Forensic Implications of Neuroscientific Advancements

Lori L. Hauser, PhD

Im draws a parallel between the neurobiology of individuals with autism spectrum disorder (ASD) and that of trauma survivors, to explain why individuals with ASD may be more vulnerable to trauma as a risk factor for violence. This commentary raises questions about how we use this information in a forensic context, including its potential misuses. It urges caution in not overstating the data before we have a more nuanced understanding of how our neural circuitry influences specific behaviors and mental states, while not allowing the science to advance faster than we can harness it, overstepping its bounds in decisions we make regarding fairness and justice. It raises these concerns against a backdrop of the diametrically opposed assumptions about human behavior embraced by the two disciplines, mental health and the law, that come together in the forensic arena.

J Am Acad Psychiatry Law 44:193–97, 2016

Recent progress in neuroscience has advanced our understanding by leaps and bounds of how changes or differences in particular neural structures in the brain contribute to different behaviors.[1,2] In his article, Dr. Im[3] compares the neurobiology of individuals with autism spectrum disorder (ASD) with that of trauma survivors, to suggest a possible mechanism for the link between trauma and violence in individuals with ASD. Studies show that structural and functional abnormalities involving the prefrontal cortex, the frontal and temporal cortices, and the limbic system are contributors to the social cognition, emotion regulation, and repetitive behaviors characteristic of ASD. As a result of these deficits, individuals with ASD have difficulty in identifying and understanding others' thoughts, feelings, and behavior, contributing to problems with empathy and attachment. They tend to have high autonomic reactivity and to lack the capacity for cognitive flexibility and adaptability. Because they fail to appreciate context or to integrate information in understanding events as conceptual wholes, they are prone to anxiety, distress, and frustration intolerance. Their stereotyped, repetitive behaviors and self-stimulation are therefore viewed as a means of self-soothing, a flooding of affect in someone without the capacity to make sense of it.

Im points out that trauma survivors show similar structural and functional abnormalities in these same regions (i.e., the prefrontal (orbitofrontal) cortex, the anterior cingulate cortex and the amygdala (in the limbic system), and the hypothalamic-pituitary-adrenal (HPA) axis) that put them at a heightened risk for aggression and violence. These deficits contribute to a poor concept of self; a lack of attachment, connectedness, or sense of belonging among others; an inability to regulate emotions; and a lack of empathy. Im posits that, given the overlap in brain dysfunction between the two (individuals with ASD and trauma survivors), individuals with ASD may be particularly vulnerable to the effects of trauma, as they may experience a "network overload" (Ref. 3, p 12) where the already sensitized prefrontal-cortical-limbic network (the diathesis) is further compromised by an overwhelmingly distressing event (the stress). Unable to make sense of their social worlds, to regulate the affect flooding their system, or to consider alternatives to violence, they may act out in aggressive ways.

Granted, Im acknowledges that his theory is just that: a theory, lacking empirical confirmation. Nonetheless, it is grounded in the scientific literature on trauma, violence, and ASD and itself

Dr. Hauser is a licensed clinical psychologist, Whiting Forensic Division, Connecticut Valley Hospital, PO Box 70, O'Brien Drive, Middletown, CT 06457. E-mail: lori.hauser@ct.gov.

Disclosures of financial or other potential conflicts of interest: None.

draws on numerous established theories for the relationship between brain functioning and the resultant behavior. There are some questions left unanswered by his theory, however. Is trauma even a necessary part of the equation? Do these brain dysfunctions not leave one more prone to violence in the first place, by virtue of what we know about the specific capacities they affect and the specific behaviors associated with them? Why are some individuals with ASD violent, whereas others are not, given that they have similar brain abnormalities and dysfunctions? If behavior could be reduced to simple brain-based causality, what accounts for the fact that individuals with similar dysfunction behave differently? Beyond these basic, theoretical questions, however, perhaps the question we should be asking ourselves is: what do we do with this information, once we obtain it?

A Brief Aside: The State of the Science

Recent advances in neuroscience make it compelling to apply such knowledge in a forensic context. As early as 1981, computed tomographic (CT) scan evidence was admitted in the John Hinckley, Jr. case to illustrate that Hinckley had widened sulci, a cardinal marker for schizophrenia, despite the fact that it was unknown whether this had anything to do with his mental state at the time of the crime, which was the forensic question at hand.[1] As the field has advanced over the past several decades, neuroimaging has been introduced as evidence in other forensic contexts as well, including culpability, aggravating or mitigating circumstances, and risk management in the criminal arena, and tort claims or functional decisional capacities in the civil arena.[4]

Despite these advancements and the temptation to apply our growing knowledge, many would consider the state of the field of neuroscience to be still in its infancy. Critics contend that there is a dearth of findings that pin a specific brain scan pattern to a specific psychiatric disorder, much less with the more complex cognitive and behavioral functions underlying many legal debates.[4] In other words, it is one thing to say that a scan shows reduced blood flow to the anterior cingulate area or structural differences in the orbitofrontal region. It is another to say that those differences mean that someone lacked the capacity to appreciate the wrongfulness of his conduct. Problems with defining what is normal, with creating predictive models, and even with the technology itself preclude our ability to state with a reasonable degree of medical certainty that specific brain pathology even correlates with, much less causes, specific behaviors or mental states.[4] However, it is distinctly possible (perhaps even likely) that continued advancements in neuroimaging and neuroscientific research will someday yield greater sophistication in our understanding of just how specific brain abnormalities contribute to certain behaviors and to mental states such as intentionality, appreciation, and moral judgment. So, again, what do we do with this information?

Science and the Law: A Cultural Divide

Scientists and philosophers have long wrestled with what is known as mind/body dualism (what some have called the *Cartesian impasse*), which is essentially, where the boundary lies between free will and determinism. This debate (which is not even settled within disciplines) takes on an entirely new dimension when applied to the law, where fundamental assumptions about human behavior widen even further. According to the law, human behavior is the product of free will or rational choice, whereas neuroscience reduces cognition, emotion, and behavior to cold, mechanistic brain functioning. Morse asserts, "brains do not commit crimes, people commit crimes" (Ref. 5, p 397), noting that brain-based causation does not absolve one of criminal responsibility. Humans are viewed as intentional agents, and it is our behavior (not our brains) that determines responsibility. Yet, neuroscientists fire back that behavior is determined by the brain, making it (and therefore, us) the true agent. These fundamental assumptions about the nature of human behavior collide when mental health experts are summoned to the courtroom to shed light on some forensic question.

Different priorities across these disparate disciplines also mean that the gaze is sometimes focused in different directions: in psychology and psychiatry, causality often is the cardinal interest, and usually it is seen as a complex interplay of biological and environmental factors, whereas for law it is the effect of a particular behavior that is most important.[1] Although the philosophical "clash of cultures" often pulls us to think of things in terms of polar opposites, both sides of the debate likely can agree that the answer lies somewhere in the middle. We are likely no more devoid of the myriad external influences in our lives (and hence, not entirely free) than we are mechanistically reduced to a complex weaving of

neural circuitry (and hence, not entirely determined). To ask the question one more time, against this philosophically different backdrop of law, what do we do with our scientific knowledge?

Returning to Im's theory, the juxtaposition of ASD and violence elicits many questions. What are the forensic implications of such a theory? Might this mean that we should not hold individuals with ASD criminally responsible for violent acts, because their complex brain dysfunction prevents them from appreciating right from wrong or from controlling their behavior once triggered? Should they be deemed ineligible for certain punishments, like the death penalty, as other groups of developmentally immature individuals (for instance, the intellectually disabled, and adolescents) have been so excluded? If a specific brain dysfunction is said to exculpate one's behavior, is this true for all individuals with a similar affliction? If not, why not? Should the data stand alone, or should they be interpreted in a moral and sociopolitical context that considers the impact on society's standards of decency? A closer look at how certain neuroscientific research has been applied in the forensic context may help to illustrate the complexities (and the potential pitfalls if we do not exercise caution in how we do it).

The Prefrontal Cortex and Its Influence on Behavior

In *Roper v. Simmons*,[6] the United States Supreme Court held that juveniles (that is, offenders under the age of 18 when they committed their crime) were ineligible for the death penalty under the Eighth and Fourteenth Amendments. The Court reasoned that adolescents' vulnerability to negative influences, poor impulse control, and overall developmental immaturity make them less culpable than their adult counterparts; thus, inflicting this most extreme form of punishment on them does not serve the fundamental elements of justice. This decision relied in part on an *amicus* brief, filed by the American Psychological Association (APA), citing neuroscientific research that showed incomplete maturation of the frontal lobes, and particularly the prefrontal cortex, in adolescents.[7]

The prefrontal cortex, the most anterior of the frontal lobe divisions, is considered to be "the most complex, selectively derived neurological feature" of the brain (Ref. 2, p 220). It has long been known to be central to numerous higher order cognitive (or executive) functions, including: behavioral motivation, impulse control, emotional processing and intelligence, recognition of conventional behavior, goal-setting and -directed behavior, analysis of reward and punishment (and avoidance of the latter), complex problem solving, and task-relevant attention and persistence. Damage to this area (or, failure to develop in the first place) thus leads to amotivation and apathy, lack of empathy, poor social judgment, impulsivity, failure to benefit from operant conditioning, and inability to organize and execute a set of behaviors aimed toward task completion. According to the APA brief, the adolescent brain is more prone to risk-taking, less capable of weighing the advantages and disadvantages of a particular course of action (especially one that involves proximal advantages and distal disadvantages), and more vulnerable to social influence than is the adult brain. One "hallmark of frontal lobe dysfunction is difficulty in making decisions that are in the long-term best interests of the individual" (Ref. 7, p 10). Thus, citing the Court's own reasoning in *Atkins v. Virginia*, the brief argued that, given their less developmentally mature brains, the imposition of the death penalty on adolescents "does not serve the judicially recognized purposes of the sanction . . . [and thus amounts to] purposeless and needless imposition of pain and suffering" (Ref. 8, p 319).

That analysis seems reasonable. The prefrontal cortex is one of the core structures Im[3] refers to in his analysis of how trauma relates to violence, as well as in his theory of how individuals with ASD are particularly vulnerable to the impact of trauma. Both groups show dysfunction in this region that impairs their social–emotional capacities and narrows their range of appropriate behavioral responses; but what about other conditions, with brain dysfunction and resulting behaviors that can be (and has been) demonstrated through neuroscience? Numerous studies suggest that psychopaths show dysfunction in the prefrontal cortex similar to that of adolescents (or individuals with ASD, or trauma survivors). Would (or should) these same proscriptions extend to that condition?

The Neurobiology of Psychopathy

Psychopathy is a personality and behavioral disorder characterized by shallow affect and reduced autonomic reactivity; a lack of empathy or concern for others; a conning, manipulative interpersonal style; and impulsive, antisocial, and often criminal, behav-

ior. Although most criminals are not psychopathic, psychopaths make up about 20 percent of the prison population and commit an inordinate number of violent acts compared with their nonaffected counterparts.[9] Research suggests that the core features of this disorder (the affective and interpersonal traits) are to various degrees biological in nature, tied to specific abnormalities in the brain.[10]

Specifically, psychopaths are said to have damage to areas of the prefrontal cortex (contributing to impulsivity, irresponsibility, poor decision-making, and deficient emotional information processing); deficits in the orbitofrontal/ventromedial area of the prefrontal cortex (contributing to disinhibition, impaired moral decision-making, and failure to process adequately reward and punishment); deficits in the dorsolateral prefrontal cortex (contributing to response perseveration in the face of punishing consequences, and failure to direct attention to relevant emotional cues); damage to the superior temporal cortex and the amygdala–hippocampus complex (contributing to rule-breaking behavior, moral judgment deficiency, and failure to avoid punishment); damage to the superior temporal gyrus (contributing to a lack of empathy for others or concern for one's actions and noncompliance with prosocial rules); reduced volume in the amygdala and in the amygdala–hippocampus complex (contributing to deficits in perspective taking or empathy, emotional intelligence, and fear conditioning); and damage to the anterior cingulate cortex (contributing to disinhibition, perseveration, and poor emotion regulation[10–17]). In short, neuroimaging studies have provided support for the theory that psychopaths have deficits in various areas of the prefrontal cortex, temporal structures, and the amygdala and other areas of the limbic system that contribute to their deficits in emotional intelligence (aligning with others', as well as regulating their own), impulse control, fear-based conditioning, and moral decision-making. These deficits in turn make them more likely to harm or exploit others, to engage in behavior that most others would avoid because of its consequences, and to feel no remorse or shame for behavior that repeatedly violates the rights of others. So, do these brain-based deficits make them less responsible for their actions, and less deserving of punishment, in the same way that adolescents' yet-to-develop prefrontal cortex makes them less responsible for their conduct?

Of course, this is not to say that adolescent and psychopathic brains are exactly alike, any more than the brains of individuals with ASD are exactly like those of trauma survivors. Clearly, numerous factors play a role in the execution of any behavior or mental state. However, given the similarity in brain structure and function among different behavioral expressions, it raises the question: if policymakers at the nexus of psychology and law are going to argue that specific brain dysfunction can absolve one of (or at least, soften one's) culpability, why is that true of one group (adolescents) and not of another (psychopaths)? What causes us to recoil at the very notion of suggesting that these individuals would be anything other than wholly responsible for the violence and damage that they perpetrate?

These comparisons are not meant to suggest that this author believes that psychopathy is a disorder akin to ASD, or that it should be the basis for a diminished capacity defense, or that such individuals should not be punished for their wrongdoings on account of their "bad brains." Indeed, some might argue that individuals with ASD are not responsible for their crimes because of deficits A, B, and C, yet those same deficits may make us perceive a psychopath to be more responsible, more at risk, or more deserving of punishment. Perhaps it is not just those specific deficits, but the presence of capacities X, Y, and Z (present in psychopaths but not ASDs) that make us hold them more reprehensible and thus more legally culpable. Nor are they meant to imply that even individuals who share the same constellation of symptoms or behaviors (that is, diagnostic categories) should be treated similarly. Every case is different, and we cannot make such sweeping generalizations that all individuals with ASD should not be held culpable for crimes, the same way that we do not make the claim that all individuals with intellectual disabilities, or dementia, or other brain dysfunctions are similarly inculpable. Rather, the questions and points raised herein are meant to stimulate thought and discussion, to encourage others to think through how our science is used, before we have the capacity to use it. If we are going to rely on neuroscience to back up our arguments as to why certain individuals are less culpable or less deserving of punishment, we ought to be prepared to explain why other individuals with similar deficits are not; else we should not pretend that it is because of the brain dysfunction itself. More broadly, if we embark on a path of using

neuroscience, not just to inform the state of reality (for instances, one's capacities with respect to various decision-making contexts or in comparison to their functioning preinjury), but also to integrate it with our principles of fairness and justice to make psycholegal judgments about people's accountability, liberties, and in some cases, lives, we must give thoughtful consideration to just how those judgments are made.

Conclusion

Im[3] adds to our understanding of violence by summarizing the neuroscientific findings and theoretical underpinnings regarding specific brain dysfunctions and ASD and by hypothesizing a mechanism for the link between trauma and violence in individuals with ASD. We, as a science, will continue to wrestle with complex questions regarding the relationship between neural circuitry and behavior. As our science advances, we, as psycholegal scholars, are going to be forced to bring this knowledge into the forensic arena, whether by defense attorneys arguing that their clients are not responsible for their criminal actions, or by parole boards attempting to make determinations regarding risk management, or by probate courts wrestling with questions regarding an individual's basic freedoms or decisional capacities. We will be confronted with complex questions involving mental states, intentionality, free will, culpability, and risk of recidivism. We must resist the temptation to apply these findings prematurely, before we have a more nuanced understanding of the complex network of factors that weave together to influence behavior in each unique case.

At the same time, we must not allow our science to advance faster than we can harness it; we must think through the ways in which we use it in combination with our principles of fairness and justice to shape a better society, one that continues to embrace evolving standards of decency. In other words, the state of the science is but one part of the equation. We should not dismiss these hard-to-answer questions simply because the data remain uncertain. Equally important is the way in which we integrate the data to make judgments in the forensic context, especially when it comes to what should and should not happen with people's lives. At some point, the science will catch up, and we will be forced to decide how it should be applied in alliance with our principles of justice and morality.

References

1. Batts S: Brain lesions and their implications in criminal responsibility. Behav Sci & Law 27:261–72, 2009
2. Knabb JJ, Welsh RK, Ziebell JG, *et al*: Neuroscience, moral reasoning, and the law. Behav Sci & L 27:219–36, 2009
3. Im, DS: Trauma as a contributor to violence in autism spectrum disorder. J Am Acad Psychiatry Law 44:184–92, 2016
4. Martell DA: Neuroscience and the law: philosophical differences and practical constraints. Behav Sci & Law 27:123–36, 2009
5. Morse SJ: Brain overclaim syndrome and criminal responsibility: a diagnostic note. Ohio State J Crim L 3:397–412, 2006
6. *Roper v. Simmons*, 543 U.S. 551 (2005)
7. Brief for the American Psychological Association and the Missouri Psychological Association as Amici Curiae Supporting Respondents, Roper v. Simmons, 543 U.S. 551 (2005) (No. 03-633)
8. *Atkins v. Virginia*, 536 U.S. 304 (2002)
9. Hare R D: The Hare Psychopathy Checklist, 2nd Edition-Revised. Toronto: Multi-Health Systems, 2003
10. Anderson NE, Kiehl KA: Psychopathy: developmental perspectives and their implications for treatment. Restor Neurol Neurosci 32:103–17, 2014
11. Boccardi M, Frisoni GB, Hare RD, *et al*. Cortex and amygdala morphology in psychopathy. Psychiatr Res 193, 85–92, 2011
12. Kiehl KA, Smith AM, Hare RD, *et al*: Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. Biol Psychiatry 50:677–84, 2001
13. Muller JL: Psychopathy: an approach to neuroscientific research in forensic psychiatry. Behav Sci& Law 28:129–47, 2010
14. Nickerson SD: Brain abnormalities in psychopaths: a meta-analysis. North Am J Psychol 16:63–78, 2014
15. Yang Y, Raine A, Colletti P, *et al*: Abnormal structural correlates of response perseveration in individuals with psychopathy. J Neuropsychiatry Clin Neurosci 23:107–10, 2011
16. Yang Y, Raine A, Narr KL, *et al*. Localization of deformations within the amygdala in individuals with psychopathy. Arch Gen Psychiatry 66:986–94, 2009
17. Yang Y, Raine A, Colletti P, *et al*: Morphological alterations in prefrontal cortex and amygdala in unsuccessful psychopaths. J Abnorm Psychol 119:546–54, 2010