerting them to the fact no one outside of prison will be endangered by the defendant if the jury rejects the death penalty.<sup>81</sup> A third response is simply to preclude a death sentence if the relevant mitigator is established.<sup>82</sup>

A final legal reform, much more radical but one with which I am not unsympathetic,<sup>83</sup> is to re-orient the entire criminal justice system toward a vehicle for addressing the causes of criminal behavior. Prevention of reoffending would be a significant goal of such a system, but so would rehabilitation.<sup>84</sup> Neuroscience experts would focus not on blameworthiness or desert, but on risk and treatment.

Under the current retributively driven system, however, neuroscience evidence is necessarily focused primarily on culpability issues. In deciding on the relevance of such evidence to those issues, courts, lawyers, and experts need to pay close attention to the precise nature of the evidence in question and the specific legal doctrine sought to be addressed. This article has attempted to outline these nuanced considerations, in an effort to clarify thinking about how the criminal justice might best use the insights of neuroscience.

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<sup>80</sup> *Zant v. Stephens*, 462 U.S. 862, 885 (1983) (noting with approval that the state had not ‘attached the “aggravating” label to factors that are constitutionally impermissible or totally irrelevant to the sentencing process, such as for example the race, religion, or political affiliation of the defendant, . . . or to conduct that actually should militate in favor of a lesser penalty, such as perhaps the defendant’s mental illness’.).

<sup>81</sup> *Simmons v. South Carolina*, 512 U.S. 154 (1994), so held, but only when dangerousness is ‘at issue’. Commentators have argued that, because dangerousness is ‘always’ on the minds of jurors, the instruction should be given in every capital case. John H. Blume, Stephen P. Garvey & Sheri Lynn Johnson, *Future Dangerousness in Capital Cases: Always ‘At Issue’*, 88 CORNELL L. REV. 397 (2001).

<sup>82</sup> Bonnie, *supra* note 78, at 21.

<sup>83</sup> For my take on these issues, see Christopher Slobogin, *Prevention as the Primary Goal of Sentencing: The Modern Cases for Indeterminate Dispositions in Criminal Cases*, 48 SAN DIEGO L. REV. 1127 (2011); Christopher Slobogin, *The Civilization of the Criminal Law*, 58 VANDERBILT L. REV. 121 (2005).

<sup>84</sup> See Eagleman, *supra* note 43 (arguing that the criminal justice system should move from a backward-looking retributive system to one that is forward looking and preventive in orientation); Nancy Gertner, *Neuroscience and Sentencing*, 85 FORDHAM L. REV. 533, 544 (2016) (‘The “new rehabilitation”—now informed by neuroscience and evidence-based science—offers the possibility of yet another shift in American sentencing away from retribution toward an approach more finely tailored to the individual, his needs, and his future.’).