

Current Controversy: Aggression and the brain: structure, function, and criminal responsibility.

Before John Hinckley tried to assassinate Ronald Reagan the requirements for an insanity defense generally included an impairment in either impulse control (“impaired volition”) or knowledge of right and wrong. Afterwards, most states modified their laws such that either the criteria for an insanity defense was narrowed with respect to what “impaired volition” meant, or this criterion was removed from consideration, or the insanity defense itself was eliminated. The well known neuroscientist, Robert Sapolsky, suggests that “contemporary neuroscience argues strongly against” the view that “an inability to tell right from wrong should be the sole basis of an acceptable insanity defense.” He further argues that data on the brain suggests we should bring back “impaired volition” as grounds for an insanity defense. Here, you will be asked to review and evaluate some of the contemporary data on brain function in relation to aggression, and then discuss their implications with regards to some issues of social policy including the position taken by Sapolsky.

Part I

Data about brain mechanisms influencing aggressive behavior have come from two related sources, the study of brain structures involved in aggression, and the study of transmitter systems and genes associated with them. There are two alternative series of questions below. The first focuses on brain structures and pathways and the second on the gene for monoamine oxidase A (the MAOA gene) and serotonergic mechanisms. Please choose one of these and address the questions under that section. Think carefully about your choice because you will use this perspective throughout the exam. We recommend that you read the entire question first before you begin. Part I should be roughly 8 pages in length. After you have answered A or B of Part I, continue on to Part II, which should be roughly 5-7 pages. Suggested page lengths exclude references.

A. Brain structures

Our understanding of brain structures associated with aggression comes from animal experimentation as well as study of humans with brain disorders. Much attention has focused on the roles played by the amygdala and the frontal cortex.

1. Describe the neural circuitry connecting parts of the cortex and amygdala that are implicated in aggressive behavior in **non-human** animals, and critically evaluate the relevant data implicating this circuit(s) in aggression. Be explicit about any specific sub-regions of the cortex and amygdala that are believed to be important for aggressive behavior.
2. Critically discuss the evidence that these same brain areas are involved in aggressive behavior in **humans**. More specifically, describe the key findings that have come from studies of each of the following three classes of people: (a) those with brain disorders (e.g. those associated with seizures, tumors, or other kinds of damage to these areas), (b) healthy people with subclinical antisocial personality disorder, and (c) individuals with clinically diagnosed antisocial personality disorder.

Or:

B. Monoamine oxidase A and serotonergic systems

Monoamine oxidase A (MAOA) is an enzyme that breaks down monoamine transmitters in the synaptic cleft. Animal studies have revealed that MAOA can influence aggression, and variants in the promoter region of the gene that encodes MAOA in humans have been associated with some indices of aggression. In addition, there is considerable evidence that serotonergic systems in the forebrain modulate aggression in humans as well as non-human animals.

1. Review and critically evaluate the key pharmacological and molecular evidence from **non-human** animal models suggesting that monoamines and serotonin are critical mediators of aggressive behavior. Discuss the extent to which you are convinced by it, and what you see as the most likely roles that monoamines and serotonin play in mediating aggressive behavior. Be sure to include information on: the type of aggressive behavior being studied, the brain regions where these changes are thought to take place, genetic background of the model, and conditions under which the aggressive behaviors are studied.
2. Examine and critically evaluate the data implicating these transmitter systems in **human** aggressive behavior. Discuss what kinds of conclusions we can draw from these data.

Part II

There are many ethical issues associated with aggression and what to do about it, and the issue here is whether our understanding of its biology should influence the decisions we make as a society about how to deal with these problems. Two important issues regarding aggression are structured around questions of **culpability and intervention**. For example, should the criminal justice system take into account the structure and/or function of the frontal cortex when decisions are made about the nature of punishment for violent crimes? Should policies be the same for an individual whose frontal lobe was damaged in war and one whose frontal lobe simply did not develop normally? Should children that may be at risk for heightened aggression because of the MAOA/5-HT genetic variation be provided special early training in impulse control or receive mandatory pharmacological treatment? **Choose either the issue of culpability or the issue of intervention, and identify the main ethical question and present your argument (either pro or con)**. Be sure to couch your answer in the context of **what is and is not known about the biology involved (that you discussed in Part I)**. In addition, make sure that you anticipate the **counterarguments, including those that have been spelled out in the literature by other scientists, when you argue for your position**.

Part I—Brain structures

Aggression and violence are a major problem in the world. While preventing aggression and violence altogether is unlikely, understanding the neural mechanisms underlying these behaviors may improve the problem. The neural foundation of aggression in humans resembles that of animals, such as cats and rodents, and the types of aggression displayed by these animals mimic the types of aggression seen in humans. Both animals and humans display affective aggression (also known as reactive, defensive, or hostile aggression) and predatory aggression (also known as proactive premeditated, or instrumental aggression) (Siegel and Victoroff, 2009).

Animal experiments

Animal experimentation has brought about a better understanding of brain structures that are associated with aggression. Two of the key players in the neuroanatomical control of aggression are the amygdala and the frontal cortex, and the connections between these two areas, namely the hypothalamus.

In 1928, a researcher by the name of Hess demonstrated his ability to induce aggressive behavior in cats by electrical stimulation of the hypothalamus. Eleven years later, a landmark study demonstrated that lesions to the medial temporal lobe abolished fear and aggressive behaviors in monkeys (Klüver and Bucy, 1939), which gave rise to the condition known as 'Klüver-Bucy syndrome'. These studies served as the foundation for the challenge of unraveling neuroanatomical substrates for aggressive behaviors. As a follow-up to these studies, researchers in 1956 tamed various male species (agoutis, domestic cats, lynxes, and rhesus monkeys) with bilateral lesions to the amygdala (Schreiner and Kling, 1956). The 'aggression

area' of the cat and laboratory rat was later described through the use of stereotaxic methods and precisely movable electrodes (Kruk et al., 1979, Kruk et al., 1983, Lammers et al., 1988, Siegel et al., 1999). Aggressive behaviors can be evoked by electrical stimulation of the amygdala, hypothalamus, periaqueductal gray (PAG), and the area around the fornix in the diencephalon, and electrical stimulation of the lateral aspect of the prefrontal cortex, lateral septum, or basomedial amygdala can inhibit some of these aggressive behaviors (Siegel et al., 1975, Albert and Walsh, 1984, Siegel et al., 1999).

A functionally defined part of the mediobasal hypothalamus (now commonly referred to as the 'hypothalamic attack area') plays a key role in the control of aggressive behaviors in animals. Electrical stimulation of this area in rats and cats consistently reduces biting attacks, and lesions to this area reduce aggression against conspecifics in territorial settings (Adams, 1971, Olivier et al., 1983). Injection of a retrograde tracer, which labels all afferents, into the mediobasal hypothalamus of rats, demonstrated that the 'hypothalamic attack area' has three distinct afferent cell populations: (1) a telencephalic midline "plate" that contains neurons from the orbitofrontal and medial prefrontal cortex, neurons from septal regions and the bed nucleus of the stria terminalis, (2) a temporal column comprised of neurons from the medial amygdala, amygdalo-hippocampal area and the subiculum, and (3) a local hypothalamic network. Afferents were also present in the brainstem nuclei, in the PAG, paralemniscal region, dorsal raphe, and locus coeruleus (Toth et al., 2010). The densest projections to the 'hypothalamic attack area' originated in the prefrontal cortex, specifically the ventromedial portion. This area is specifically activated during aggressive encounters (Halasz et al., 2006), and damage to this area is implicated in human aggression (Brower and Price, 2001). The lateral

septum formed another major input to the 'hypothalamic attack area'. When stimulated, as mentioned previously, this area inhibits aggressive behavior (Albert and Walsh, 1984). The medial amygdala, medial part of the bed nucleus of the stria terminalis (BNST), and the ventral subiculum also formed a major inputs to the 'hypothalamic attack area', which is in congruence with previous findings that lesions to these temporal regions provide a taming effect in animals and humans (Kluver and Bucy, 1939, Schreiner and Kling, 1956, Emery et al., 2001, Mpakopoulou et al., 2008). In cats, the hippocampus regulates aggression via projections to the perifornical lateral hypothalamus and the midbrain PAG, and these regions mediate both predatory attack and defensive rage (Gregg and Siegel, 2001). Therefore, many of the analogous brain structures implicated in human aggression (discussed below) are afferents to the 'hypothalamic attack area' in rats.

A study published this year in Nature elicited aggressive responses in mice by optogenetically stimulating an area of the hypothalamus. This area, the ventrolateral subdivision of the ventromedial hypothalamus (VMHvl), was identified by immediate early gene mapping (Lin et al., 2011). Optogenetic, but not electrical stimulation of the VMHvl elicited an aggressive attack. This 'hypothalamic attack area' in the mouse is more discrete than the 'hypothalamic attack area' identified in rats, which might suggest a species difference, or the fact that electrical stimulation acts on both axons-of-passage and neuronal somata, while optogenetic stimulation of the VMHvl activates only neuronal somata (Lin et al., 2011). Optogenetic stimulation of the rat 'hypothalamic attack area' has yet to be performed, but this novel technique could greatly improve our understanding of the aggression in both animals and humans.

The 'hypothalamic attack area' plays a key role in the control of aggressive behaviors, and similar hypothalamic structures exist in all species studied so far, including humans (Ramamurthi, 1988). Many of the inputs to the 'hypothalamic attack area' are implicated in human aggression, particularly the cortex, limbic system and subcortical structures, including the amygdala. While animal research can greatly improve our knowledge of aggression, neuroimaging and disorders affecting the aggression circuit have also provided much insight into the underlying neurobiology of aggression.

Human experiments

Nearly two hundred years ago Phineas Gage survived a terrible accident in which an iron rod was driven completely through his head, damaging his orbital frontal cortex (Damasio, 1994). After this accident, Gage's personality had changed enough that his family and friends had claimed that he was 'no longer Gage'. This unfortunate accident gave insight regarding particular regions of the brain controlling particular aspects of personality. Gage's behavioral problems following his accident concurs with several other individuals with damage to the same brain region (Damasio, 1994). People with brain disorders such as temporal lobe epilepsy, and individuals with antisocial personality disorder display heightened levels of aggression.

A mounting body of evidence suggests that aggressive behavior is a component of various clinical disorders associated with dysfunction of the temporal lobe, such as temporal lobe epilepsy (TLE). People with temporal lobe epilepsy can show outbursts of aggressive behavior (Serafetinides, 1965, Ounsted, 1969), violence with little provocation (in the case of a patient who stabbed a stranger after he was accidentally bumped) (Mark and Sweet, 1974), interictal aggression (Falconer, 1973) and episodes of aggressive hypersexuality following

seizure (Arnedo et al., 2009). In a study comparing aggressive or non-aggressive TLE patients with healthy control subjects, a subset of individuals with temporal lobe epilepsy who were also aggressive displayed very severe amygdala atrophy, with volumes < 3 SD below the mean (van Elst et al., 2000). A quantitative magnetic resonance imaging (MRI) study comparing 24 aggressive TLE patients with 24 non-aggressive TLE patients and 35 control subjects, found that aggressive TLE patients had decreased prefrontal grey matter compared with non-aggressive TLE patients and controls (Woermann et al., 2000). The brains of non-aggressive TLE patients did not differ from healthy control subjects in grey matter volume or amygdala volume. These data suggest that the previously mentioned brain abnormalities in aggressive TLE patients may underlie the pathophysiology of aggression.

Antisocial personality disorder (ASPD) is a behavioral disorder characterized by a pervasive pattern of disregard for, and violation of, the rights of others (American Psychiatric, 2000). Currently there is an intense debate as to whether or not a separate diagnosis for psychopathy should exist. Neither antisocial personality disorder, as defined in the *Diagnostic and Statistical Manual of Mental Disorders- fourth edition (DSM-IV)*, or its corresponding diagnosis in the *International Classification of Diseases- tenth edition (ICD-10)*, identifies psychopathy as a separate diagnosis. Therefore, for the remainder of this discussion I will include psychopathy data along with antisocial personality disorder data.

It is estimated that 5% of males are characterized by a pattern of antisocial behavior and that these men commit 50-70% of all violent crimes (Moffitt, 1993, Tiihonen et al., 2008). A study by Adrian Raine and colleagues investigated brain abnormalities in 41 murderers that pled not guilty by reason of insanity. Positron emission tomography (PET) brain imaging found

that murderers showed reduced glucose metabolism in the prefrontal cortex, superior parietal gyrus, left angular gyrus, and the corpus callosum, as well as activity asymmetries (left lower than right) in the amygdala, thalamus, and the medial temporal lobe (Raine et al., 1997).

Therefore, the brain dysfunction seen in murderers pleading not guilty by reason of insanity might motivate and predispose these individuals to violent acts. Using these same subjects, Raine and colleagues investigated glucose metabolism using PET in the left and right medial and lateral prefrontal cortex, as well as the amygdala, midbrain, hippocampus, and thalamus to compare predatory and affective murderers. Affective murderers had lower left and right prefrontal functioning, higher right hemisphere subcortical functioning, and lower right hemisphere prefrontal/subcortical ratios, compared to controls. Predatory murderers, on the other hand, had excessively high right subcortical activity and had prefrontal functioning that did not differ from controls (Raine et al., 1998).

One of the first studies to identify a structural difference in the brains of men with diagnosed ASPD and healthy men used structural MRI to compare 21 men that had a diagnosis of ASPD, and 27 men who were substance-dependent, with 34 men who had neither ASPD nor substance dependence. Men with diagnosed ASPD showed an 11.0% reduction in prefrontal grey matter volume, compared to healthy men, and reduced autonomic activity during a social stressor (Raine et al., 2000). A possible confound in this study is that some of the sampled men with ASPD also were schizophrenic, although because of this the authors created a psychiatric control group that were matched with the 21 subjects in the ASPD group on schizophrenia-spectrum disorders, affective disorders, anxiety disorders, and other personality disorders that did not fall under the category of ASPD. As a follow-up to this study, Raine and colleagues used

MRI to determine structural and functional measures of the corpus callosum. Comparing 15 men with diagnosed ASPD and high psychopathy scores with 25 matched controls, they found that psychopathic antisocial individuals showed a 22.6% increase in estimated callosal white matter volume, a 6.9% increase in callosal length, and a 15.3% reduction in callosal thickness (Raine et al., 2003). These results may indicate a neurodevelopmental dysfunction in individuals with ASPD. In other species it has been identified that approximately two-thirds of callosal fibers are postnatally pruned (LaMantia and Rakic, 1994), thus an increase in callosal fibers may indicate a lack of proper postnatal pruning, and possibly a developmental arrest.

Another study used magnetic resonance imaging volumetry and voxel-based morphometry to compare brain volumes between 26 persistently violent offenders with diagnosed ASPD and substance dependence against 25 healthy men. The violent offenders had larger white matter volumes, bilaterally, in the occipital and parietal lobes, and in the left cerebellum, and larger grey matter volume in the right cerebellum, compared to healthy men (Tiihonen et al., 2008). A possible confound in this study is that all of these violent offenders were also substance abusers. Individuals with conduct disorder (a prerequisite for antisocial personality disorder) may abuse drugs and alcohol at an early age, leading to an interruption of synaptic pruning that typically occurs during puberty (Blakemore and Choudhury, 2006). Prefrontal and temporal grey matter loss is also correlated with substance abuse (Lyyo et al., 2006). Therefore, the brain abnormalities seen in these patients could be the result of substance abuse. These data suggest that brain abnormalities observed in people with antisocial personality disorder might underlie aggressive tendencies in these individuals.

As mentioned above, part of the hippocampus is involved in the aggression circuit. Laakso and colleagues used MRI to compare regional hippocampal volumes in 18 habitually violent offenders with ASPD and type II alcoholism, with 34 age-matched control subjects and found a strong negative correlation between psychopathy scores and regional volumes of the posterior half of the hippocampi, bilaterally (Laakso et al., 2001). Interestingly, lesions to the dorsal hippocampus impair associative learning (Sziklas et al., 1998) and psychopathic individuals show abnormal or absent neurophysiological responses compared to controls, in studies of aversive conditioning (Laakso et al., 2001).

People suffering from ‘subclinical’ antisocial personality disorder are sometimes referred to as ‘successful psychopaths’, in that they share many of the same features as antisocial individuals but lead successful, non-incarcerated lives. These people are (in theory) politicians, CEOs, lawyers, and doctors—they are callous and lack empathy, but their scores of impulsivity and irresponsibility do not differ from other normal community members (Mullins-Sweatt et al., 2010). It is of worth to investigate the structural or functional abnormalities in these individuals, as it may give insight into underlying deficits that may contribute to the impulsivity seen in ‘unsuccessful psychopaths’ which leads to incarceration.

The first study characterizing successful psychopaths found that these people were similar to institutionalized or unsuccessful psychopaths, but they did not show frontal deficits or an unwillingness to delay gratification the way unsuccessful or institutionalized psychopaths do (Widom, 1977). The authors speculated that the autonomic dysfunction found in psychopaths compared to controls might only be pertinent to unsuccessful psychopaths, and not successful psychopaths. Concurring with this notion, Ishikawa and others in 2001 found that

successful psychopaths do not show the same neurophysiological or psychophysiological abnormalities that are seen in unsuccessful psychopaths. Twenty-six controls, 16 unsuccessful psychopaths, and 13 successful psychopaths were assessed on their executive functioning (using the Wisconsin Card Sorting Test), psychophysiological measures during a stressful emotional manipulation, and childhood stressors such as domestic violence, sexual abuse, and parental absence (if someone other than their parents raised them). Unsuccessful psychopaths showed reduced cardiovascular responsivity during an emotional stressor, compared to successful psychopaths and controls. Interestingly, compared to both unsuccessful psychopaths and controls, successful psychopaths showed *greater* executive functioning, significantly greater cardiovascular responsivity during an emotional stressor and *more* parental absence during childhood (Ishikawa et al., 2001).

Raine and colleagues used structural MRI in 16 unsuccessful psychopaths, 13 successful psychopaths, and 23 control subjects to compare left and right hippocampal volumes. Unsuccessful psychopaths showed an exaggerated structural hippocampal asymmetry, with the right side larger than the left, compared to both successful psychopaths and controls (Raine et al., 2004). A another study using MRI to compare prefrontal grey and white matter volumes in 16 unsuccessful psychopaths, 13 successful psychopaths and 23 control subjects found that unsuccessful, but not successful psychopaths, had a 22.3% reduction in prefrontal grey matter volume compared to controls (Yang et al., 2005). Finally, Yang and others used volumetric segmentation, cortical pattern matching, and surface-based mesh modeling methods to investigate prefrontal and amygdala structures in 16 unsuccessful psychopaths, 10 successful psychopaths, and 27 control subjects. Successful psychopaths did not differ from controls,

however, unsuccessful psychopaths showed decreased grey matter volume and thickness in the orbitofrontal cortex and the middle frontal cortex, and a volume reduction in the amygdala (Yang et al., 2010). Unsuccessful, but not successful, psychopaths show volume reductions in key brain regions that regulate behavioral control and aggression.

Together, these results demonstrate that unsuccessful psychopaths show executive dysfunction, reduced cardiovascular responsivity while anticipating an emotional stressor, hippocampal asymmetries, and prefrontal grey matter deficits, reduced orbitofrontal and middle frontal cortex grey matter volume and thickness, and reduced amygdala volume *compared to successful psychopaths*. Because successful psychopaths do not show deficits in prefrontal grey matter, they might be capable of better behavioral inhibition than unsuccessful psychopaths. Also, since they do not show hippocampal asymmetries the way unsuccessful psychopaths do, they might be less predisposed to aggression.

Part II—Culpability

Much research has been done in an attempt to uncover the neural mechanisms underlying aggression. As with any other field of research, while a great deal of progress has been made, many unanswered questions remain. Currently there is an intense debate as to whether or not neuroscience can help courts assess criminal responsibility. One person writes “Matching neurological data to legal criteria can be much like performing a chemical analysis of a cheesecake to find out whether it was baked with love” (Aharoni et al., 2008). Nevertheless, I believe that our understanding of the biology of aggression should influence the decisions that we make as a society, and that brain abnormalities, whether developmental or injury-induced,

should be taken into consideration when determining an individual's culpability of a violent crime.

Functional imaging has shown that aggressive individuals show different activation patterns than non-aggressive individuals. In the case of temporal lobe epilepsy patients, individuals with TLE that are aggressive have reduced amygdala volumes and decreased prefrontal grey matter volumes, compared to *other TLE patients that are not aggressive* and control subjects. These data certainly complicate matters regarding culpability. Is it the decrease in amygdala volume and/or the decrease in prefrontal grey matter that predispose these individuals to displays of aggression? Because an individual is predisposed to aggression does it mean that they should not be held responsible for the crimes that they commit?

Animal studies have shown that stimulation of certain areas of the brain such as the 'hypothalamic attack area' reliably elicits attack responses. While much of the neural mechanisms are conserved between non-human animals and humans, humans exhibit much more thorough executive functioning than do animals. Animals do not have to abide by moral and criminal laws—humans do. Perhaps the brain abnormalities seen in aggressive individuals demonstrate that they are more like animals in the respect that aggressive behaviors are more of a reflex—they cannot control these behaviors. Maybe these individuals *do* know right from wrong but maybe, under certain conditions, their brains lacked the neural mechanisms that would inhibit a normal person from carrying out such aggressive acts. Therefore, I agree with Robert Sapolsky in that "impaired volition" should be brought back as grounds for insanity.

In 2005, the Supreme Court ruled that it is unconstitutional to inflict the death penalty on adolescents under the age of 18 (Sowell et al., 2001). This ruling was partly based on the fact

that complete myelination of neurons in the prefrontal cortex does not occur until between the ages of 18 and 25, and therefore, these individuals may have impaired volition, may underestimate risks, and might not be able to regulate their emotions because their brains have not fully matured (Yang et al., 2008). Those with prefrontal deficits are essentially in a state of arrested development; they have impaired volition and they underestimate risks. Their prefrontal deficits put them at a disadvantage regarding behavioral inhibition, and their hippocampal abnormalities predispose them to aggressive behavior.

The brain regions that are associated with regulating aggression and impulsivity are dysfunctional in patients with antisocial personality disorder. Neuroimaging has allowed us to peak inside the brains of violent individuals. Particularly of interest regarding issues of culpability is the finding that predatory murderers (those who murdered in cold blood with purposeful, planned aggression) do not differ from control subjects in prefrontal functioning, but affective murderers (those who murdered in a way that was uncontrolled, unplanned, emotional and reactive) show deficits in prefrontal functioning (Raine et al., 1998). Excessive subcortical activity can predispose an individual to aggressive and violent acts, but without the proper prefrontal functioning, affective murderers cannot control their impulses. Further evidence that antisocial individuals are (in a sense) in a state of arrested development comes from data showing that ASPD individuals with high psychopathy scores have a corpus callosum abnormalities: 22.6% increase in estimated callosal white matter volume, a 6.9% increase in callosal length, and a 15.3% reduction in callosal thickness, compared to matched control subjects (Raine et al., 2003). Ten years ago the scientific community found out that individuals with antisocial personality disorder have an 11.0% reduction in prefrontal grey matter volume

compared to control individuals (Ishikawa et al., 2001). These individuals are simply lacking the area of their brains that allows them to make good decisions.

I believe that a great amount of insight can be gained regarding volition and culpability by comparing the brains of successful psychopaths with unsuccessful psychopaths. In all the cases that I am aware of, successful psychopaths do not differ from controls in the above-mentioned studies, except that successful psychopaths showed *greater* executive functioning, experienced more parental absence as a child, and showed *greater* autonomic reactivity during an emotional stressor compared to controls and unsuccessful psychopaths (Ishikawa et al., 2001). These successful psychopaths, while they share many of the same behavioral traits as unsuccessful psychopaths, can seemingly control themselves enough to stay out of jail or prison, even though these two groups of people did not differ on rates of self-reported drug abuse, theft, or violence. The brain dysfunction seen in unsuccessful psychopaths may therefore point to impaired volition. Yang and colleagues found that unsuccessful psychopaths had a 22.3% reduction in prefrontal grey matter compared to successful psychopaths and control subjects (Yang et al., 2005). These prefrontal impairments decrease behavioral inhibition, leading to impaired volition. This decrease in behavioral inhibition, paired with hippocampal abnormalities, which predisposes an individual to inappropriate, aggressive, psychopathic behaviors, results in an aggressive individual with inhibited inhibition—an unsuccessful psychopath.

Many researchers have concerns using brain imaging in the legal system to determine criminal responsibility. One of the main concerns raised by researchers (Mobbs et al., 2007, Vincent, 2011) is that imaging cannot tell what a person was thinking at the time of the violent

act. However, the fact that imaging cannot give insight into what a person was thinking at the time of a violent act is irrelevant in regards to whether or not that person should be held responsible for that crime. What an individual was thinking during a crime is irrelevant to whether or not that person could control their act, based on the neural circuitry that controls that person's behavior. Neuroimaging *can* however give insight into the neural circuitry controlling impulsivity and aggression in an individual, and one can elucidate that person's culpability based on those findings. If a person shows prefrontal deficits, including reduced prefrontal grey matter volume, along with corpus callosum abnormalities, reduced amygdala volume, or hippocampal asymmetries, this person might have increased aggression paired with impaired volition and therefore, reduced culpability. Another common concern with using imaging in the court system is that brain images might be too influential to jurors, who might be swayed by the brightly colored images (Mobbs et al., 2007). Instructing jurors and judges on the implications and limitations of brain imaging can ease this concern. Eastman and Campbell claim that there is a mismatch between the questions the legal system asks and the answers that neuroscience can provide. Neuroscience can only make statements about increased likelihoods of a certain behavior based on that person's association to a certain group of people with similar attributes (Eastman and Campbell, 2006). In a way this is true, nevertheless neuroimaging evidence can be regarded as evidence the same way other biological, psychological, or psychosocial evidence is used in courts to determine an individual's culpability.

While no one is saying it, I believe one of the main reasons that people are so upset with individuals like John Hinckley for receiving the verdict of 'not guilty by reason of insanity' is that

it is just not fair. We want individuals that do something wrong to feel guilty and to feel remorse, but the fact of the matter is we cannot make antisocial and psychopathic individuals feel badly for what they have done, even if we give them a 'guilty' verdict. And, even harder for people to stomach, is the fact that these violent acts might not be these people's fault, but instead a result of, for example, dysfunctional brain development. Human behavior is extremely complicated, but understanding the underlying neurobiology of certain behaviors such as aggression can give insight into their behavioral and emotional capabilities. Studies in both animals and humans have shown that abnormalities in the amygdala, prefrontal cortex, and the connections in between these two structures, including the hippocampus and hypothalamus, can lead to abnormal aggressive behaviors and impaired volition.

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Student 6
Grade: Pass

This exam was highly successful. The committee felt that your review of the brain structures associated with aggression was thorough and clearly written. Furthermore, we felt that in the second half of the exam, you espoused a clear opinion regarding culpability and a theory for why s/he believes that impaired volition should be used as grounds for legal defense. In the future, however, we would encourage you to work on synthesizing the information that you read. At times, your review in Part I read as a laundry list of scientific findings. Please remember that it is your job, as a writer, to integrate and synthesize this information to make your argument clear and to make it easier for your reader to understand your argument.