# Prefrontal cortex vs criminal behavior

Law, Criminology

This very broad overview of prefrontal cortex function allows us to appreciate circumstances in which prefrontal cortex function is compromised in a human. Humans comprise a special case when considering this brain region. Despite evidence that the prefrontal cortex in rodents and non-human primates regulates cognition and behavior in ways quite similar to that of the human, we are the most ‘ frontal’ of species, insofar as the frontal cortex is its largest, in both absolute and relative terms, in the human.

The first realm to consider where prefrontal cortex function is compromised in humans is, quite reasonably, during development. Children show only minimal frontal function, from the standpoints of cognition (for example, in reversal tasks), emotional regulation, control of impulsive behavior and moral reasoning. One of the myths ofchild developmentis that the brain is fully developed at some remarkably early age. Instead, brain development is far more prolonged and, not surprisingly, the prefrontal cortex is the last region of the brain to fully myelinate. Remarkably, this process extends well beyond adolescence into early adulthood. Various transient states can compromise prefrontal cortex function.

Alcohol is long recognized for its capacity to impair reasoning and impulse control, and surprisingly small quantities of alcohol impair the capacity of the prefrontal cortex to detect errors of commission or omission, as assessed electro-physiologically.

Another example concernsstress. Most individuals have experienced severe and/or

prolonged stress as disrupting attention, judgment and other purviews of the prefrontal cortex, and this has been shown more formally in both humans and animals;

In making sense of this, it should be appreciated that the prefrontal cortex contains some of the highest levels of receptors in the primate brain for stress hormones. Moreover, stress or stress hormones will dramatically alter the turnover of several classes of neurotransmitters in the prefrontal cortex.

Prefrontal cortex function is also compromised in another circumstance experienced by all individuals. With the onset of sleep and the transition to deep, slow wave sleep, there is a characteristic decrease in activity throughout the brain, particularly in the cortex. However, with the transition to paradoxical rapid eye movement sleep, there is increased activity in a variety of brain regions, including associational cortex and limbic systems; strikingly, metabolic rate can even be higher than during wake periods.

Amid this shift, there is a virtually complete cessation of activity in the prefrontal cortex, producing a relatively metabolically active brain that is unconstrained by the regulatory effects of the prefrontal cortex. It has been speculated that this, in effect, accounts for whydreamsare ‘ dream-like’: characterized by emotional liability, non-sequential thinking and extreme disinhibition.

Amid that, general intelligence and executive function can remain intact. By contrast, when damage occurs at earlier ages, executive function is impaired and the impulsivity takes on a more global and malign nature that has been termed ‘ acquired sociopathy’, where antisocial behaviors can be markedly

premeditated. The issue of brain development becomes relevant when considering individuals with sociopathic and antisocial behavior in which there is no obvious history of prefrontal cortex damage.

Moreover, when sociopaths must engage the prefrontal cortex, they activate more of the prefrontal cortex than control individuals to achieve the same level of efficacy. In other words, even when these individuals actively attempt to do the ‘ harder thing’, their prefrontal cortexes are less effective. Importantly, among such sociopathic individuals, the smaller the volume of the prefrontal cortex, the greater the tendency towards aggressive and antisocial behavior.

The Prefrontal Cortex and the Criminal Justice System:

We have come to recognize numerous realms in which a biological abnormality gives rise to aberrant behavior, and such recognition has often then given rise to an expectation that people now exert higher-order control over that abnormality. For example, as noted, we would never consider an epileptic violent who strikes someone in the process of a seizure: ‘ it is not him; it is his disease’. However, we expect that pileptic to not drive a car if their seizures are uncontrolled. Or we are coming to understand the euro chemistry of context-dependence relapse into drug dependency in organisms. Thus, we have come to expect ex-addicts to avoid the settings in which they previously abused drugs.

There is a false dichotomy in this manner of thinking. It is as if we artificially demarcate an area in which biology dominates: yes, there is something organic that gives rise to

this person having uncontrolled and synchronous neuronal discharges, or who has certain pathways potentiated that project onto dopamine-releasing ‘ pleasure’ pathways. But it is as if, with that area of organic impairment identified and given credence, we expect it to be bounded, and for the rest of our ‘ us-ness’, replete with free will, to now shoulder theresponsibilityof keeping that organic impairment within the confines of its boundaries. It cannot possibly work this way.

What the literature about the prefrontal cortex shows is that there is a reductive, materialistic neurobiology to the containment, resulting in the potential for volitional control to be impaired just as unambiguously as any other aspect of brain function. It is possible to know the difference between right and wrong but for reasons of organic impairment, to not be able to do the right thing. The most obvious implication of this concerns how individuals with demonstrable prefrontal cortex damage are treated in the criminal justice system. As the simplest conclusion, everything about this realm of contemporary neurobiology argues against the retrenchment back towards a sole reliance on M’Naghten that has gone on in recent decades.

Amid the seeming obviousness of this conclusion, there is always a valid counter-point that can be raised: there are individuals with substantial amounts of prefrontal cortex damage who, nonetheless, do not commit crimes. At present, knowing that someone has sustained prefrontal cortex damage does not give much power in predicting whether that person’s disinhibition will take the form of serial murder or merely being unable to praise a nearly inedible meal prepared by a host. This seems to weaken the ‘ volition can be organically impaired, just like any other aspect of brain

function’ argument; in these interstices of unpredictability seem to dwell free will.

However, we can begin to imagine tree diagrams of variables that, with each new layer, add more predictive power. We can already see two layers in the realm of prefrontal cortex function. The first layer might query, ‘ prefrontal cortex: normal or damaged?’ The second might then query, ‘ if damaged: damaged inchildhoodor later?’ This same structure of increasing predictive power was shown in a recent, landmark study concerning clinicaldepression.

Having a particular variant of the gene 5-HTT (which codes for a protein that regulates synaptic levels of the neurotransmitter serotonin) increases the risk of depression. However, ‘ 5-HTT: pro-depressive variant or other variant?’ gives only a moderate predictive power, but the authors then demonstrated the adding in of a second layer, ‘ if the pro-depressive variant: major stressors during childhood or not?’ now generates an impressive predictive power as to which adults succumb to clinical depression. If free will lurks in those interstices, those crawl spaces are certainly shrinking.

Recent U. S CollegeCase Study:

By Daniel Strueber, Monika Lueck and Gerhard Roth.

On September 13, 2006, Kimveer Gill walked into the cafeteria at Dawson College in Montreal and, without apparent motive, shot 21 people, injuring 19 and killing two, including himself. The same day a judge in West Virginia sent a woman to jail for, among other atrocities, forcing her six children and stepchildren to gorge themselves onfoodand then eat their own vomit. Also on the 13th, a court in New York sentenced a man for killing his girlfriend by

setting her on fire--in front of her 10-year-old son. There was nothing special about that Wednesday. From around the world we hear reports of murder, manslaughter, cruelty and abuse every day. Violenceis ubiquitous.

But what drives one person to kill, maim or abuse another, sometimes for little or no obvious reason--and why do so many violent offenders return to crime after serving time in prison? Are these individuals incapable of any other behavior? We have evaluated the results of studies conducted around the world, focusing on acts ranging from fistfights to murder, in search of the psychobiological roots of violence. Our key conclusion is simple: violent behavior never erupts from a single cause. Rather it results from a combination of risk factors--among them inherited tendencies, a traumatic childhood and other negative experiences--that interact and aggravate one another. This realization has a silver lining: positive influences may be able to offset some of those factors that promote violence, possibly offering hope for prevention.

Indeed, male gender is the most important risk factor for violent behavior. As criminal statistics show, boys and young men commit the majority of physical assaults. According to the Federal Bureau of Investigation's statistics on crime in the U. S., 90. 1 percent of murderers apprehended in 2004 were male and men accounted for 82. 1 percent of the total number arrested for violent crimes. Girls and women are not necessarily less aggressive, as was assumed until the 1990s. But women engage in more indirect, covert aggression, whereas men tend toward immediate, outward physical aggression.

The causes of these gender differences are manifold. Learned sex roles certainly enter into it: " girls don't hit," for example, but " boys need to be able to defend themselves." Also, indirect aggressive strategies require a relatively high level of social intelligence, which girls develop earlier and faster. Moreover, neuropsychological discrepancies almost certainly play a role. The small group of males who exhibit chronic violent behavior from an early age typically share other telltale traits, among them a low tolerance for frustration, deficiencies in learning social rules, attention problems, a decreased capacity for empathy, low intelligence and, most characteristic, extreme impulsiveness.

Similarly, repeat offenders--particularly those who have long prison records seem unable to keep their aggressive urges in check. The late neuroscientist Ernest S. Barratt and his colleagues at the University of Texas Medical Branch interviewed imprisoned criminals in Texas in 1999 and found that many inmates consistently picked fights, even though they knew that their lives would be made more difficult as a result. When asked why they continued to behave in ways that hurt them, many responded that they had no idea. Even though they understood the consequences and resolved to act with greater self-control the next time, they did not trust their own ability to keep their impulses at bay.

Preliminary research indicates that biology may handicap some of these individuals, making it more difficult for them to show restraint. Among violent offenders, neuroscientists have found anatomical and physiological differences in both the limbic system and the prefrontal cortex, brain regions that are involved in the development and control of emotions. Some

scientists propose that the orbitofrontal cortex, a region of the prefrontal cortex where decision making takes place, inhibits areas of the limbic system--specifically the hypothalamus and the amygdala, primitive brain regions that are a source of fear and aggressive impulses. Thus, if some defect or injury impairscommunicationbetween the limbic system and the frontal cortex, a person might not be entirely able to moderate his or her emotional reactions.

References

1. www. nature. com/mp/journal/v8/n2/full/4001256a. html
2. www. jnnp. bmj. com/cgi/content/full/71/6/720
3. www. pubmedcentral. nih. gov/articlerender. fcgi? artid= 1693445
4. www. sciammind. com/print_version. cfm? articleID= 33EF147A-E7F2-99DF-3696EF69D814FCFF