Part I

Introduction
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Neuroscience in Forensic Settings: Origins and Recent Developments
Anthony R. Beech and Dawn Fisher

Key points
- The aim of the chapter is to give both an overview and history of the burgeoning field of neuroscience.
- In the chapter, it is noted that the interest in understanding why individuals commit crime, from a neurobiological perspective, dates as far back as the early 19th century with Franz Joseph Gall’s phrenology and the work of Italian criminologist Cesare Lombroso.
- The heavy focus on the brain rather fell into abeyance in the early part of the 20th century, with there being more interest in sociological explanations of crime and only a relatively few researchers noticing the importance of the brain in understanding offending.
- An understanding of the relationship between brain dysfunction and criminal behavior really started to pick-up again in the 1980s. Attention started to turn to why humans need such large brains, and the idea that this is needed for coalition formation and tactical deception, which interestingly are rarely seen in other species (the social brain hypothesis).
- The most important area of the brain associated with social functioning is the limbic system. This area is a loosely defined collection of brain structures that play crucial roles in the control of emotions and motivation.
- It is noted that a number of genetic and environmental problems (e.g., adverse developmental courses, early deprivation, and other suboptimal rearing conditions) can have an effect upon these areas.
- The ensuing atypical morphological organization could result in social withdrawal, explosive and inappropriate emotionality, pathological shyness, and an inability to form normal emotional attachments (Joseph, 2003). It can also set the scene for later antisocial behaviors.
Structural and functional evidence and neuropsychological and neurophysiological evidence of problems in offenders are then outlined, as well as techniques to examine these problems.

The chapter also provides an outline of the structure of the book.

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**Terminology Explained**

The **autonomic nervous system** is a control system that acts largely unconsciously and regulates the heart rate, digestion, respiratory rate, pupillary response, urination, and sexual arousal. This system is the primary mechanism in control of the fight-or-flight response.

**Conduct disorder (CD)** in childhood is a repetitive, and persistent, pattern of behavior in which the basic rights of others or social conventions are flouted. Many individuals with CD show little empathy and concern for others, and may frequently misinterpret the intentions of others as being more hostile and threatening than they actually are.

**Cortisol** is a steroid hormone, and is produced in humans by the adrenal cortex within the adrenal gland. It is released in response to stress and low blood glucose. High levels are associated with social withdrawal.

**Epigenetics** refers to heritable changes in gene expression (active to inactive genes or vice versa) that do not involve changes to the underlying DNA sequence (i.e., is a change in phenotype without a change in genotype). Epigenetic change can be influenced by a number of factors including: age, environment, lifestyle, and disease state. New and ongoing research is continuously uncovering the role of epigenetics in a variety of disorders.

The **limbic system** is a collection of structures that includes the hippocampus, amygdala, anterior thalamic nuclei, fornix, columns of fornix, mammillary body, septum pellucidum, habenular commissure, cingulate gyrus, parahippocampal gyrus, limbic cortex, and limbic midbrain areas. It supports a variety of functions including emotion, behavior, and motivation. Emotional life is largely housed in the limbic system, and it has a great deal to do with the formation of memories.

**Monoamine oxidases (MAO)** are enzymes that are involved in the breakdown of neurotransmitters such as serotonin, norepinephrine, and dopamine. They are capable of influencing the feelings, mood, and behavior of individuals. A deficiency in the MAO-A gene has been shown to be related to higher levels of aggression in males.

**Neuroscience** is defined as the study of the brain and nervous system. It is a discipline that collaborates with other fields such as chemistry, computer science, engineering, medicine (including neurology), genetics, philosophy, physics, and psychology.
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Phrenology was a science of character divination, faculty psychology, theory of brain, and what the 19th-century phrenologists called “the only true science of mind.”

The thalamus is the brain’s “junction box,” its main functions include relaying motor and sensory signals to the cerebral cortex. It is located just above the brain stem between the cerebral cortex and the midbrain.

White and grey matter White matter (consisting of myelinated axons and glial cells) actively affects how the brain learns and functions. While grey matter is primarily associated with processing and cognition, white matter modulates the distribution of action potentials, acting as a relay and coordinating communication between different brain regions.

XYY syndrome is a genetic condition in which a human male has an extra male (Y) chromosome, giving a total of 47 chromosomes instead of the more usual 46. This produces a 47,XYY karyotype, which occurs every 1 in 1,000 male births. The syndrome has been associated with increased risk of learning disability and criminal behavior in some cases.

Introduction

There is a growing body of evidence that suggests that predisposition to offend can be associated with genetic, hormonal, or neurobiological factors. Nita Farahany, Professor of Law at Duke University, North Caroline, USA, and an advisor on President Obama’s bioethics advisory panel, reported at the Society for Neuroscience in San Diego in 2013 on more than 1,500 judicial opinions in which a judge mentioned neurological or behavioral genetic evidence that had been used as part of a defense case in a criminal trial. Specifically, she noted that: “the biggest claim people are making is: ‘Please decrease my punishment because I was more impulsive than the next person, I was more likely to be aggressive than the next person, I had less control than the next person’” (reported by Stix, 2013).

Of course, the rise of so-called “neurolaw” cases is becoming more pressing in that forensic practitioners are grappling with understanding the impact neuroscience is having upon the forensic field, both in terms of the court system, in a number of countries, and in producing effective treatments to reduce re-offending. As for the former, courts (particularly in the USA) are facing a huge increase in the numbers of legal councils mounting sophisticated defenses to indicate that individuals are not fully responsible for their crimes, due to their “dysfunctional” brains. As Farahany noted, the use of such brain science evidence is “challenging fundamental concepts of responsibility and punishment.” As for the treatment question, Farahany added, “Should we hold people responsible for their actions … or do we need to rethink what we do and instead focus more on rehabilitation?” Stix (2013) observed, “whichever way things go, jurors and judges are going to be hearing a lot more about [the] amygdala and orbitofrontal cortices.” (see Box 1.1 for a description of these areas).

As for the etiology of offending behaviors, we are probably in a better position than ever before to understand how offending may come about, through the interaction between impact of genetic and environmental factors and their effect upon the brain,
and how such an understanding can affect what we are able to do in treatment. We are clearly not currently in a position to tell a parole board, for example, to release someone based on a brain scan; but we may be nearer than we think in getting to this position. Hence, the aim of this book is to outline the importance of neuroscience to the understanding of the etiology of criminal behaviors, and to pull together the extant literature regarding forensic neuroscience.

Of course, this is an ongoing process, but what this volume attempts to do is to take stock of where we are in such an understanding of what neuroscience can tell us about offending. It goes without saying that any complete answer will encompass evolutionary, genetic, biochemical, neuropsychological, and cognitive factors as well as social factors (familial and societal), all of which will be described in some detail in this book. The genesis of the book came about through conversation between the editors regarding the understanding of sexual offending from a neuroscientific perspective. This seemed a tall order in. But once we started to mull over the idea we came up with an even bigger plan – that this should also include a wider consideration of the issues that forensic researchers, practitioners, and students in the field are currently grappling with, namely the profound leaps forward in knowledge that have been made in the last ten years in the understanding of the brain. It is self-evident that at the root of (anti)social behaviors are feelings, cognitions, and actions underpinned by the neurobiological actions in the brain. We will now give a brief history of the background and developments in neuroscience preceding the evidence base contained in the chapters of this book.

**Forensic Neuroscience: Origins and Developments in 19th-Century Phrenology**

Understanding why individuals commit crime from a neurobiological perspective probably dates back to Franz Joseph Gall in the 19th century and the pseudoscience of phrenology (although techniques such as trepanning – a surgical intervention in which a hole is drilled into the skull, to treat health problems related to intracranial diseases – predate these ideas by several millennia. Since Neolithic times, a person who was behaving in what was considered an abnormal way had holes drilled into them to let out “evil spirits”). Phrenology generally has had a bad press over the last 100 years, but Rafter (2005) noted that “Phenology [at its inception] produced one of the most radical reorientations in ideas about crime and punishment ever proposed in the Western world.” (p. 65). She further noted that this approach was instrumental in: (1) developing a rehabilitation model (going against the 19th-century tide of retribution); (2) opposing capital punishment; and (3) proposing sentencing policy that was way ahead of its time. The system originally developed by Gall (1835) was based on the following propositions:

1. The brain is an organ of the mind.
2. The brain is as an aggregation of 52 different organs grouped around the following: ten **props** (from adhesiveness to secretiveness); four **lower sentiments** (cautiousness to truthfulness); nine **superior sentiments** (benevolence to wonder); 17 **intellectual faculties** (coloring to weight); and two **reflecting faculties** (causality and comparison).
The relative size of the organs can be increased through exercise and discipline. The more active the organ is the larger its size. The relative size of the organ can be estimated by inspecting the contours of the skull.

Phrenology can therefore be seen as producing the first “comprehensive explanation of criminal behavior” (Rafter, 2005, p. 66), in that through this complete description of nearly all cognitions, emotions, behaviors, and phenomenological experience, every form of criminality can be explained. However, like many other 18–19th-century pseudosciences, it soon became little more than mere entertainment with the reading of personality from the bumps on an individual’s head. Interestingly, though, some aspects of phrenology have influenced the concepts of deviance in the 20th century (Rafter, 2005), and the notion of brain modules continued to have a long history, for example, through Chomsky’s language module (1980) and Fodor’s (1983) modularity of mind. More recently, the concept of the brain as a series of subsystems with different structures and function has gained increasing support from structural and functional scanning techniques as outlined in Boxes 1.1 and 1.2, although sceptics of such scanning techniques have disparagingly likened the results of this work to little more than “colored” phrenology.

The Case of Phineas Gage

As for problems in specific areas of the brain causing antisocial behaviors, probably the best-known case study is the 19th-century case of Phineas Gage. Gage was a foreman for the Rutland and Burlington Railroad in the USA who suffered a serious brain injury. Contemporary accounts indicate that he was a highly regarded “model citizen” prior to his accident. He was always on time for work, never swore, and abstained from tobacco and alcohol use (Beech, Nordstrom, & Raine, 2012). However, in September 1848, while supervising the blasting of rock to clear the way for more railway track to be laid, the tamping iron to compact the gunpowder that was used scraped the side of the hole generating a spark, which prematurely ignited the explosive. This caused the tamping iron to shoot out of the hole, enter Gage’s head from under his chin, and pass straight through his skull. He was knocked unconscious but, despite serious injury, he surprised everyone by regaining consciousness almost immediately, talking, sitting up, and walking to the horse-drawn cart that took him to seek medical attention. Dr. John Harlow carried out extensive work on Gage including combating an infection that occurred sometime after the accident.

Most of the first-hand information about Gage before and after the accident comes from Harlow, who noted that Gage was a dramatically changed man after the accident – becoming impulsive, irascible, unreliable, and rude. Specifically, he summed up Gage’s personality change by saying, “the equilibrium … between his intellectual faculties and his animal propensities seems to have been destroyed.” His friends simply said that Gage “was no longer Gage.” As a result of the changes in his personality, the railroad refused to reinstate Gage as a foreman in their company. So he began traveling around New England instead, as an itinerant, displaying himself in travelling circuses. There is a slightly happier end to his story: Gage eventually found gainful employment driving a horse-drawn carriage.
As for the actual damage to Gage’s brain, Damasio, Grabowski, Frank, Galaburda, and Damasio (1994) reconstructed his skull from previously taken measurements, and suggested that Gage had suffered damage to his left and right prefrontal cortices, specifically damaging the lower medial parts of the prefrontal cortex, an area known as the ventromedial prefrontal cortex (vmPFC), part of the orbital prefrontal cortex (OPFC) (see Box 1.4 for a description of the functions of this area of the brain). Damasio et al. suggested that such damage to the part of the brain that we now know is responsible for higher order executive functioning, and the modulation of emotional processing, led to the profound changes observed in Gage’s personality and behavior. However, this conclusion may be not as clear cut as it seems. Two more recent studies (i.e., Ratiu & Talos, 2004; Van Horn et al., 2012) have questioned Damasio et al.’s conclusions as to the amount, and type, of brain damage that Gage actually suffered. For example, Van Horn et al. (2012) examined millions of possible trajectories for the iron rod, and ruled out all but a few, concluding that the rod could not have crossed over to the right hemisphere. They further speculate that it may be only 4% of Gage’s grey matter that was actually destroyed, while more than 11% of his white matter suffered damage, including damage to the tracts that connect into both hemispheres. Van Horn et al. in fact compare this damage to that observed in neurodegenerative diseases such as Alzheimer’s, in that Gage could have been displaying some of the symptoms of this disorder, such as an inability to complete tasks, poor judgment, and changes in mood and personality. Despite different interpretations, the tamping iron clearly destroyed a significant amount of brain tissue, and the flying bone “shrapnel,” and subsequent infections would have produced further damage to Gage’s brain. What this case does indicate is that damage to the brain has a clear effect upon an individual’s behavior.

Other Early Genetic and Neurobiology Insights

In the 19th century the Italian criminologist Cesare Lombroso (1835–1909) attempted to explain criminality and took into account genetic and organic factors. He was the first to formally classify criminals in his influential criminological work L’uomo delinquente (1876), and also probably the first to think about understanding and treating offenders (see Box 1.1 for his typology of offenders).

**Box 1.1 Lombroso’s (1876) Typology**

- **Born criminals** – degenerate, primitive offenders who were lower evolutionary reversions in terms of their physical appearance.
- **Criminaloids** – those without specific characteristics but whose mental and emotional make-up predisposes them to criminal behavior under certain conditions.
- **Insane criminals** – those suffering from mental/physical illnesses/deficiencies.
Lombroso’s research methods were both clinical and descriptive, with precise details of skull dimension and other measurements. His methods can be seen to be broadly influenced by Darwin’s evolutionary theory, in that he believed that criminals represent a reversion to a more primitive state of being, and that such individuals will behave contrary to the rules and expectations of modern civilized society. As for the three types of criminal outlined in Box 1.1, he suggested that born criminals (or evolutionary “throwbacks”) could be identified by the following:

- A sloping forehead
- Ears of unusual size
- Asymmetry of the face and the skull
- Excessive length of arms and other physical abnormalities
- Less sensibility to pain and touch

Less controversially Lombroso noted the following psychological characteristics of criminals:

- A lack of moral sense, including an absence of remorse
- Vanity
- Impulsiveness
- Vindictiveness
- Cruelty
- Excessive use of tattooing
- [But interestingly] acute insight

Lombroso notes that the psychological characteristics outlined above underpin a “moral insensitivity.” Individuals with these problems, but without the overt anatomical differences outlined above, were termed criminaloids. Today such individuals would probably be described as either having antisocial personality disorder (ASPD) (see Chapter 10) or in extremis psychopathy (Hare, 1991; 2003) (see Chapter 9). Lombroso also described insane criminals, who would nowadays be described as individuals with mental health problems (mental illness as a risk factor is described in Chapter 20).

Contrary to current popular opinion, Lombroso recognized the interaction between predisposing organic and genetic factors, and precipitating factors such as an individual’s environment. But although he gave recognition to such psychological and sociological factors in the causes and background to crime, he remained convinced of criminal anthropometry (measurement of the human individual); and this, plus its association with eugenic ideas, meant that (quite rightly) such ideas fell into disrepute.

Lombroso’s ideas regarding predisposing organic and genetic factors were most notably taken up by Kraepelin (1856–1926). In many ways his ideas formed the basis for later psychiatric classification and for the proposition that different mental illnesses stem from discrete areas of the brain. Hence, Kraepelin is widely regarded as the founder of modern psychiatry, psychopharmacology, and psychiatric genetics. As for his ideas on criminality, in the various editions of his influential psychiatry textbook Psychiatry: A Textbook for Students and Physicians (Kraepelin, 1899) there is a section on moral insanity (i.e., a disorder of the emotions or moral sense without apparent delusions or hallucinations). This can probably best be described as a psychiatric
A redefinition of Lombroso’s “born criminal,” although Kraepelin noted that it was not yet possible to recognize such individuals by their physical characteristics.

This concept of moral insanity was in fact strongly influenced by the work of Philippe Pinel (1745–1826) (founder of the Pinel Institute) who noted that sufferers were “mentally ill” in just one area, while their intellectual faculties were unimpaired in other areas of functioning. The psychiatrist Julius Koch (1841–1908) sought to make the moral insanity concept more scientifically rigorous and suggested the phrase *psychopathic inferiority* (later *personality*) should be used instead. This referred to continual and rigid patterns of misconduct or dysfunction in the absence of apparent intellectual disability or illness. The diagnosis was meant to imply a congenital disorder, and to be made without moral judgment. Whitlock (1982) noted that the definition was later changed to *moral imbecility*, which is akin to what we now term *psychopathy*.

As for early notions of psychopathy, from 1904 onwards, versions of Kraepelin’s textbook included a chapter on *psychopathic personalities*, where four types are outlined: (1) born criminals, (2) pathological liars, (3) querulous persons, and (4) *Triebmenschen* (persons driven by a basic compulsion, including vagabonds, spendthrifts, and dipsomaniacs [alcoholics]). However, an examination of Kraepelin’s work would suggest that he had no evidence or explanation suggesting a congenital cause. For example, Kurt Schneider (1887–1967) criticized Kraepelin’s categorical system (Schneider, reprinted in 1976) for appearing to be a list of behaviors that were considered undesirable, rather than specific medical conditions. Early versions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (e.g., American Psychiatric Association (APA), 1952), can be criticized for the same reason; homosexuality, for example, is described as a “sociopathic personality disturbance,” and in fact homosexuality was not actually removed from the DSM until the seventh printing in 1974.

It would be remiss of us not to note that Kraepelin was a strong and influential proponent of eugenics and racial hygiene, and the appropriate backlash against these ideas, together with the growing influence of psychodynamic theory (Freud, 1904), with its emphasis on drives, and unconscious psychodynamic processes, broadly meant that any study of the genetic or biological underpinning of criminality was broadly frowned upon (even though Freud was, by background, a neurologist!) for the first half of the 20th century. For example, the first edition of the DSM (APA, 1952), was heavily influenced by Freudian ideas about neurosis, psychosis, and “character disturbances,” without ascribing any neurobiological underpinnings to these disorders. In fact, the inclusion of a section on genetic, physiological, and prognostic risk factors, as related to ASPD, has only been fully described in the DSM 5. These observations may explain a relative dearth of research from anything other than psychodynamic, drive theory, and sociological perspectives in explaining and understanding crime until the second half of the 20th century.

**Approaches to Explaining Crime from a Brain-based Perspective**

A few psychologists and psychiatrists in the 1950s and 1960s can be seen as having bucked the trend for mainly sociological or criminological explanations of crime. Probably the foremost psychologist, in the UK, was the personality theorist Hans Eysenck who, in his book *Crime and Personality* (1964), noted that some individuals are
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predisposed to crime through having a particular personality type. Specifically, he argued that criminals would be highly extraverted (E) and highly neurotic (N). Eysenck was keen that any identified personality dimensions were orthogonal (i.e., had no relationship to each other – extraversion as being essentially unrelated to neuroticism). By the removal and addition of a number of items to tap different dimensions of personality, he later added a dimension of psychoticism (P). However, in order to produce such a third orthogonal scale, he ended up with a measure that is really more akin to psychopathy, in that individuals scoring highly on the P scale are described as aggressive, antisocial, cold, and egocentric.

Eysenck hypothesized that extraversion was associated with an under-arousal of the cortex due to low functioning of the ascending reticular activation system (the part of the brainstem that plays a central role in bodily and behavioral alertness). Therefore, extraverts are chronically under-aroused, constantly seek stimulation (to stay awake), and are difficult to condition (i.e., they have an inability to learn the associations between their behaviors and rewards/punishments). In contrast, introverts are over-aroused, hence they have a tendency to avoid arousing situations, and easily learn from experience. Neuroticism in Eysenck’s system is associated with the lability of the autonomic nervous system. People with very labile autonomic nervous systems can be seen as being highly anxious. Eysenck did not really report any neurobiological underpinnings of P and the concept of impulsivity (one of the primary drivers of many types of violent and acquisitive offending) remained elusive in Eysenck’s system. In early factor analyses impulsivity was seen as part of extraversion, but then it cropped up on the psychoticism dimension. In fact, it was only relatively later that Eysenck and Eysenck (1978) reported impulsivity as a personality construct in its own right, together with venturesomeness and empathy. More will be discussed about the neurobiological basis of these constructs in Part II of the book.

Eysenck suggested that people who are highly extraverted and neurotic do not condition easily, and therefore their behavior is developmentally immature, in that it is selfish and concerned with immediate gratification. As they do not learn easily, they respond to antisocial impulses with high levels of anxiety. The predictions from Eysenck’s theory are that offenders will have higher E, N, and P scores. Rushton and Chrisjohn (1981) compared E, N, and P scores with self-reports of delinquency in adolescents, finding some support. However, Farrington, Biron, and LeBlanc (1982) looking at official studies of delinquency, found that such individuals have higher P scores (not unsurprising given that P is in part a measure of aggressive antisociality), and higher N scores, but not E. Importantly, impulsivity, as a crucial personality dimension, was found in this study (as it has in a number of other studies) to be a better predictor of subsequent offending. But like many theories of criminology since Lombroso, Eysenck’s theory tells us little about why some people commit the crimes that they do. However, Eysenck’s ideas do flag up the notion that underlying tendencies towards crime are identifiable in childhood, and hence that it may be possible to modify socialization experiences so that some individuals do not become criminals in adulthood. These ideas are more fully covered in Part V, where some potential interventions to reduce crime by addressing problematic brain function are outlined.

As for specific genetic explanations of crime, there was a great deal of excitement in the 1960s when a number of researchers (e.g., Jacobs, Brunton, Melville, Britain, & McClement, 1965; Price & Whatmore, 1967) reported that a specific genetic
marker (an extra Y chromosome in men) was a physical indicator for criminal behavior. The condition occurs in 1 in 1,000 boys, individuals may be taller than average, and the condition can be associated with an increased risk of learning disabilities and delayed development of speech and language skills. A small percentage of males with 47,XYY syndrome are diagnosed with autistic spectrum disorders (see Chapter 11). However, in a recent study, Stockholm et al. (2012) found that the incidence of convictions was increased in those with the syndrome, compared to controls. However, adjusting for socioeconomic variables (education, fatherhood, retirement, and cohabitation) reduced offending levels to similar levels as the controls, although, some specific crime types (sexual abuse, arson, etc.) remained increased. This study’s authors suggested that the increased risk of convictions may be somewhat explained by the poor socioeconomic conditions related to the chromosomal aberrations rather than the aberrations themselves.

In the 1980s attention started to turn to why humans need such large brains. The adult brain weighs about 2% of the individual’s weight, but consumes about 20% of total energy take (Aiello & Wheeler, 1995). Dunbar (1998) commented that it is difficult to justify why we, and other primates, need larger brains than other species to perform in the same ecological niche. Hence, in the 1980s, a social brain hypothesis was put forward that suggests that our large brains, and those of other primates, reflect the computational demands that characterize our environments. Additionally, in some higher primates there is, from time-to-time, tactical deception (Whiten & Byrne, 1988) and coalition formation (Harcourt, 1988), which are rarely observed in other species. These noted manipulations led to the development of the Machiavellian intelligence hypothesis to explain the size of our brains (Byrne & Whiten, 1988). We will now examine some of the areas of the social brain.

The Social Brain

As a brief introduction, the most important area of the brain associated with social functioning is the limbic system, involving areas in the midbrain and the cerebral cortex. This area is a loosely defined collection of brain structures that play crucial roles in the control of emotions and motivation. The principal limbic structures involved are the amygdala and the anterior cingulate cortex along with the orbital prefrontal cortex and associated areas of the brain including the insular, as shown in Box 1.2.

Box 1.2 Important Areas Underpinning the Social Brain

The orbitofrontal cortex (OFC) is situated at the very front of the brain, is considered to be the apex of the neural networks of the social brain, and is critical to the adaptation of behavior in response to predicted changes in reinforcement. It bridges the cognitive analysis of complex social events taking place within the cerebral cortex, and emotional reactions mediated by the amygdala and the autonomic nervous system. The orbitofrontal cortex therefore acts as a “convergence zone” with its connections allowing it to integrate internal and external information. As this is part of the brain associated with reasoning, it would be expected
to be under the most intense evolutionary pressure to improve the effectiveness of its functioning. The vmPFC is the medial part of the orbitofrontal cortex and is associated with morality and bodily awareness. It was also implicated as being abnormal in psychopathic individuals (Blair, 2007).

The amygdala is a set of almond shaped interconnected nuclei (large clusters of neurons) found deep within the temporal lobes, which are on the left and right sides of the brain. Amygdala functions are related to arousal, the control of autonomic responses associated with fear, emotional responses, and emotional memory, and are therefore centrally involved in attention, learning, and affect. The amygdala can be split into two major subdivisions: the basolateral complex and the centromedial complex. The basolateral complex can be roughly thought of as being the principal input region of the amygdala with afferents (incoming projections) arising principally from the OFC and the hippocampal regions (to do with memory), which exerts potent effects upon sexual behaviors. The basal nuclei, in conjunction with the lateral nuclei, also play a role in reinforcement more generally. The centromedial complex is involved in responding to fearful stimuli. The sensory inputs that drive these fear responses arise principally from cortical and thalamic projections to the lateral nuclei of the amygdala. These sensory inputs form synapses, which have a high degree of plasticity. This enables encoding of conditioned emotionally significant stimuli, and enables the amygdala to play a central role in aversive conditioning. The medial nuclei of the centromedial complex are also responsible for sexual and reproductive behaviors.

The anterior cingulate cortex (ACC), situated below the cerebral cortices and wrapped around the corpus callosum, first appeared in animals demonstrating maternal behavior. This indicates that the ACC appears to provide the basic circuitry for communication, cooperation, and empathy. The ACC is involved in the simultaneous monitoring of personal, environmental information and allocation of attention to the most pertinent information in the environment and a particular moment in time. The ACC can be subdivided into affective and cognitive parts and therefore integrates emotional and attentional processing.

The insular cortex is a portion of the cerebral cortex folded deep within the lateral sulcus (the fissure separating the temporal lobe from the parietal) situated behind the frontal lobe and frontal lobes. It is a long-neglected brain region that has emerged as crucial to understanding what it feels like to be human. It is suggested that it is the source of social emotions like lust, disgust, pride and humiliation, guilt and shame. Together with the premotor cortex, it is part of the circuitry that allows us to vicariously share the actions and emotions of others. Hence, this area of the brain helps give rise to moral intuition, empathy and the capacity to respond emotionally.

The basal ganglia are a set of interconnected nuclei in the forebrain that are strongly interconnected with the cerebral cortex as well as several other brain areas. The basal ganglia are associated with a variety of functions including procedural learning, routine behaviors, cognitions, and emotions.

The habenula is a pair of small nuclei situated above the thalamus that receives information from the limbic system and basal ganglia. It sends information to
areas of the midbrain that are involved in dopamine release. The habenula also
has neurons that project to areas like the raphe nuclei, which are involved in sero-
tonin release. Therefore, the habenula is one of the few known structures in the
brain that can exert influence on the experience of reward (through dopamine
release) and mood (serotonin release).

Mirror neurons can be seen as providing the infrastructure for empathy, in
that they are a specific form of nerve cell that fire both when an individual acts
and when the individual observes the same action performed by another. Thus,
the neuron “mirrors” the behavior of the other. A number of experiments using
functional fMRI, EEG, MEG (see Boxes 1.3 and 1.4 for a description of these
imaging techniques) have shown that certain brain regions (in particular the
anterior insula and ACC) are active when people experience an emotion (dis-
gust, happiness, pain, etc.) and when they see another person experiencing an
emotion.

Although a great deal is now being written about the neurobiology/neurochemistry
of the “social brain,” relatively less has been written about these in relationship to
offending. But now a number of lines of evidence have pointed to impaired structure
and function in areas of the social brain (i.e., the amygdala, the OFC, and the ACC, the
insula), and in other associated areas. Problems here, it is argued, lead to increases
in violence and instrumental aggression, and, in extremis, ASPD and psychopathic
behaviors. These issues are further explored in Part III of this book.

Recent Approaches to Understanding Criminality from a
Neurobiological Perspective

It is fair to say that Adrian Raine (1993) really set the scene for current thinking about
the origins of crime in his book The Psychopathology of Crime: Criminal Behavior as
Clinical Disorder. In the preface to this book he referred to “the rapidly growing and
influential body of knowledge on the biological bases of criminal behavior,” observ-
ing that “if we are to fully understand criminal behavior we need to fully are of all
influences that bear on it” (p. xvii); that is to say biological as well as psychosocial and
environmental factors. In Raine’s seminal work he covers evolutionary, genetic, neu-
rochemistry, brain imaging, psychophysiology, as well as what he called other biological
factors, such as head injury, pregnancy, birth complications, diet, cognitive influences,
and familial and non-familial influences.

Although Raine’s ideas were deeply unpopular in some quarters at the time, this
approach has certainly come of age, given what we now know about brain structure
and functioning through some of the techniques outlined in Boxes 1.3 and 1.4. In
fact, great strides have been made in the last 20 years in understanding how the brain
actually works, and how it is wired up, as well as the functions of different parts of the
brain and how they function together, using the developing technologies outlined in
Boxes 1.3 and 1.4.
Box 1.3 Techniques Used to Examine the Structural Integrity of Different Areas of the Brain

**Computerized axial tomography (CAT)** scans are produced using a series of X-rays taken along the axis of the body. The X-rays pass unevenly through tissues of different densities, allowing for distinctions between fluid, bone, and brain tissue to be made. A computer then assembles these “slices” into a sequence of cross-sectional images.

**Magnetic resonance imaging (MRI)** scans are created by using powerful magnetic fields to orient all of the hydrogen atoms (primarily found in water molecules) in the brain in the same direction. A radio frequency electromagnetic field is introduced, which produces a signal that is detected by the MRI scanner’s receiver. These signals are then assembled into high-resolution images that can distinguish the grey from the white matter of the brain. MRI scans do not use radiation and produce more detailed pictures than do CAT scans, but they also take much longer to obtain and are much more expensive. Both types of imaging produce images of brain structures that can then be measured and studied.

**Diffusion tensor imaging (DTI)** is a relatively new technique, allowing images to be taken of the structural integrity of the white matter tracts connecting various parts of the brain.

Box 1.4 Techniques Used to Examine the Functional Aspects of Different Areas of the Brain

In **electroencephalography (EEG)** the subject has electrodes placed in specific points over the scalp. These electrodes detect the brain’s electrical impulses, which are then recorded and analyzed by a computer. The frequency and amplitude of the resultant signals can then be interpreted. Increasing frequency is associated with increasing arousal, and lower frequency is associated with lower arousal in particular areas of the brain.

**Photon emission tomography (PET)** is a technique that relies on injecting subjects with a radioactively labelled substance, such as glucose. Images of their brains can then be obtained, showing areas of higher radioactive signal due to glucose metabolism, which indicates level of neural activity.

The **single photon emission tomography (SPECT)** form of imaging also involves the injection of a radioactive tracer. The camera detects the amount of radiation coming from different parts of the brain. These differences are due to variations in **regional cerebral blood flow (rCBF)** and reflect different levels of activity in various parts of the brain.

**Functional magnetic resonance imaging (fMRI)** is an imaging technique that measures changes in blood oxygen in regions of interest in the brain before and after cognitive tasks are undertaken. These **blood oxygen level dependent**
(BOLD) signals are used as a proxy for how active a region of the brain is. By comparing groups of interest with matched controls, the patterns of activation, or inactivation, in their brains can be studied to learn how the functioning of various brain regions relates to the condition in question.

**Magnetoencephalography (MEG)** is a functional neuroimaging technique for mapping brain activity by recording magnetic fields produced by electrical currents occurring naturally in the brain, using very sensitive magnetometers. Arrays of superconducting quantum interference devices (cooled by liquid helium) are currently the most common magnetometer. This technique is used in clinical settings to find locations of abnormalities, as well as in experimental settings, to measure brain function.

Techniques like these allow us to examine the brains of criminals in order to give a better idea of the problems that many such individuals face. This is not to say we are at any level advocating genetic determinism to criminality, but, as outlined in Chapter 19, in many cases offenders have had to cope with a number of adverse risk factors (i.e., prenatal, perinatal, diet, traumatic head injury) and, as noted in that chapter, these can have a very big effect upon brain structure and function. Other studies (e.g., as outlined in Chapter 18 by Nathalie Fontaine and colleagues) suggest that recent twin and molecular genetic studies have the potential to inform a model of developmental vulnerability to offending more generally, and psychopathy in particular. Hence, in Part V of this book on rehabilitation, there are chapters describing interventions that we would suggest can ameliorate, to some extent, problems that either have a genetic basis or have arisen through adverse upbringings. We will now provide brief digression about the social brain, but of course the book itself will provide a lot more detail of these and other brain areas implicated in offending.

We will now very briefly outline some evidence of structural and functional problems that have been previously observed in offenders (until recently this has been mainly regarding psychopaths, ASPD, and CD individuals). These ideas will be broadened and deepened in subsequent chapters of this book.

### Structural and Functional Evidence of Problems in Offenders

Raine, Lencz, Bihrlle, LaCasse, and Colletti (2000) examined individuals with ASPD and compared them to a matched group of substance users, and non-offending controls. They found an 11% reduction in the grey matter of the OPFC of the ASPD group compared to the other two matched groups. Other researchers have found that, compared to non-offender controls, ASPD individuals had smaller temporal lobes (Dolan, Deakin, Roberts, & Anderson, 2002; Laakso et al., 2002), as well as reductions in their dorsolateral, medial frontal cortices, and the OPFC (Laakso et al., 2002). Laakso et al. (2000) found violent offenders with alcoholism and ASPD had smaller posterior hippocampi (an area that is associated with fear conditioning). Huebner et al. (2008) found smaller grey matter volumes in the OPFC and the temporal lobes of children with CD compared to normal controls. Sterzer, Stadler, Krebs, Kleinschmidt, and...
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Poustka (2005) found reduced grey matter volumes in the amygdala and the insular of adolescents with CD compared to normal controls. Kruesi, Casanova, Mannheim, and Johnson-Bilder (2004) reported that diminished right temporal lobe volume (which includes the amygdala) was associated with CD.

As for functional evidence of problems in offenders, Birbaumer et al. (2005) found that psychopaths show no significant activity in the limbic-prefrontal circuit (amygdala, OPFC, insula, and the ACC), using fMRI, during a task involving verbal and autonomic conditioning. This lack of recognition of fear in psychopaths, due to a measurable lack of amygdala and insular function, suggests that it is easier to offend, as a key component of committing interpersonal violence towards others is the requirement to not recognize, understand or empathize with the mental state of victims.

Raine et al. (1993) in a PET study, found that a sample of murderers demonstrated reduced glucose metabolism in the OPFC, the anterior medial and superior frontal cortices compared to a normal comparison group, after a continuous performance task. A follow-up study with a larger sample using a similar methodology found the same pattern of reduced glucose metabolism in the anterior frontal cortices, and in the amygdala and hippocampus as well (Raine, Buchsbaum, & LaCasse, 1997), suggesting reduced functions in these areas.

Sterzer et al. (2005) examined patterns of brain activation, using employed fMRI, in CD adolescent males compared to controls, as they looked at neutral pictures and pictures with a strong negative affective valence. It was found that when the CD youths viewed the distressing pictures they had significantly reduced activity to their left amygdalae compared to the controls. Marsh et al. (2008) using a similar methodology studied children and adolescents with callous-unemotional traits (CU) and controls. Those with CU traits demonstrated significantly reduced amygdala activation on viewing the fearful (but not the angry or neutral) faces. Further, on a functional connectivity analysis, the CU children showed reduced connectivity between the ventromedial prefrontal cortex and the amygdala. The degree of reduction in this connectivity was negatively correlated with the score on a scale that measured the degree of CU traits. Similar findings have been described in adult populations (Muller et al., 2003; Kiehl et al., 2004).

Neuropsychological and Neurophysiological Evidence of Problems in Offenders

Neuropsychological tests have provided another method for testing the functional level of various brain areas. One of the most consistent findings in the neuropsychological aspects of criminality is that antisocial populations have lower verbal IQs compared to non-antisocial groups even in adolescence (Brennan, Hall, Bor, Najman, & Williams, 2003; Déry, Toupin, Pauzé, Mercier, & Fortin, 1999). Additionally, researchers have found that verbal deficits on testing at age 13 predict delinquency at age 18 (Moffitt, Lynam, & Silva, 1994). However, it should be noted that such neuropsychological deficits show interactive effects with social risk factors (Aguilar, Sroufe, Egeland, & Carlson, 2000; Brennan et al., 2003). Other neuropsychological tests have focused on how antisocial populations respond to affectively charged stimuli. For example, Loney, Frick, Clements, Ellis, and Kerlin (2003) found that juveniles with CU traits showed slower reaction times after being presented with emotionally
negative words, while those with impulsive traits showed faster reaction times to such stimuli.

A number of studies have also found biological correlates to be predictive of criminal behavior. Probably the longest term, and most important of these, is the Cambridge Study in Delinquent Development, headed by David Farrington, which is a prospective longitudinal survey of the development of offending and antisocial behavior in 411 males, all living in a deprived inner-city area of South London, first studied at age 8 in 1961. The findings of this study describe these individuals’ criminal careers up to age 50, looking at both officially recorded convictions and self-reported offending. The most important risk factors were family criminality, risk taking (the neurobiology of which is described in Chapter 5), low school attainment, poverty, and adverse parenting. A series of analyses were also carried out to identify the most predictive risk factors for violence (Farrington, 1997). Only two risk factors were identified here – low resting heart rate and poor concentration – which were found, independently of all other potential risk factors, to predict violence.

Low resting heart rate is, in fact, the best-replicated biological correlate of antisocial behavior in juvenile samples, in that in longitudinal studies, low resting heart rate has been shown to accurately identify individuals who are at risk of later developing antisocial behavior (Ortiz & Raine, 2004). This finding has been replicated in many countries (Farrington, 1997; Mezzacappa et al., 1997; Moffitt & Caspi, 2001; Raine, Venables, & Mednick, 1997). However, it should also be noted that having a high resting heart rate is negatively correlated with later violent behavior (Raine, Venables, & Williams, 1995).

Raine (2013) notes that there are several different possible explanations for these findings. One is that a low resting heart rate is associated with a lack of fear, in that people whose heart rates are relatively low are relatively more fearless than the rest of the population. Raine notes that bomb disposal experts, for example, have low heart rates. Another explanation for these results is “sensation-seeking.” Low heart rate has been linked with low physiological arousal (Ortiz & Raine, 2004), and hence some individuals may seek out stimulation to increase arousal. However, again, although low resting heart may be a bio-marker there is still an interaction between biology and the environment. Another area of interest to researchers attempting to understand from a biological level why some people commit antisocial acts is brain chemistry. Here work has looked at, for example, the relationship of testosterone and with aggressive behavior (e.g., Archer, 1991). Herbert (2015) noted that without testosterone none of us would exist, as its main purpose is to drive reproductive function in men. Testosterone is also central to sexual behavior in humans (Bancroft, 2009), in that sexual activity increases the level of testosterone, as does talking to an attractive other. However, there is an interaction between an array of neural and hormonal systems (including the neurotransmitters serotonin and dopamine) as well as sexual activity.

Clear evidence suggests that testosterone is one of the primary hormones underpinning aggression in non-human animals, such as rats, monkeys, hamsters, dogs, and deer (Rada, Kellner, & Winslow, 1976). Vom Saal (1983) proposed a model of testosterone and its relationship to aggression. According to this model, androgen prenatally influences the neural networks that mediate aggressive behavior. When these networks are again exposed to androgens (due to this hormone’s release from the testes, where about 95% is located, with the other 5% in the adrenal glands) in males at puberty and beyond, the neural networks are again activated and aggression is released in
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relevant environmental stimuli, such as protection of territory, fighting over females, or position in a dominance hierarchy. Testosterone release in humans starts two weeks after birth and lasts until six months, where the child’s testes are producing as much testosterone as an adult male, then recedes, and returns at high levels in puberty and adulthood.

However, adult human male testosterone levels vary in different circumstances, and are associated moderately with levels of aggression and violence. In a meta-analysis of 106 articles in the field reporting the relationship, Book, Starzyk, and Quinsey (2001) only found a weak correlation ($r = .14$) between levels of testosterone and aggression. As the square of the correlation gives the amount of variance accounted for from one variable to another in a correlation, then it can be that testosterone only accounts for around 2% of the variance in aggression across a number of studies.

It might be expected that those involved in warfare would have increased levels of testosterone, but in fact they often have levels similar to men who have been castrated. Here it is the stress hormone cortisol that significantly reduces the level of testosterone. For example, a study from the 1990s (Dabbs, Jurkovic, & Frady, 1991) examined testosterone and cortisol levels in male adolescent offenders. While they found that offenders with high levels of testosterone committed more crimes, there was a significant interaction between testosterone and cortisol, with the latter moderating the effect of testosterone.

Seo, Patrick and Kennealy (2008) have suggested that other neurochemical imbalances, specifically reduced levels of serotonin and higher levels of dopamine in the prefrontal cortex (the apex of the social brain, see Box 1.2), are also implicated in impulsive aggression. Serotonin hypofunction may represent a biochemical trait that predisposes individuals to impulsive aggression, while dopamine hyperfunction contributes in an additive fashion to the serotonergic problems.

Another example, of gene/environment interaction, is work that has found a connection between a version of the MAO-A gene (3R) (commonly known as the “warrior gene”) and several types of antisocial behavior. Although overall MAO-A has been found to have no overall effect on antisocial behavior, low MAO-A activity in combination with abuse experienced during childhood results in an increased risk of aggressive behavior as an adult (Frazzetto et al., 2007). But high testosterone, maternal tobacco smoking during pregnancy, poor material living standards, dropping out of school, and low IQ can also trigger violent behavior in men with the low-activity alleles (which are overwhelmingly the 3R allele) (see, e.g., Fergusson, Boden, Horwood, Miller, & Kennedy, 2012). Another large study of gene–environment interaction identified people who carried a genotype that conferred a low expression MAO-A (Caspi et al., 2002). The researchers looked at the people with high versus low MAO-A activity, and also whether or not the individual had been abused as a child. They found evidence of a strong interaction between low MAO-A activity and childhood maltreatment in the likelihood of developing CD.

However, the evidence suggests that the impact of genetic or biological factors diminishes as young people are exposed to environmental factors that shape behavior. For example, genetic research focusing on studies of identical twins (some reared together, others reared apart) has found that heritability accounts for about 41% of childhood conduct disorder, but by adulthood accounts for only 28% of adult APD. The effect of biological factors is therefore highly likely to be mediated by other situational or environmental conditions. Therefore, increasingly influential in the
coming years will be the science of epigenetics. The ability of the environment to switch genes on and off may be an important factor underpinning a number of risk factors for offending, which will be explored in Chapter 16 of this volume.

Conclusions and Structure of the Book

Genetic contributions, adverse developmental courses, early deprivation and other suboptimal rearing conditions, and substance abuse are often associated with severe problems in social and emotional functioning that potentially endure throughout life. It would be predicted that these early experiences would be reflected in long-term changes in the underlying neurobiology and the neurochemistry of the attachment/social brain systems as outlined in Box 1.3. The ensuing atypical morphological organization could result in social withdrawal, pathological shyness, explosive and inappropriate emotionality, and an inability to form normal emotional attachments (Joseph, 2003) and this sets the scene for later criminality. This broad thesis will be developed in subsequent chapters of the book, as outlined below.

The first volume contains three parts. In Part I we have begun by providing a background to the area in the current chapter. Chapter 2 provides an introduction to the principles of neuroscience. The aim of Part II of the book is to provide a more complete understanding of the principles of general neuroscience: aggression, sex, risk taking and decision making, emotional regulation, empathy and morality, and deception. Hence, Part II provides the necessary platform to understand the following sections of the book. Part III then explores what we know about the neuroscience of psychopathy, APD, and other personality disorders, offenders with autism, violent offending, sexual offending, homicide, adolescence, and alcohol.

The second volume contains four parts. Part IV contains what is currently known about the neurobiology of risk factors for offending, such as genetics, prenatal, developmental trauma, substance abuse problems, and mental illness, and how to modify such risk factors. Part V discusses current thinking about how the risk factors outlined in Part IV can be tackled in treatment. This part provides a broad sweep of approaches to treatments, with the first chapter (Chapter 22) describing engaging with offenders based on what we now know about biology and neurobiology. The rest of Part V considers a number of different treatment approaches as follows: the use of brain scanning to inform treatment; therapy for acquired brain injury developmental treatment models for adolescents; applications of mindfulness and controlled breathing techniques to improve self-regulation; compassion focused therapy; eye movement desensitization reprocessing therapy (EMDR); the impact of physical exercise; and drug treatments (specifically for sexual offenders). Part VI considers implications of neglect and trauma in understanding offending, as well as the ethical, legal, clinical, social, cultural, and political implications of forensic neuroscience and implications for forensic mental health programs and policy. Part VII provides some conclusions in terms of explanations in forensic neuroscience and considerations for the practitioner.

Notes

1 The fatty white substance that surrounds the axon of some nerve cells.
2 Glial cells surround neurons and provide support for and insulation between them. Glial cells are the most abundant cell types in the central nervous system.
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4 For a discussion on this, see http://www.scientificamerican.com/article/a-new-phrenology/.
5 However, it is worth noting that he did not engage in such statistical comparisons with non-criminals.
6 Echoes can still be seen in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (American Psychiatric Association, 2013) and the World Health Organization’s International Classification of Diseases psychiatric classifications systems (see http://www.who.int/classifications/icd/en/).
7 Although it should be noted that prior to the menopause women have five times more testosterone than oestrogen (Herbert, 2015).

Recommended reading


References


Gall, F. G. (1855). *On the functions of the brain and each of its parts (6 volumes)*. Boston, MA: Boston, Marsh, Capen and Lynn.

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