Encyclopedia of Criminological Theory

Neurology and Crime

The beginning of the search for the neurological basis of crime can be traced back to Cesare Lombroso, an Italian criminologist and physician who provided one of the most influential arguments that criminals are born with a nature favorable to crime. Although no direct means exists to examine in vivo the brain anatomy of criminals at the time, Lombroso managed to identify several distinct physical features, which he called "stigmata." This included a slanting forehead, long/no ear lobes, a large jaw with no chin, heavy supraorbital ridges, excessive/absent hair on the body, and an extreme sensitivity/insensitivity to pain. According to Lombroso, the possession of multiple physical abnormalities indicated that the individual was less developed, a "born criminal," and thus could not adjust to the rules of modern society. Although Lombroso's argument was less than sound, the idea that criminal behavior is influenced by biological predispositions has endured and gained significant interest since then.

With the development of brain imaging, the emphasis has since shifted to establish the connection between disruptions in the neural system and elevated criminal behavior. Criminal behavior, especially aggression, can be observed even in toddlers at the age of 1 to 2 years, when the brain is far from mature to allow full control over behavior. As the neural systems mature, children learn to deal with their aggressive impulses in a socially appropriate manner, and aggressive behavior diminishes as a result. Therefore, it has been predicted that if development of the neural system were interrupted (e.g., prenatal or postnatal damage to the brain), the maldeveloped, immature brain would be unable to function properly in behavior control and moral reasoning and continue to use aggressive behavior as a means to obtain goals.

Several theories have been proposed to further explain the association between neurological deficits and criminal offending. Among them, three major theories have been most widely accepted and intensively tested: Terrie Moffitt's *developmental theory*, Antonio Damasio's *somatic marker hypothesis*, and Jeffrey Gray's *dual biological model*. Below, each hypothesis and the supporting evidence for it is reviewed. The discussion is extended by drawing additional evidence for these hypotheses from individuals with traumatic brain injuries (TBIs) to demonstrate the high prevalence of neuropathology in criminal offenders. This entry concludes by assessing the hypothesized links between neurology and crime and by discussing implications for future studies.

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Theories on Neurology and Crime

Moffitt's Developmental Theory of Crime

This theory was developed based on Moffitt's 1993 groundbreaking work indicating that signs of persistent deviant behavior during adolescence can be detected as early as the preschool year and are influenced by the behavior of peer groups. This theory identified two groups of delinquents—the life-course-persistent (LCP) and the adolescent-limited (AL) offenders—based on their ages of onset and trajectories of conduct problems. The AL group may only be engaging in criminal activities as a way of expressing their adolescent rebellion and usually desist from any pathway toward crime. By contrast, the LCP group precociously escalates into serious criminal offenses as a way of expanding the versatility of their antisocial tendencies and usually maintain a lifestyle of repeated criminal offending. According to the theory, the LCP offenders may suffer prenatal and perinatal disruptions in neural development that contribute **[p. 662 ↓]** to their persistent criminal behavior. These neurological deficits, which in most cases were too subtle to require clinical remediation, often manifested as behavioral problems such as inattention, hyperactivity, irritability, and impulsivity. Thus, neurological deficits in the LCP offenders may put them at higher risk for early-onset conduct disorders, which often escalate to persistent delinquent behaviors when interacting with an unsupportive environment.

Moffitt's developmental theory has received strong support from neuropsychological studies confirming that LCP offenders may indeed have neurocognitive impairments that reflect underlying neurological disturbances. For example, one study found that LCP offenders show lower intelligence, impaired spatial memory, and poor performance on tasks targeting frontal functions such as the continuous performance task. Findings from these studies suggest that neuropsychological impairments are especially prominent in LCP offenders. However, the lack of empirical studies assessing the structural and functional integrity in the neural system in these individuals prevent direct testing of the neuropathology in LCP offenders predicted by this hypothesis.

Damasio's Somatic Marker Hypothesis

The somatic marker hypothesis, formulated by Damasio in 1994, argues that emotion could guide or bias the decision-making process through the neural system of the somatic marker mechanism. This theory, although developed based on findings of patients with brain lesions, represents a complementary theory that readily applies to the neurological basis of criminal behavior. Damasio suggested that in real life, decision making involves both cognitive and emotional processing to assess the reward value of the various behavioral options available in any particular situation. When the situations are complex and conflicting, the reward values of the actions are uncertain and ambiguous, which induce physiological affective states (e.g., changes in skin conductance levels) followed by the forming of action-outcome associations (i.e., somatic markers). The somatic markers from all previous experiences are summed to produce a net somatic state to assist future decision making in similar situations by directing the selection of an appropriate action to achieve the most beneficial outcome. This process allows healthy individuals to categorize and learn from negative experiences. Because the ventromedial prefrontal cortex (VMPFC) is the essential component of this somatic marker hypothesis, damage to this structure may disrupt the mechanism and prevent the individual from experiencing the feedback necessary for producing somatic markers to avoid future aversive consequences. Such failure in the process will likely predispose to the inability to learn from punishment thus recidivate during conflict situations.

This hypothesis received a great deal of support from subsequent lesion studies. Patients with bilateral damage to the VMPFC often suffer from behavioral disinhibition, social dysfunction, emotional deficiency, poor decision making, and a lack of insight into their behavioral problem, which may lead to criminal offending. One such case is that of Phineas Gage, a railway foreman who had an iron stake blown through his frontal lobe in an accident involving explosives. Gage survived the injury, recovering his physical and intellectual abilities, but his personality changed dramatically and he became markedly antisocial. In general, the social, emotional, motivational, and behavioral dysfunction of these patients with damage to the VMPFC was often accompanied by a change in personality, which put them at greater risk for criminal offending.

Furthermore, it was observed that these patients show aggressive behavior that is exclusively impulsive in nature (Anderson et al., 1999; Grafman et al., 1996), which reflects a limitation of this theory: it cannot account for some types of criminal offending, particularly instrumental aggression (Blair et al., 2005).

Gray's Dual Biological Model

Gray proposed a dual biological model, which at the core are two competing motivational systems: the behavioral inhibition system (BIS) and the behavioral activation system (BAS) (Fowles, 1988; Gray, 1982). The BIS represents an inhibitory system for withholding behavior in ambiguous threatening situations, whereas the BAS is the underlying system for impulsivity. The two systems are located in different parts of the brain. The BIS is in the septo-hippocampal region and the BAS is in the basal ganglia, thalamic nuclei, and the ventral tegmental area (Gray, 1994). This theory has been employed specifically for explaining aggression by Angela Scarpa and Adrian Raine, who suggested **[p. 663 ↓]** that violent behavior is a function of an underactive BIS, an overactive BAS, or a combination of both. This pattern of weak BIS and possibly a strong BAS in this dual biological mechanism has also been referred to when explaining clinical conditions for which criminal recidivism is a core symptom, such as psychopathy (Fowles, 1988). Therefore, it may be predicted that individuals with criminal tendencies would show behavioral features of decreased anxiety, increased impulsivity, and a reward-driven decision-making process due to neurobiological impairments in septo-hippocampal, basal ganglia, thalamic nuclei, and ventral tegmental structures.

Although no study to date has simultaneously examined these behavioral and neurobiological characteristics in criminal offenders, this theory has received considerable support from socio-behavioral and neuroimaging studies separately. For example, significantly high in impulsivity and low in anxiety compared to controls has been found in a group of violent offenders. With regard to neuroimaging literature, studies to date have provided some evidence for deficits in the neural system underlying the BIS system, particularly in the hippocampus, which may contribute to the weakness of this system in antisocial, criminal individuals. For example, it has been demonstrated that violent offenders with APD and type-2 alcoholism show reduced

volume in the right hippocampus. In addition, an exaggerated structural hippocampal asymmetry (right > left) has been found in criminal psychopaths. Functional imaging studies also showed reduced blood flow in the hippocampus in violent offenders and murderers However, the integrity of the neural system underlying BAS remains to be examined.

Supporting Evidence from Traumatic Brain Injuries

Although different mechanisms were proposed, all three major theories on neurology and crime predict that neurological deficits in several brain regions, through impairing the processes of attention, decision making, and impulse control, may result in the elevation of criminal behavior. This argument is consistent with findings showing higher rates of traumatic brain injuries in criminal offenders.

Traumatic brain injuries (TBIs), ranging from subclinical to fatal in severity, are one of the leading causes of morbidity and mortality in children and adolescents in the United States. Most TBIs were suffered during a fall, motor vehicle accident, assault, or suicide attempt, and put the individuals at higher risk for developing functional impairments. Evidence has been accumulated suggesting a high correlation between violent crime and neurological brain damage. For example, in a study of 15 death-row inmates, it was found that all violent offenders had a history of severe head injury and 5 had major neurological impairment. Several investigations on delinquent youths also reported similar associations between TBI and antisocial behavior. For example, one study found half of the delinquent youths in their sample had experienced one or more TBIs, and one third of those with TBI histories reported diminished ability in regulating behavior and emotion, sustaining attention, and performing in social and school settings as a result of their TBIs. In a large study of 279 Vietnam veterans, it was found that veterans who suffered penetrating head injuries during their service had higher ratings of violence, aggression, anger, and hostility than those without brain injury. Similarly, one study reported that 27.7 percent of the delinquent youths they studied had suffered significant head injury involving loss of consciousness/amnesia with ongoing cognitive or social impairment.

The severity of antisocial criminal behavior in individuals with TBI not only fails to improve but also becomes greater over time. For example, in a longitudinal study, it was found that the frequency of aggressive behavior and severity of temper bursts in a group of TBI patients increased over time, and the aggressiveness was reported by caregivers as moderate or severe in 31 percent of cases by 2 years post-injury. Similar patterns were documented in a 5-year study following 42 patients with severe TBIs. They exhibited a significant increase in threats of violence at 5 years after the injury (54 percent) compared to 15 percent at 1 year post-injury. They also noted that 7 percent of their sample had been in trouble with the law during the first year post-injury, and that the rate increased to 31 percent at 5 years post-injury. The arrest and conviction rates of individuals with TBIs were especially alarming when compared to the rate of 2 percent of the general population arrested annually. In addition, the increasing arrest and incarceration rates over the years post-injury **[p. 664 ↓]** raise concerns about the long-term effects of traumatic brain injuries.

These findings consistently show that individuals with TBI are more likely to misperceive elements of a situation (e.g., interpret other's sarcasm as a threat), make poor social judgments, overreact to provocative stimuli, and lack the communication skills to verbally dissolve the conflict, especially when the neural damage involves the frontal lobe. As a result, these emotional and behavioral dysfunctions associated with TBIs may increase the likelihood that one would resort to antisocial, criminal behavior when encountering complicated and conflicting situations. The supporting evidence from TBI studies provide strong evidence for a neurological basis of crime and support the theories in connecting deficits in the neural system including the frontal cortex and hippocampus to deviant behavior and violent offending.

Conclusion

The increasing evidence from lesion and brain imaging studies has confirmed the association between neurological deficits and crime, and has prompted the development of several theories including Moffitt's developmental theory, Damasio's somatic marker hypothesis, and Gray's dual biological model. Although supported by a number of empirical studies, these theories fall short in accounting for the wide range of criminal offending. These theories have provided empirically based

explanations for impulsive, aggressive types of criminal behavior. However, they are unable to predict the neural mechanisms underlying other types of offending such as instrumental aggression. Future theories incorporating findings from genetic imaging, neuropsychological, and brain imaging methods, while addressing the distinct neurological etiology underlying subgroups of criminal offenders, are needed for the development of a more comprehensive theory on neurology and crime.

YalingYang and AdrianRaine

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See also

- Brain Abnormalities and Crime
- Ellis, Lee: Evolutionary Neuroandrogenic Theory
- Fishbein, Diana H.: Biosocial Theory
- Mednick, Sarnoff A.: Autonomic Nervous System (ANS) Theory
- Moffitt, Terrie E.: A Developmental Model of Life-Course-Persistent Offending
- Psychophysiology and Crime

References and Further Readings

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