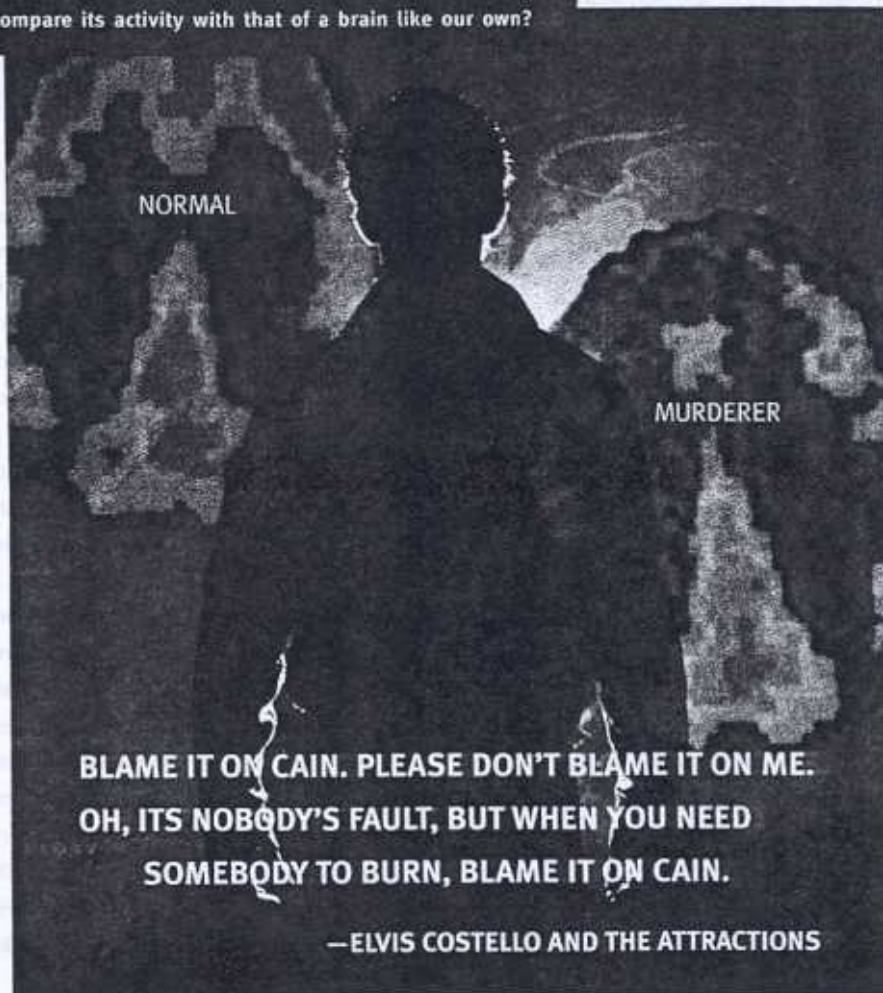


Some of our debates seem eternal, like the frozen figures on a Grecian urn. Point: Criminals must be held responsible for their crimes; we want no excuses, no sob stories. Counterpoint: Our behavior is shaped by complex, often overpowering factors; let our understanding temper our vengeance. What happens to these positions when technology enables us, for the first time in history, to look into the brain of the murderer and compare its activity with that of a brain like our own?

by Adrian Raine, D. Phil.



*Murderous Minds: Can We See
the Mark of Cain?*

Adrian Raine, D.Phil., a leading researcher on the biosocial bases of violent behavior, used positron emission tomography (PET) in the first brain-imaging study of the brains of murderers. Comparing their brains with those of a matched group of normal individuals, and exploiting PET technology's ability to measure differences in function in many brain areas, he asked questions that philosophers, theologians, and legal theorists have pondered for centuries.

If you could literally look into the mind of a murderer, what would you see? Would the activity of Jeffrey Dahmer's brain look like yours or mine? Would his brain be much less active? More active? And how would the brain activation profile of serial killers like Dahmer differ from those of less memorable, but more common, single killers? Not long ago, answers to questions like these were the province of pulp fiction or space-age movies. Now advances in brain imaging are giving us far more than a glimpse into the mind of the murderer. Although studies are in their infancy, they not only provide a base for future research into violence and aggression but raise provocative, important questions about free will, blame, and punishment.

22 MURDERERS: THE FIRST LOOK

In the first published brain-imaging study of murderers,¹ my colleagues and I scanned the brains of 22 murderers who had pleaded not guilty by reason of insanity, or had been judged incompetent to stand trial. We compared them with the brains of 22 non-murderers matching the murderers in sex

"INTIMIDATING BUT COOPERATIVE" MURDERERS

Until I began this research, there had never been a brain imaging study of murderers. This is not surprising, given the difficulty of recruiting and testing a substantial number of such individuals. But one reason I emigrated from England to California in 1987 was that, in addition to the good weather, there were plenty of murderers and other violent offenders in California who could be recruited into research studies.

Credit for recruiting the unusual sample I studied goes to my colleague Monte Buchsbaum (then at the University of California, Irvine). The subjects were identified through referrals from defense attorneys. Because California has the death penalty, their clients will die unless mitigating circumstances such as an abnormal PET scan revealing brain abnormalities can be found. Monte had the wisdom to ensure that all cases went through exactly the same procedures, thereby allowing a unique and sizable research sample to be developed.

All the subjects signed human consent forms approved by the University's Institutional Review Board. There are always ethical concerns related to research on institutionalized populations. Can they truly give informed consent? Do the benefits outweigh the risks? In these cases there was a compelling reason to participate. It was literally a life-or-death choice. The risk of exposure to a radioactive isotope in PET is more than outweighed by the possible benefit of avoiding the death penalty.

So, complete with shackles and guards, they trooped into the PET scanning facility. They looked formidable, intimidating, and ominous, but in reality were cooperative. We forget that, for 99 percent of their lives, murderers are just like us. Tragic actions in the other 1 percent of their lives set them apart. As we shall see, the structure and functioning of their brains may also set them apart.

and age. The scanning technique we used was positron emission tomography (PET), a method pioneered in psychiatry by Monte Buchsbaum. This enabled us to measure the metabolic activity of different regions of the brain, including the prefrontal cortex (which sits above the eyes and immediately behind the forehead).

Let me briefly describe our method. In PET, first a cyclotron makes a short-lived radioactive isotope, fluorine-18 (its short half-life holds down the dose of radiation to the subject). The isotope is then “tagged,” or mixed, with an analogue of glucose-2-deoxyglucose. The resulting compound is injected into the subject right after he begins a cognitive task that “challenges” or activates the part of the brain one suspects may be dysfunctional. In this case, we used the “continuous performance task,” a standard visual task known to activate the frontal region of the brain, since we hypothesized that poor functioning of the prefrontal region might be particularly involved in predisposing to violence. In this task, numbers from 0 to 9 are flashed on a screen at the rate of one per second, with subjects pressing a button every time they see “0.” This goes on for 32 minutes and, believe me, it is a very boring task. It requires the subject to maintain focused attention and mental vigilance for a sustained period. The prefrontal region of the brain is responsible, in part, for this vigilance.

While subjects were performing the task, we took a series of blood samples from them to measure glucose and deoxyglucose. Because glucose is the only source of energy used by the brain, the radioactive fluorine

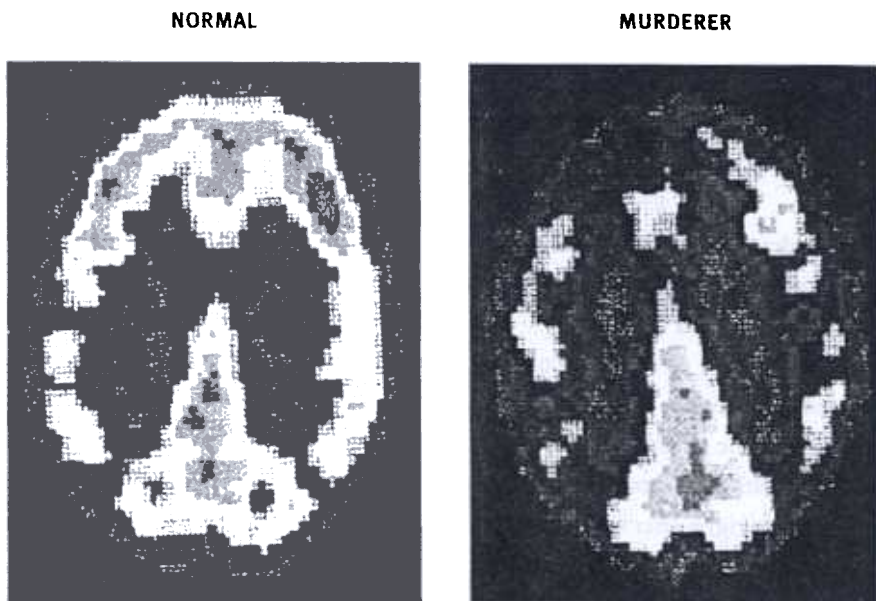
isotope is quickly carried to the brain, where the most active regions receive the largest quantities in order to meet their high energy needs. By the end of the 32-minute cognitive task, the radioactivity is “fixed” in the subject’s brain.

The subject was then taken to the PET scanner. In this procedure, crystals positioned around the head detect gamma photon rays that are emitted by the isotope, while a computer maps the origin of the emitted rays. Because more rays are emitted by brain regions that are more active (and absorbed more radioactive glucose), the activity levels of different brain regions can be picked up by the scan. And because radioactivity is mostly “fixed” in place by the end of the cognitive task, the measurements during scanning are of the levels of activity at the time of the earlier task—not afterward when the subject is in the scanner.

A PREDISPOSITION TO VIOLENCE

What did the study reveal? The key finding is illustrated in the images on the next page, which show the brain scan of a normal individual used as a control (left) and the brain scan of a murderer (right) who had impulsively killed his victim.

The striking difference is at the top of the images. The normal subject, on the left, shows much activation, while the murderer, on the right, shows little. As we had hypothesized, the area showing the difference is the prefrontal cortex. At the bottom of each scan, however, you can see that the occipital cortex is activated about equally in the normal person and in the murderer. (This brain area is activated because the task was visual.)



Brain scan (PET) of a normal control (left) and a murderer (right), illustrating the lack of activation in the prefrontal cortex in the murderer. The figures are a transverse (horizontal) slice through the brain, so you are looking down on the brain. The prefrontal region is at the top of the figure, and the occipital cortex (the back part of the brain controlling vision) is at the bottom. Warm colors (e.g., red and yellow) indicate areas of high brain activation; cold colors (e.g. blue and green) indicate low activation.

Thus the deficit in the murderers was selective. We found no deficit in the temporal region and, as is indicated in the illustration, the occipital cortex was at least as well activated in the murderer as in the control.

What does this mean? We think that poorer functioning of the prefrontal cortex predisposes an individual to violence in several ways:

- At a neurophysiological level, reduced prefrontal functioning can result in loss of control over evolutionarily older subcortical structures deep in the brain—such as the amygdala—that are thought to give rise to aggressive feelings.
- At a neurobehavioral level, prefrontal damage has been linked with risk taking, irresponsibility, rule-breaking, emotional and aggressive outbursts, and argumentative behavior—all of which predispose to violent criminal acts.
- At a personality level, frontal damage in neurological patients is associated with impulsiveness, loss of self-control, immaturity, lack of tact, inability to modify and inhibit behavior appropriately, and poor social judgment. These, too, could predispose to violence.
- At a social level, the loss of intellectual flexibility, problem-solving skills, and

reduced ability to use information provided by verbal cues—all resulting from prefrontal dysfunction—can impair social skills essential for formulating nonaggressive solutions to fractious encounters.

- At a cognitive level, poor reasoning ability and divergent thinking, both results of prefrontal damage, can lead to school failure, unemployment, and economic deprivation, which may set the stage for a criminal and violent way of life.

Note that although there are many associations between poor prefrontal function and violence, brain dysfunction may create only a predisposition to violence, which environmental, psychological, and social factors may enhance or diminish.

GOING DEEPER

We took our imaging research another step by increasing our sample from 22 to 41 murderers, and also by increasing our control group to 41. This larger sample gave us more statistical power to detect group differences. In 1997 we reported our updated findings,² which, first of all, confirmed the significant reduction in prefrontal activity in murderers.

We found that the brain structure known as the left angular gyrus functioned more poorly in the murderers. The angular gyrus lies at the junction of the temporal, parietal, and occipital regions of the brain, integrating information from the three lobes. Diminished activation in the left angular gyrus has been correlated with reduced verbal ability,³ while damage to this region has been linked to deficits in reading and arithmetic. Such cognitive deficits could predispose to

educational and occupational failure, in turn predisposing to crime and violence. (One interesting confirmation of this interpretation is that learning deficits are unusually common in violent offenders.)

We found reduced functioning of the corpus callosum, the band of white nerve fibers that allows communication between the left and the right cerebral hemispheres. We speculate that poor connection between hemispheres may mean that the right hemisphere, which is involved in the generation of negative emotion,⁴ may be less well regulated and controlled by the inhibitory processes of the more dominant left hemisphere. This may contribute to how and whether violence is expressed. Interestingly, rats stressed early in life are right-hemisphere-dominant for mouse killing.⁵ Severing the corpus callosum in these rats results in an increase in mouse killing,⁶ indicating that the left hemisphere normally acts to inhibit the right hemisphere's mouse-killing tendency. Furthermore, researchers have observed inappropriate emotional expression and inability to grasp long-term implications in human split-brain patients (who have had their corpus callosum surgically severed). This suggests that such emotional expression by violent offenders, and their lack of long-term planning, may result in part from poor functioning of the corpus callosum. Callosal dysfunction, though unlikely to cause aggression, may contribute to violence in those with other limbic and cortical abnormalities.

We observed unusual functioning in subcortical regions, including the amygdala, hippocampus, and thalamus. Murderers

tended to show relatively lower activation of these structures in the left hemisphere but higher activation in the right hemisphere. Interpretations of these findings are speculative, but it is not surprising that these structures function abnormally in murderers. The amygdala has been repeatedly associated with aggressive behavior in both animals and humans.⁷ As part of the neural network that underlies the processing of socially relevant information, its disruption could explain the anti-social behavior of some violent people and the misappraisal of ambiguous stimuli in social situations that have potential for violent encounters.⁸

The amygdala, hippo-campus, and prefrontal cortex are involved in governing emotion; together with the thalamus, they are critical to learning, memory, and attention. Their abnormal functioning may contribute to the failure to learn from experience that characterizes criminal and violent offenders.⁹ Also, the amygdala seems to mediate the experience of fear in animals, and autonomic arousal in humans. Abnormalities in the amygdala could accord with the fearlessness theory of violence, since offenders have been found to have reduced autonomic arousal.¹⁰

FACT OR ARTIFACT?

Could all of these findings have been produced by some difference between the murders and controls other than the factor of violence? We think not. Although six of the murderers were schizophrenic, we controlled for that by including six nonviolent schizophrenics in the matched normal group. Nor did group differences in brain

functioning correlate with differences in age, sex, handedness, history of head injury, medications, or illegal drug use.

Could the scans of the murderers have revealed poorer prefrontal functioning because the murderers could not do the continuous performance task? We checked this; their performance was almost identical to that of the controls. But this parity in performance on a challenge task raises still another question. How could the murderers do as well as the controls if the murderers had a dysfunction in the part of the brain critical to performing the task? The answer, we think, lies in the occipital cortex, which was more activated in murderers than in controls. The murderers seem to have recruited this visual brain area into vigorous action to help them to perform the visual task, thus compensating for their poor prefrontal functioning.

We believe this study is the first evidence from brain imaging that the brains of a large sample of murderers are functionally different from those of normal people. Prefrontal deficits have also been found in schizophrenia and depression, but those studies have never revealed our specific pattern of findings involving the prefrontal cortex, corpus callosum, angular gyrus, amygdala, hippocampus, and thalamus. This suggests a unique PET "signature" of the brains of some murderers.

What were the limitations of our study? For one thing, our sample was highly selective, consisting of murderers pleading not guilty by reason of insanity—an unusual subgroup of violent offenders but one important in forensic psychiatry. We do not

yet know if violent offenders in the community who commit serious but nonlethal violent acts also have prefrontal dysfunction, nor have we established causality. It is possible, for example, that prefrontal dysfunction does not cause violence; instead, living a violent life (including substance abuse and fights) may cause the brain dysfunction we observed. No previous study, however, has shown that substance abuse and head injuries produce the specific profile of brain deficits that we observed. Finally, although we controlled for schizophrenia, other psychiatric disorders in the control group could have contributed to the findings. But again, this specific pattern of brain deficits has never been reported in any psychiatric group.

Is the predatory killer more controlled and regulated in his brain functioning, and is the murderer who kills in a moment of passion—the “affective” murderer—lacking in brain regulation and control?

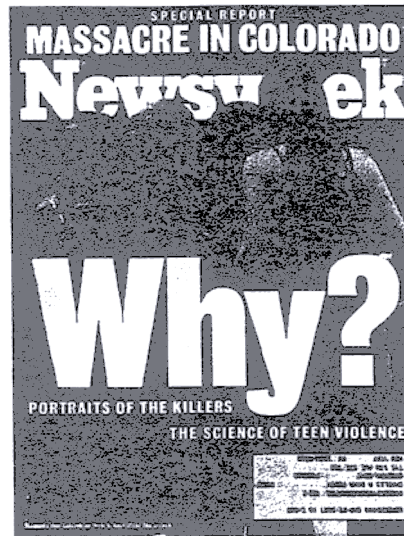
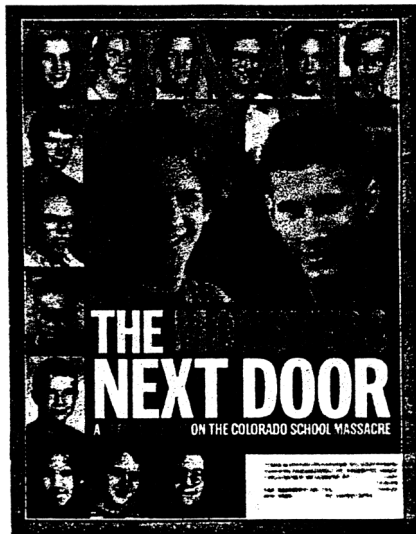
If brain deficits directly contribute to violence, what causes the deficits? What are the roles of environment and genetics? Although we controlled for a history of head injury, more subtle injuries early in life could contribute to brain dysfunction in the murderers. For example, we know that violent offenders are more likely to have grown up in abusive homes. If a baby is repeatedly and roughly shaken, the white fibers that link the prefrontal cortex with other brain structures can be lacerated, effectively cutting off the

rest of the brain from prefrontal regulatory control. Could early infant abuse have contributed to prefrontal deficits in the murderers? Drug and alcohol abuse may also contribute. Conversely, the real culprit may be genetics. Although definitive studies have not been conducted, there could be a heritable basis to prefrontal functioning. Behavioral and molecular genetic studies increasingly suggest that crime and violence are rooted in genetics.

IN COLD BLOOD

Newspapers regularly refer to the “cold-blooded” predator who kills with little or no emotion. Such a criminal is contrasted with the passionate, hotheaded individual who kills in a moment of unbridled emotion. Is the predatory killer more controlled and regulated in his brain functioning, and is the murderer who kills in a moment of passion—the “affective” murderer—lacking in brain regulation and control? Animal research has established that different neural pathways underlie predatory aggression and affective or defensive aggression. Now imaging research in humans is beginning to suggest the same thing, at least with respect to homicide.

With Reid Melloy, a forensic expert from the University of California, San Diego, we developed a way to divide our murderers into either “predatory” or “affective.”¹¹ The predatory murderers were regulated and controlled, were lacking in emotional affect, were more likely to attack strangers, and tended to plan their murders. In contrast, the affective murderers showed much less planning. Their acts were more



When sudden, murderous violence strikes, we ask: How could this happen? The murderers seemed just like us; how could they do this? Will the answers be found in the brain?

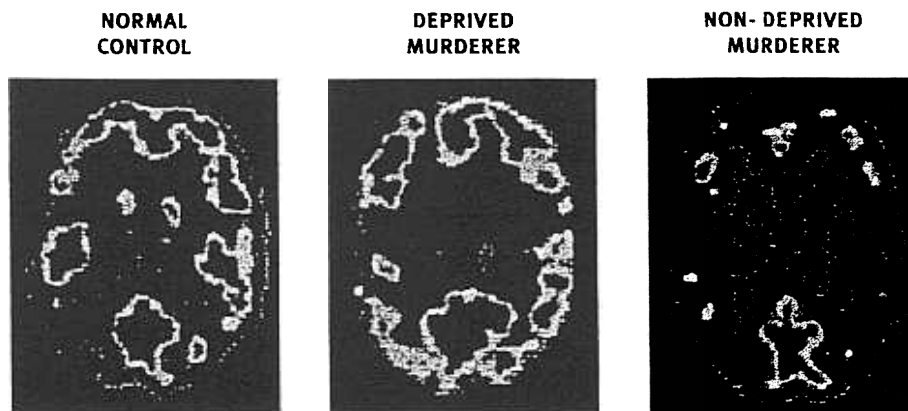
likely to occur in the home and be driven by a high degree of emotion. Although some murderers could not be assigned with confidence to either category, we were able to assign 15 of the 41 murderers to the predatory group and 9 to the affective group.

Our study found that affective murderers lacked the prefrontal functioning that can control aggressive impulses. Conversely, the regulated, controlled, predatory killers showed relatively good prefrontal functioning; an intact prefrontal cortex seemed to enable them to regulate their behavior for nefarious ends. This does not explain, however, why the predatory murderer is murderous in the first place. Does the explanation lie submerged in subcortical structures? Both groups of murderers had higher functioning of the right subcortex (defined

as the midbrain, amygdala, hippocampus, and thalamus) than did controls. We speculate that this excessive subcortical activity may predispose to an aggressive temperament in both groups; but while those in the predatory group have sufficiently good prefrontal functioning to regulate their aggressive impulses (bullying and manipulating others to achieve their desired goals), affectively violent individuals lack prefrontal modulatory control, resulting in more impulsive, dysregulated, aggressive outbursts.

GOOD AND BAD HOMES

On Tuesday, March 24, 1998, Andrew Golden (age 11) and Mitchell Johnson (age 13) killed 5 and wounded 10 of their schoolmates in the heartland of America. A year earlier, Jeremy Strohmeyer, a high-



Brain scan (PET) of a normal control (left), a murderer from a deprived home background (middle), and a murderer from a good home background (right). Prefrontal dysfunction is particularly characteristic of murderers from good home backgrounds.

school student from Long Beach, California, killed a seven-year-old girl in a restroom in Nevada. In many cases, murderers come from backgrounds of neglect, abuse, or poverty (“bad homes”), and it is easy to point an accusing finger at these possible causes of violence. But the young killers in these two cases came from homes (“good homes”) that, although not perfect, were not typical of the homes of those who now share their prison cells. In these cases, is the real culprit not poor family functioning but poor brain functioning?

We tested this hypothesis by dividing our sample of murderers into those from relatively good home backgrounds and those from relatively bad homes.¹² To do so, we took into account early physical abuse, sexual abuse, neglect, extreme poverty, foster home

placement, having a criminal parent, severe family conflict, and divorce. On this basis, we identified 12 murderers as having experienced significant psychosocial deprivation; we viewed 26 as having experienced minimal deprivation.

The results are in the illustration above, which shows the scan of a normal control (left), a murderer from a bad home (middle), and a murderer from a relatively good home (right). The odd man out is the murderer from the good home. While the deprived murderer shows relatively good prefrontal functioning, the non-deprived murderer shows the characteristic lack of prefrontal functioning. We found that murderers from good homes had a 14.2 percent reduction in functioning of their right orbitofrontal cortex. This brain area is of particular interest.

When previously well-controlled adults suffer damage to the orbitofrontal cortex, they begin to display personality and emotional deficits that parallel criminal psychopathic behavior, or what Antonio Damasio and colleagues term “acquired sociopathy.”¹³

At first, these findings seemed unexpected, but from another perspective they made sense. If a seriously violent offender comes from a bad home, it is plausible to seek the causes of his violence there. If he comes from a good home, however, environmental causes seem less plausible. Biological deficits become a more likely explanation. Consistent with our findings through brain imaging, previous research has shown that lack of a normally developed sense of fear in schoolchildren is more often coupled with antisocial behavior if the child comes from a good home environment.¹⁰ That is, the biological deficit (poor development of fear) is found in those who display antisocial behavior but lack a social predisposition to it. Not surprisingly, the right orbitofrontal cortex seems important in fear conditioning.

HOW “FREE” IS OUR WILL?

What are the social implications of these brain-imaging studies? Do all of us have freedom of will in the strict sense?

Many theologians, philosophers, and even scientists would argue that, barring exceptional circumstances (such as severe mental illness), we each have full control over our actions. Theologians, for example, suggest that we can elect to let God into our souls, choose whether or not to commit sin, and must therefore accept that criminal

actions (or sins) are products of a will under our full control. Other scientists and philosophers eschew the idea of a disembodied soul with a free will. Francis Crick, for example, believes that free will is nothing more than a large assembly of neurons and that it would be possible to build a machine that would believe it had free will.¹⁴ Perhaps we are nothing more than gene machines that mislead ourselves into believing we have choices in life.

I argue for a middle ground. Some people have almost complete freedom of will in their actions; others have relatively little. Instead of viewing “intent” in all-or-nothing terms as (with few exceptions) does the law, I see a continuum. Early social, biological, and genetic developments play substantial roles. For some, freedom of will is constrained early in life by forces beyond their control.

When people speak of free will, they point to our overriding sense of having the power to choose at this instant: to drink from a glass or drop this issue of *Cerebrum*. Surely this demonstrates free will? But take a different example. We know that alcoholism is a disease with a substantial genetic component. If we seat an alcoholic and a nonalcoholic in front of a glass of beer, then yes, in an absolute sense they can choose whether or not to drink it. But we also know that the probability is that the alcoholic will be less able to resist drinking. In this situation, his free will is constrained by genetic, biological, and environmental forces beyond his control. Probably we will never be able fully to predict behavior, but we can predict it to some extent and that suggests constraints on

freedom of will. Decisions made by a computer's hardware are produced by its software program and decisions that our brains make in specific situations are products of our unique genetic coding and early life experiences.

If brain deficits make a person more likely to commit violent crime, and if the cause of the brain deficits was not under that person's control, then should he be held fully responsible for his crimes? Suppose that a baby (we'll call him Elvis) is born in difficult labor to a mother who used drugs and alcohol during pregnancy. This prenatal and perinatal trauma causes the infant to be born with some brain damage, which is made worse by the father's vigorously shaking the baby during his bouts of non-stop crying. The baby's whiplash injury leads to disconnection of the prefrontal cortex from the rest of the brain, disengaging the emergency brake on impulsive, aggressive behavior. The impulsiveness and lack of reflection induced by the brain dysfunction lead the growing child to be more likely to fall out of trees, be hit by cars, and thereby suffer further head injury that leads to more brain damage. The brain damage renders the adult Elvis less likely to be able to control his aggressive feelings, so that one day he shoots a friend in a fit of temper. In his panic, leaving the scene of the crime, he shoots a passerby who attempts to intervene, killing him also. A PET brain scan reveals substantially poorer functioning of the prefrontal cortex and the corpus callosum in Elvis.

Should we execute him? We strongly suspect that brain damage made him much more likely to commit violent acts. We

ascertain that the causes of the damage were early in life and beyond his control. Of course, we have to protect society, and unless we can treat this brain dysfunction we may need to keep Elvis in secure conditions for the rest of his life, but does he deserve to lose his life, given the demonstrable constraints on his free will?

In debating this issue at a conference on Neuroscience and the Human Spirit in Washington, DC, in September 1998, one scientist responded to this scenario by arguing that if a person has risk factors he has to take responsibility for them. Like a person at

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risk for seizures who decides not to drive a car, the person at risk for violence must take steps to ensure that he does not harm others. He is still responsible, still has freedom of will.

This is good, practical sense for some people. But there is a problem with this argument for executing Elvis. Responsibility and self-reflection are not disembodied, ethereal processes; they are rooted in the brain. Patients with a damaged ventromedial prefrontal cortex (a part of the brain above the middle of the eyes) are known to become irresponsible, lacking in self-discipline, and unable to consider the consequences of their actions. The very mechanism that

supports the ability to take responsibility is damaged in Elvis. He may no longer be able to reflect on his behavior or take responsibility for his risky predispositions.

Most prisoners I have met who are suspected of brain dysfunction have no idea that anything is wrong with them. Often they grew up with this problem; it has always been part of them. Even when this is pointed out to them, they believe (like much of the general public) that the causes of their violence lie in poverty, unemployment, bad

Poverty and bad parenting, after all, can be seen and recognized; biological factors are invisible and intangible.

influences, and (sometimes, but not always), poor parenting and child abuse. They are shocked and disbelieving if I tell them that brain dysfunction and biology contribute to violence. Poverty and bad parenting, after all, can be seen and recognized; biological factors are invisible and intangible. So it is not just that the brain mechanisms underlying responsibility are damaged in the violent offender, preventing him from acting to rectify the causes of his violence. Even if he could reflect on himself, he probably would end up accusing the wrong risk factors.

BLAMING IT ON CAIN

Cooped up at home at Christmas time, we get on top of each other. Herbert Weinstein was no exception. In 1991, by the end of the Christmas holidays, he had had about as much as he could take of his wife in their twelfth-floor Manhattan apartment. After

strangling her, he threw her out of the window to make her death look like suicide. He was charged with second-degree murder. Things looked bad for Weinstein until his defense lawyer had his brain scanned and found poor functioning caused by a cyst in the left frontal and temporal brain regions. The prosecution tried to have the PET evidence ruled inadmissible but after an extensive hearing, the judge admitted it. This was apparently the first time that PET data had been admitted as evidence for an insanity defense during a trial itself.

Before this ruling, PET had been strictly limited to the sentencing hearing, when all possible grounds for mitigation may be introduced. In Weinstein's case, the PET data proved compelling. In a plea-bargain arrangement, the charge against him was reduced to manslaughter, yielding a much lighter sentence.¹⁵

In cases like Herbert Weinstein's, the mark of Cain may literally be on the brain. But blaming Cain is unpopular. At a scientific level it is hard to know whether brain dysfunction caused the violent behavior or whether living a violent life resulted in the brain damage. Studying the brain basis of violence is in its infancy. Even if other scientists reproduce and extend our findings, there will be the difficulty of going from findings based on groups of offenders in research studies to conclusions about an individual killer.

I suspect, though, that these are not the main reasons for opposition to applying biological findings on violence to legal cases. Behind the opposition are multiple political, theological, legal, and moral

issues.¹⁰ Biological research on violence is politically unpopular with both right and left. Conservatives worry that biological research will be used to let vicious offenders off the hook. Liberals fret that brain-scan technology may someday be used preventively to lock up an innocent person with the profile of a violent offender. Theologians dislike the idea that biology constrains individual freedom of will because they believe that God made us equally able to embrace good and reject sin. Legally, we are guilty of a crime if we have criminal intent and know that what we are doing is wrong; and even violent offenders with substantial brain damage can tell the difference between right and wrong, know that they are doing wrong, and sometimes carefully plan their actions. In doing so, they show criminal intent and demonstrate legal sanity. At the broadest moral level, most of us feel that offenders have to be punished if they do wrong; not to punish would be morally wrong. In addition, practically, what would happen if society excused serious crimes because of bad brains? Could this not become a license to kill?

THE FUTURE

Against this broad opposition, will brain-imaging research on violence ever make a difference? I think that it could and should make a difference; but because it profoundly challenges our way of conceptualizing crime (not to mention our “gut reaction” to crime, a reaction perhaps rooted in our evolution as a species), I am far from certain that it will make a difference. We have much to lose if it does not. Let me elaborate.

The political, legal, and moral questions

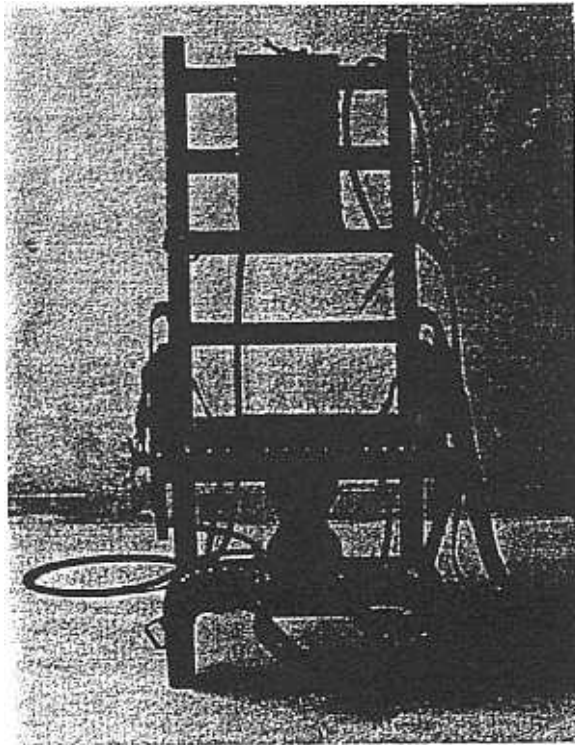
raised by this research are complex and worrisome, but they are too important to ignore. The question that law courts ask when dealing with a defendant is “did he do

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it”? There are obvious reasons for this question. But the more important question—one that courts rarely ask—is “Why did he do it?”

Violent crime may still be with us because we have largely ignored that question. Continuing to ignore it may ensure that we remain vulnerable to violence. In part, prevention programs have failed to stop violence because they systematically ignored the biological part of the biosocial equation that explains violence.

The violent offender is like a jigsaw puzzle. Decades of careful psychosocial research have identified some of the pieces (e.g., child abuse, poverty, poor parental supervision, delinquent peers, and gangs). Recently we have begun to identify the biological pieces (e.g., low physiological arousal, high testosterone, birth complications, and

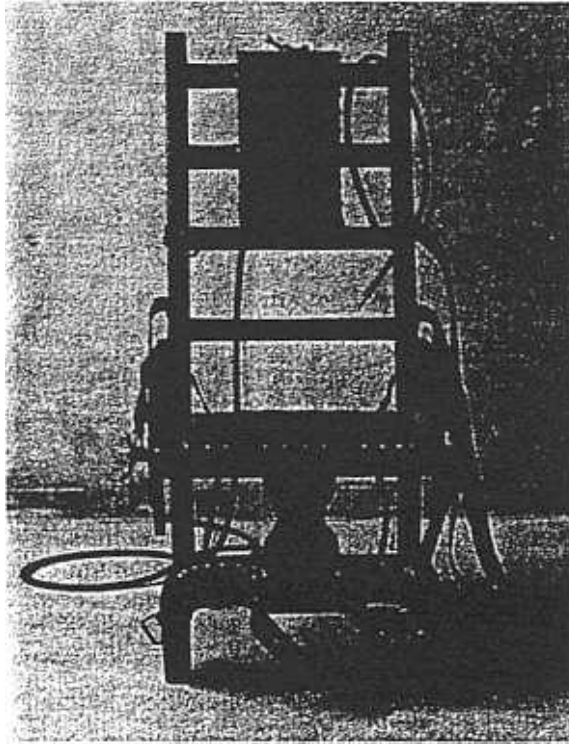


Brain imaging research on violence challenges the way we think about crime. Two hundred years from now will we consider some kinds of serious criminal behavior to be a critical disorder and look aghast at the execution of violent offenders?

low serotonin). Now brain-imaging research is beginning to identify the brain mechanisms (involving the prefrontal cortex, amygdala, hippocampus, and corpus callosum). The challenge is to uncover more neurobiological pieces and put them together with the social pieces to fill out our picture of the violent offender.

Some thinkers see a strong sociobiological basis for homicide, violence, and property crimes.^{10,16} In evolutionary terms, it has paid some individuals to be antisocial, parasites on the rest of us, and to seize others'

resources to increase their own genetic fitness as measured by producing more offspring. Perhaps this gave rise to a genetic predisposition to crime and aggression.¹⁰ But I would argue that evolutionary forces also built into all of us (including antisocials) strong emotional reactions against criminals: vengeance, hate, retribution, and a desire to ostracize. These leave little room for understanding and forgiveness. Whether or not there is a genetic or a biological predisposition to violence, when a violent crime is committed, we want to



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blame someone. We want someone to pay. Evolution seems to have forged not only crime and violence but our armor against any evidence that might "excuse" them.

Brain-imaging research on violence troubles us by challenging the way we think about crime. It questions our treatment of murderers in the way that, looking back 200 years, we question the shackling of the mentally ill. The history of civilization suggests that, at least over the long term, society has

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tended to become more humane. Two hundred years from now will we have reconceptualized recidivistic, serious criminal behavior as a clinical disorder with roots in early social, biological, and genetic forces beyond the individual's control? Will we look back aghast at the execution of seriously violent offenders? Will we view execution of prisoners as we now view the burning of witches?

I would like to think so, but our gut reaction to crime may never change. A key reason for our success as a species is our effective mechanism for shutting out those in our midst with the mark of Cain. In the end, that mechanism may block all change in how we think about and deal with crime.

I believe such a refusal to change would be an enormous mistake. When we learned to control our environment, the human race was freed from the yoke of the evolutionary forces that once shaped us. Now we can think and act more freely, and use the scientific knowledge base we are acquiring. What was an adaptive response to violent crime 100,000 years ago is no longer so adaptive. Our time-immemorial practices do protect society from particular criminals, but they do nothing to prevent the next generation of violence.

Brain-imaging research is beginning to give us new insights into what makes a violent offender. These early findings may at least lead us to rethink our approach to violence and goad us into obtaining new answers to the causes and cures of crime while we continue to protect society. ■

*Quotation on p.10: "Blame It On Cain" by Elvis Costello.
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