Encyclopedia of Criminological Theory

Brain Abnormalities and Crime

Contributors: Yaling Yang & Adrian Raine
Editors: Francis T. Cullen & Pamela Wilcox
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Despite the large body of knowledge accumulated by neuroimaging research in the last few decades associating criminal behavior to various neurobiological risk factors, the most well-known theories of criminology remained focused on the use of social and environmental variables to explain criminal and antisocial tendencies. The lack of inclusion of a neurobiological aspect in these theories prevents an in-depth understanding of the complicated biosocial mechanisms underlying criminal behavior, specifically regarding the role that brain abnormalities play in predisposing individuals to criminal and/or violent behavior. This gap in theories can be viewed as problematic especially since several reports have confirmed high rates of brain abnormalities among death row **[p. 106 ↓]** inmates, forensic psychiatric inpatients, and other individuals with a history of violence.

The phrase *brain abnormalities* is often employed, as an umbrella term, to describe congenital or acquired anatomical or functional alterations in the brain. Brain abnormalities have long been associated with psychiatric disorders, such as antisocial personality disorder (APD) and psychopathy, of which criminal behavior is a prominent feature. Several hypotheses and models have been proposed based largely on empirical evidence from brain imaging literature.

This entry reviews a key hypothesis, the frontal lobe dysfunction hypothesis, which has received a great deal of attention in the past few decades with regard to the neural bases of antisocial, criminal behavior. Specifically, the theory as well as the supporting empirical evidence from lesion, neuropsychological, and neuroimaging findings of structural and functional brain abnormalities in the frontal lobe of criminal offenders and antisocial individuals is discussed. This is followed by a discussion on other models of brain abnormalities and crime, specifically the somatic marker hypothesis and the violent inhibition mechanism, which focuses on brain deficits underlying poor decision making and moral reasoning aspects of criminal offending. This entry concludes with an assessment of the hypothesized link between brain abnormalities and crime, and with a discussion of implications for future studies.

Frontal Lobe Dysfunction Hypothesis

The frontal lobe has long been linked to antisocial criminal behavior due to the critical role of this brain region in executive function. The development of this hypothesis has been prompted by lesion, neuropsychological, and neuroimaging studies to explain the behavioral tendency for rule-breaking in criminal offenders. The frontal lobe dysfunction hypothesis proposes that structural and functional deficits to this brain region impair the higher cognitive functioning such as moral decision making, impulse control and emotion regulation, which contribute to criminal offending. This hypothesis received support from Brown and Price, who reviewed the neuropsychological and neuroimaging findings of incarcerated criminals and concluded a high correlation between frontal dysfunction and increase in aggression and antisocial behavior.

Lesion studies have reported changes in personality, specifically an increase in antisocial criminal behavior, following injuries to the frontal lobe. The increase in risky deviant behavior was particularly noticeable when the damage to the frontal lobe involved the orbitofrontal regions. Several case studies have described patients with damage to the orbitofrontal regions, particularly the medial orbitofrontal cortex, to show marked deficits in real-life tasks demanding moral judgment, impulse control, interpersonal sensitivity, and the evaluation of future consequences, while revealing minimal impairments on standard neuropsychological tests of intelligence and executive functions. These patients were also found to show frequent explosive aggressive outbursts. In addition, it has been demonstrated that when prefrontal lobe damage occurred earlier in life (e.g., before the age of 8), the patient suffered a more severe outcome of behavioral disturbances, including executive dysfunction, poor abstract conceptual thinking, impaired theory of mind, and immature moral reasoning later in life.

The hypothesis of frontal lobe dysfunction in predisposing to criminal behavior is also supported by several large systematic studies on cohorts of war veterans with head injuries. For example, the Vietnam Head Injury Study found that individuals with frontal lobe lesions engaged in aggressive behavior (e.g., fights, property damages) at a higher rate (14 percent) than those without frontal lobe damage (4 percent). This study also showed that increased aggressive behavior was significantly associated with localized damage to the medial and orbitofrontal regions identified using computed tomography.

Other studies reported similar behavioral symptoms as a result of frontal damage in patients with frontotemporal dementia. These patients showed higher rates of criminal behavior—including stealing, physical assault, and sexual comments or advances—compared to patients with Alzheimer's disease who exhibited equally impaired cognitive function. In short, these lesion findings suggest that dysfunction as a result of frontal damage may be associated with increased criminal behavior, particularly that of an impulsive nature, due to the inability of the disrupted frontal network to suppress impulse.

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The frontal lobe dysfunction hypothesis for crime is also supported by studies using anatomical magnetic resonance imaging (aMRI) to examine the frontal lobe structures of antisocial individuals and criminal offenders (for a review, see Yang, Raine, et al., 2009). For example, one aMRI study on individuals with APD and found significant gray matter volume reduction in the prefrontal cortex compared with normal controls, a substance-dependent control group, and also a broader psychiatric control group. Similar findings of reduced gray matter volume in the frontal cortex have been reported in aggressive epileptic patients and criminal psychopaths compared to their non-aggressive, non-criminal counterparts. Consistent with lesion findings, aMRI studies further revealed that the reduced gray matter volume in antisocial individuals is localized in the dorsolateral prefrontal, medial frontal, and orbitofrontal cortex. Supporting the predictions of the frontal lobe dysfunction hypothesis, these studies showed a consistent pattern of structural deficits in the frontal lobe in criminal offenders, which may impair their frontal functioning for effective behavioral control and decision making.

More direct evidence has also been accumulated from functional imaging studies using positron emission tomography (PET), single photon emission computed tomography (SPECT), and functional magnetic resonance imaging (fMRI) to assess the functional integrity of the frontal lobe in individuals with antisocial criminal behavior. For example, several PET studies on psychiatric patients with a history of repetitive violence found decreased metabolic activity in the frontal cortex compared with normal controls. Similarly, significant correlation between higher severity of aggression and lower metabolism in the bilateral medial prefrontal cortex has also been found in a group of aggressive children with epilepsy. Other studies that examined the metabolic

abnormality in antisocial individuals by using a challenge task, most commonly a continuous performance task (CPT), also discovered frontal lobe dysfunction in violent individuals. For example, in 1994, Raine and colleagues found reduced glucose metabolism in the anterior medial prefrontal, orbitofrontal, and superior frontal cortex in murderers compared to normal controls using a CPT task. Findings were replicated by the same research team in a follow-up study of a larger sample of murderers and controls (*N* = 82) in 1997. Using SPECT, Daniel decreased regional cerebral blood flow (rCBF) activity in the frontal cortex was found in aggressive psychiatric patients (Amen et al., 1996) and violent perpetrators (Soderstrom et al., 2000). In another study of 21 individuals convicted of impulsive violent offenses, it was revealed that violent patients showed reduced rCBF in the bilateral dorsofrontal cortex compared to non-violent patients (Hirono et al., 2000).

The fMRI represents the latest development in brain imaging techniques employed to examine the association between frontal lobe dysfunction and criminal offending, which quickly became popular due to its non-invasive and low-risk nature. In the last several years, clinical neuroscientists have seized upon this technology to examine brain functioning in criminals and antisocial individuals and found abnormal brain activity in the frontal regions in these individuals during an emotional or a cognitive task. For example, Frank Schneider and colleagues found increased activation in the dorsolateral prefrontal cortex during the acquisition phase of fear conditioning in individuals with APD. Another study conducted by Niels Birbaumer and colleagues revealed that criminal psychopaths showed significant reduced activation in the orbitofrontal cortex and anterior cingulate cortex compared to healthy controls. Using a working memory task, V. Kumari and colleagues found activation deficits in the left frontal gyrus and anterior cingulate cortex in violent offenders compared with normal controls. These functional imaging studies have provided strong evidence supporting the frontal lobe dysfunction hypothesis by demonstrating that several frontal subregions failed to respond properly in antisocial individuals and criminal offenders.

Although these brain imaging findings have been highly suggestive of a strong link between criminal offending and brain abnormalities, particularly within the frontal cortex, there has been criticism regarding the under-specificity of this hypothesis, in that it did not specify which subregions of the frontal lobe are more closely connected with criminal behavior. Furthermore, it has been argued that this hypothesis seems

applicable for reactive types of criminal offending (e.g., reactive aggression) but not for instrumental aggression.

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Other Theories on Brain Abnormalities and Crime

Several other models have been proposed for the neurobiological basis of criminal behavior. Many have targeted the poor decision making and aggressive aspects of crime and have hypothesized more localized brain abnormalities in the amygdala and the orbitofrontal cortex (OFC) to be linked to increased deviant behavior. One of the theories is the somatic marker hypothesis, which predicts deficits in the medial OFC may lead to risky decision making by impairing an individual's ability to accurately assess the reward/punishment value of his or her actions. Another hypothesis, the violent inhibition mechanism model, proposes that brain abnormalities in the amygdala would result in disrupted moral development and the continuation of using aggressive behavior to achieve goals.

The Somatic Marker Hypothesis

The somatic marker hypothesis was developed by Antonio Damasio based on findings of patients with brain lesions. It represents a complementary theory for brain abnormalities and crime by suggesting that the neural system underlying emotional processing and regulation (i.e., the medial orbitofrontal cortex) plays a role in decision making. In real life, decision-making processes involve the formation of the action-outcome associations (somatic markers), which are stored in the orbitofrontal regions to assist future decision making in similar situations. This process allows healthy individuals to categorize and learn from punishments. This hypothesis predicts that deficits in the medial OFC would disrupt this process and prevent the individual from experiencing the negative emotional feedback necessary for forming the somatic markers. Such failure in the somatic marker mechanism likely results in an individual's

inability to learn from punishment, therefore recidivating during conflict situations. This hypothesis received a great deal of support from case reports, which found patients with damage to the medial OFC to show changes in personality and behavior. The changes include disinhibition, social dysfunction, emotional deficiency, risky decision making, and a lack of insight into their behavioral problems which may put patients at higher risk for criminal offending. Neuroimaging literature also supports the hypothesis by showing that criminal offenders have structural and functional deficits in the orbitofrontal regions (Yang & Raine, 2009). However, the more specific prediction of this hypothesis that criminal offenders would show reduced autonomic response to emotion-inducing reward or punishment behavioral outcome has not yet been tested.

The Violent Inhibition Mechanism Model

The importance of empathy for the development of moral socialization was said to be one of the reasons for proposing the violent inhibition mechanism (VIM) model. This model proposed that distress cues (e.g., sad and fearful facial affects of others) activate the VIM and in turn result in the aggressive act that caused the distress cue aversive as well. Blair and colleagues define this as the process of moral socialization. This model suggests a crucial involvement of the amygdala in the VIM system due to its role in emotion regulation, fear conditioning learning, and moral decision making. Through the process of classical conditioning, one associates these representations of moral transgressions as triggers for the VIM. Through moral socialization, the normally developing child finds thoughts of aggressive act aversive, and becomes less likely to engage in physical aggression in the future.

Although proposed for psychopathy and moral development in particular, the same principle applies to criminal offenders. Based on this model, it may be hypothesized that criminals, particularly violent ones, have a disrupted VIM, possibly an impaired amygdala, resulting in maldeveloped moral reasoning. This prediction has received empirical support from lesion and imaging studies showing amygdala deficits in violent criminal individuals. Although they did not routinely become aggressive, lesion studies showed that patients with amygdala lesions, often showed symptoms such as the inability to recognize facial emotions and had poor recollection of emotional events that may result in the abnormal preference for antisocial behavior. Relationships between

abnormalities in the amygdala and individuals with criminal psychopathic behavior have also **[p. 109 ↓]** been found in several brain imaging studies. For example, volume reduction in the amygdala has been found in criminal psychopaths and violent offenders. Functionally, a 1997 study by Raine and colleagues involving murderers found abnormal asymmetries of functioning, with murderers showing lower left and increased right functioning in both the amygdala and hippocampus compared with controls. More recently, using tasks involving emotional processing, several fMRI studies have found abnormal amygdala activation in criminal psychopaths, adolescents with conduct disorders and in individuals with APD.

The VIM model has an advantage over frontal lobe dysfunction and somatic marker hypotheses in that it provides an explanation for the emergence of instrumental aggression. Although supported by some neuropsychological and neuroimaging studies, the VIM model for many criminologists still seems too narrow to fully explain the wide range of criminal acts.

Conclusion

With the advancement of brain imaging techniques, abnormalities in several brain regions have been revealed in criminal offenders. One of the major theories in the field, the frontal lobe dysfunction hypothesis, has received great support from neuroimaging studies showing both structural and functional disturbances in the frontal lobe in individuals with antisocial criminal behavior. Several other theories including Damasio's somatic marker hypothesis and violent inhibition mechanism model have provided more specific predictions associating brain abnormalities in the amygdala and orbitofrontal cortex with criminal offending and violent behavior. Future development of a more complex hypothesis incorporating genetic imaging, neuropsychological, psychophysiological, and neuroimaging findings may prove to be of great help in the understanding of brain abnormalities and crime.

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See also

- Ellis, Lee: Evolutionary Neuroandrogenic Theory
- Fishbein, Diana H.: Biosocial Theory
- Mednick, Sarnoff A.: Autonomic Nervous System (ANS) Theory
- Neurology and Crime
- Psychophysiology and Crime

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