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The Neuroscience of Acquisitive/Impulsive Offending

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Key points

- The focus of this chapter is on the neurocognitive issues that affect the development of the acquisitive offender, as a result of many very early risk factors.
- Here the authors take a developmental perspective towards the understanding of acquisitive offending, specifically noting the importance of impulsivity in this type of offending.
- The chapter also outlines the changes in the adolescent to adult brain, and the associated area of risk taking, in helping to understand acquisitive offending.
- To more fully understand these changes from a brain-based perspective the chapter outlines the neuroscience/neurochemistry of impulse control.
- It is also noted that Attention Deficit Hyperactivity Disorder, and related issues such as the impact of substance misuse, and traumatic brain injury in childhood and adolescence, are important in understanding the etiology of acquisitive offending.
- The chapter concludes with what an understanding of the neuroscience of acquisitive offending can give in terms of forensic practice.

Terminology Explained

A-not-B task is a test of object permanence in babies. The experimenter hides a toy under Box A several times and allows the baby to see it. They then openly move the toy and place it under Box B. Most babies under about 10–12 months still reach for Box A even though they saw the toy being put under Box B. This mistake is known as the A-not-B error.

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Appearance-reality task tests whether a child can understand the difference between appearance and reality. Typically, an object is shown that looks like one thing but is actually another; for example, a stone that is painted to look like an egg. The participant is asked what the object looks like and what it really is. A child who can distinguish between appearance and reality will answer each question correctly; but a child who cannot, will respond on both occasions that the object is either what it looks like or what it really is. They are not able to understand that an object can look like one thing but be another.

The basal ganglia are a set of interconnected nuclei in the brain that are strongly interconnected with the cerebral cortex as well as several other brain areas. The basal ganglia are important in a variety of functions but particularly related to smooth movement and goal-oriented behavior, and enacting habitual behaviors as well as learning new behaviors.

Dopamine is a neurotransmitter that is involved in reward-seeking behavior. Reward increases dopamine levels, and the same effect is caused by many addictive drugs.

Epigenetics is the study of changes to genes that occur in addition *(epi)* to genetic structure determined by birth *(genesis)*. Gene expression is affected by developmental and environmental factors even though these things do not change DNA itself.

False belief tasks are designed to assess the extent to which a person, usually a child, recognizes that others can have beliefs about the world that are different to their own. The extent to which a person can do this is an indicator of the presence of theory of mind (see below). The classic false belief task involves two characters, Sally and Ann. Sally places a chocolate in a basket and then leaves the room. Ann then moves the chocolate to a box. The participant is asked to say where Sally will look for the chocolate when she comes back. Children under the age of about four, who do not yet possess theory of mind, will say that Sally will look in the box, because that is where they know the chocolate is. Children with theory of mind will say that Sally will look in the basket, because they understand that Sally will have a false belief that is different to their own belief.

The flanker task (Eriksen & Eriksen, 1974) is a method designed to test the development of information processing and selective attention. A target stimulus, such as a letter, is flanked by either congruous or incongruous stimuli (e.g., other letters that have been identified in the instructions as correct or incorrect). It measures the ability to inhibit or suppress responses that are inappropriate in a particular context. The ability to fully inhibit inappropriate responses is thought to develop between the ages of five and 15.

The go/no-go task is another method for measuring the ability to inhibit an inappropriate response. The task involves presenting stimuli in a continuous stream and participants must decide to either make a response or withhold a response based on initial instructions (e.g., press *y* for all positive words and *n* for all negative words). Accuracy and reaction time are measured for each event. The ability to fully inhibit inappropriate responses is thought to develop between the ages of five and 15.

The hippocampus is a brain structure located in the medial temporal lobe of the brain. It is part of the limbic system and is particularly associated with memory and spatial navigation.

The Iowa gambling task (IGT) is an experimental task to assess decision making and how participants weigh up reward versus penalty. Participants are presented with four virtual decks of cards and told that they can win money from choosing certain cards but that others incur a penalty. Of the four decks, two lead to wins over time and two lead to losses over time. Gradually, most participants learn which deck of cards bring rewards, and get better at choosing them routinely. However, some participants continue to choose "bad" decks, showing an apparent desire for reward that overcomes a sensitivity to punishment.

Pre-eclampsia is a disorder that affects some women in pregnancy, usually after about 32 weeks but sometimes as early as 20 weeks. Two key signs are high blood pressure and a high concentration of protein in the urine. Because pre-eclampsia reduces the nutrition passed from mother to baby in the womb, some babies are born smaller than usual or may have to be delivered early, when they are not fully formed within the womb.

The rule–use paradigm (Luria, 1959) involves tasks that test for a disparity between understanding and applying instructions. At younger ages (e.g., below five years), children can understand the rule of what they are being asked to do in rule–use tasks (such as squeezing a ball when a green light shows but not a red one) but are unable to actually follow the instruction.

Serotonin (or 5-hydroxytryptamine, 5-HT) acts as a monoamine neurotransmitter in the brain. Its function in the brain includes the regulation of mood, appetite, and sleep. Serotonin also has some cognitive functions, including assisting with controlling impulses and delaying gratification. Modulation of serotonin at synapses is thought to be a major action of the classes of pharmacological antidepressants known as SSRIs (selective serotonin reuptake inhibitors).

The **Stroop test** asks participants to name the color in which words are printed. The Stroop effect refers to the difficulty that participants have in naming the color of a word when the word itself is a different color. For instance, it will take someone longer to identify that the word GREEN is printed in red than it would for a non-color word.

Theory of mind, also known as mentalization, refers to the ability which develops around the age of four, to understand that others have beliefs, desires, intentions, and perspectives that are different from one's own. This involves a recognition that others have their own mind, which is separate to yours, and that they may know things you don't know, or not know things that you do know.

Introduction

Acquisitive and property crime is defined in the UK as "the various ways that individuals, households or corporate bodies are deprived of their property by illegal means or where their property is damaged (or where there is intent to do so)" (Murphy &

Eder, 2010, p. 79). Currently, acquisitive crime constitutes over 80% of all recorded incidents of "victim-based crime"[1] (Office of National Statistics, 2013), and over half of offenders found guilty in the year ending March 2012 were acquisitive offenders. These figures exclude those convicted of drugs offences (a further 18%), where a significant proportion will have carried out acquisitive crimes to support their substance misuse habit (Ministry of Justice, 2012).

Therefore, it is reasonable to suggest under these circumstances that a large proportion of the offending population is made up of acquisitive offenders. Despite their number, these offenders are often less the focus of attention than those who engage in more extreme, serious, and violent behavior (see the other chapters in this part of the current volume). They are less likely to have diagnoses of severe mental illness or personality disorder (though about a quarter of male prisoners in the UK will suffer from depression and anxiety, UK Prison Reform Trust, 2013) and are very unlikely to receive any structured interventions or rehabilitation as these are reserved for (higher-risk) offenders serving longer sentences.

The majority of prisoners in the UK (68% in the year ending March 2012, Ministry of Justice, 2012) serve sentences of 12 months or less with the bulk falling into the "acquisitive" category (National Audit Office, 2010). This said, being arguably the most common type of offender, research shows that they are likely to have been subject to many if not the entire array of prenatal, perinatal, childhood, and adolescent risk factors that have been empirically associated with the onset of criminality (Farrington, Piquero, & Jennings, 2013; Fergusson & Horwood, 2001; Moffit & Caspi, 2001).

Alongside well-established psychosocial factors including those associated with impoverished familial profiles and practices, as well as neighborhood disadvantage, recent years have seen an increasing focus on prenatal and perinatal issues that can detrimentally affect neurocognitive development and function (see Chapter 19 in this volume; for detailed summaries see Beech, Nordstrom, & Raine, 2012; Liu, 2011). These include exposure to alcohol, nicotine, and other legal and illegal substances in utero, malnutrition before and after birth and birth complications such as pre-eclampsia, gestational diabetes and perinatal obstetric interventions (Liu, 2011). Importantly, recent emphasis has been put on the reciprocal nature of neurological and psychosocial development rather than seeing them as independent or competing explanations for healthy development.

Most of these issues are dealt with in detail in other chapters within this book and are mentioned here just in order to set a context. The focus of this chapter will be on the neurocognitive issues that affect the development of the acquisitive offender, as a result of many of these very early risk factors, namely impulsivity, Attention Deficit Hyperactivity Disorder (ADHD), and related issues such as the impact of substance misuse and traumatic brain injury in childhood and adolescence. We will now examine the concept of impulsivity.

Defining Impulsivity

Andrews (1995) noted that "for many writers, from Lombroso and Freud through the Yale school and up to Gottfredson and Hirschi (1990), the essence of criminality has to do with a lack of self-control" (p. 37). Few would argue that lack of self-control is not strongly related to or even synonymous with impulsivity. As the quote above suggests, the concept of impulsivity has had one of the longest and closest associations with

antisocial and offending behavior. Reducing impulsive decision making and behavior has been an aim of offender rehabilitation programs since they began in earnest in the 1980s (McGuire, 1995).

But what exactly is impulsivity? Impulsivity and its control appear to encompass a number of complex mechanisms involving the response to a stimulus (which could be internal such as a remembered image or external such as a physical threat) that simultaneously rouses emotion, thought, and memory. It involves various stages including alerting to a stimulus (which may not be a conscious process in itself), developing the ability to orient toward that stimulus and then attend to it as we move through infancy and beyond. As we develop, the impact of executive functioning on control becomes clearer – involving working memory, decision making, long-term memory, planning, and inhibitory control (Tarullo, Obradovic, & Gunnar, 2009)).

Infants are born with little ability to control their impulses at a neurobiological level, though there are early signs of being able to orient away from distressing stimuli in the early months of life (Harman, Rothbart, & Posner, 1997). The initial process is thought to be an automatic, emotional response, but as the development of attentional and executive functioning really takes off from around three years onwards, effortful voluntary control emerges as part of the repertoire (though the more primitive, automatic responses stay with us for life). When the ability to control these inbuilt impulses, that is, to think before acting (especially when immediate reward is involved), does not develop, the resulting lack of self-regulation is thought to be important in a number of negative outcomes in adolescence and adulthood including ADHD, borderline personality disorder (BPD), and bipolar disorder, as well as antisocial behavior and criminal activity (Dalley & Roiser, 2012). We will now examine the neuroscience of impulse control.

The Neuroscience of Impulse Control

Three areas of the brain consistently emerge in the literature as implicated in impulsive behavior and impulse control: (1) the amygdala (which is part of the emotion-oriented limbic system); (2) the prefrontal cortex (especially the ventromedial prefrontal cortex (vmPFC) and the orbitofrontal cortex OFC)); and (3) the anterior cingulate cortex (ACC).

The amygdala is probably the major brain area to consider in understanding impulsive responses. It is an almond shaped nucleus in the anterior temporal lobe and is central to what is called the "somatic marker hypothesis" (Bechara & Damasio, 2005; Damasio, 1994). According to this hypothesis visceral reactions in the brain and body to emotion related signals (somatic markers) from our environment are either innate (evidenced in early infancy) or highly learned (as we develop through childhood). These instant, emotional "gut" reactions, or impulsive responses to stimuli, function to "mark" potential choices as being advantageous or disadvantageous in terms of survival. The process stays with an individual throughout their life, and aids in the type of decision making in which there is a pressing need (often in an impoverished environment with little information) to weigh positive and negative outcomes that may not be predicted decisively through "cold" rationality alone.

In other words, it is an instantaneous reaction informed by both emotion and memory. These processes are sometimes referred to as a central part of the *impulsive system* (Gupta, Koscik, Bechara, & Tranel, 2011). However, as the brain develops through

childhood and adolescence, the need to override immediate response to "prepotent" stimuli (those made salient because of their association with immediate reward) becomes progressively more advantageous. The vmPFC plays an increasingly important role in integrating such information and is critical to a more reflective kind of decision making and effortful control – also known as the "reflective system" through which executive function develops. See Box 14.1 for more detail on the impulsive/reflective systems.

Box 14.1 The amygdala and decision making

Conventional research documents the amygdala as an emotional relay center. Strategically placed to evaluate stimuli of an emotional nature, contemporary research has also attributed the amygdala with a role in the decision-making process by signaling "somatic markers" of reward and punishment through the awakening of the autonomic nervous system (Baxter & Murray, 2002; Bechara & Damasio, 2005). Decision making is guided by two major systems: the amygdala and the vmPFC with the IGT being the tool of preference for the study of complex decision making (participants learn to choose "good" card decks rather than "bad" decks, Bechara, Damasio, Damasio, & Anderson, 1994). Lesion studies have shown that the amygdala belongs to the "impulsive" decision-making process, since damage to this structure hinders somatic response to immediate rewards and punishments making individuals unable to pair information with the value of novel stimuli. vmPFC lesion patients seem to have impaired physiological responses to rewards and punishments that have been acquired by past experiences. Whereas the (impulsive) amygdala codes for the value of present stimuli, the (reflective) vmPFC allows planning of future reward or avoidance by recalling information that has already proven its value. The somatic marker hypothesis provides a good explanation of the above mechanisms (Damasio, 1994).

The vmPFC is part of the brain that allows a more flexible pursuit of longer-term goals, which may be more advantageous in the long run for an individual (e.g., keeping out of prison) but are less immediately rewarding. The vmPFC is thought to link together two types of memory: (1) current instances in working memory that the individual is attending to and (2) knowledge-based, long-term (declarative) memories that are relevant to the working memory data in question. See Box 14.2 for a current understanding of these types of memory.

Box 14.2 Types of memory

Knowledge-based long-term memory is described as the long-term storage of acquired information (Hebb, 1949). This type of memory has been divided in two main components, implicit (procedural) and explicit (declarative)

memory. ***Implicit (procedural) memory*** influences behavior with no conscious awareness of the causal memory. It appears be related to the basal ganglia processes (Foerde, Knowlton, & Poldrack, 2006). ***Explicit memory*** provides conscious control over recall of past events, and is associated with the hippocampi.

Working memory is defined as the cache in which temporary information is held and processed, and is typically not en route to long-term information storage. Baddeley and Hitch (1974, 1994) firstly introduced this term as an alternative to the short-term memory concept. Working memory has been associated with the prefrontal cortex (Smith, Rapp, McKay, Roberts & Tuszynski, 2004).

Note: Memory related responses are not restricted only to the above-mentioned regions but they also exist in other regions of the brain such as the anterior and posterior temporal lobe, the amygdala, the vmPFC, as well as the cingulate cortex.

Simultaneously to memory recall, somatic emotional responses to different potential outcomes (from the amygdala) are re-invoked, in order to evaluate the decision being made and how the consequences might affect an individual (Bechara, 2005). Thus, adults with amygdala damage have impaired somatic/autonomic responses to reward and punishment, while those with vmPFC damage are unable to re-integrate factual memories with visceral responses to reward and punishment which impairs their decision making about future behavior (Gupta et al., 2011). An additional way that the amygdala, as part of the brain's limbic system, is important in offending behavior is through its central involvement in the recognition of aggressive and fearful responses in others and reactions to these. Amygdala damage results in impoverished recognition of fear and aggression (Adolphs et al., 1994) and had been shown to be dysfunctional with the same outcomes in those scoring high on psychopathy. Between the amygdala and the vmPFC, information is mediated and moderated firstly by the orbitofrontal cortex, which gives a simple approach/avoidance response, and then by the ACC, which acts as a performance monitor and decides whether messages should be passed up for higher processing in the vmPFC or can satisfactorily be handled in situ.

Research shows there is a dramatic increase in development of executive functioning from around three years of age, and this includes the ability to inhibit responses to stimuli that we have decided are not worth our attention (Rueda, Posner, & Rothbart, 2005), but which remain in working memory while we choose to select other stimuli; and later to resolve conflict between incompatible responses during demanding cognitive tasks (e.g., the Stroop test, Carlson & Moses, 2001). The ACC has also been found to be central to this process, particularly in the *detection* and monitoring of conflict in functional magnetic resonance imaging (fMRI) studies (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999), and is considered the main node of the executive functioning system. Different parts of it appear to be involved in cognitive and affective (emotional) control. In tasks involving complex emotional processing, two areas have been seen to be consecutively activated and deactivated suggesting the possibility of reciprocal effortful and emotional controls of attention (Bush, Luu, & Posner, 2000). See Box 14.3 for more detail on cognitive conflict studies.

Box 14.3 Cognitive and brain consequences of conflict

In conflict resolution tasks, participants are given two dichotomous cognitive cues. The cues are not always congruent and as a prerequisite for the experimental task participants have to select a subdominant object or a response over the presence of a conflicting dominant one (Botvinick, Braver, Barch, Carter, & Cohen, 2001). The Stroop task (MacLeod, 1991) and the Flanker task (Fan, McCandliss, Sommer, & Posner, 2002) provide excellent examples of laboratory designed conflict stimuli. The Stroop task involves language stimuli and the conflict between the word's name and its color (e.g., "blue" written in green ink). The Flanker task on the other hand entails non-language spatial conflict in which a shape depicts a general direction (e.g., →) and is flanked by congruent, incongruent, and neutral shapes (Fan et al., 2002). Researchers from Cornell University have studied the phenomenon of cognitive incongruence using fMRI observing regions that are common in both linguistic and spatial related conflict studies. What was observed was that, despite the expected fact that incongruent stimuli had longer reaction times than congruent ones, both models of conflict shared similar brain networks. The ACC and prefrontal cortex were common in both tasks; however, as researchers argue, these sites seem to be only monitoring conflict and not resolving it as unique activation sites were observed according to the nature of the task (Fan, Flombaum, McCandliss, Thomas, & Posner, 2003).

The Neurochemistry of Impulsivity

As well as the structure of the developing brain, neurochemistry is also vitally important and again differences can be seen in the functioning of impulsive individuals compared to their more controlled counterparts from this aspect (Dalley & Roiser, 2012). There is evidence that dopamine and serotonin are implicated in the control of impulses. Dopamine is a neurotransmitter manufactured in the nucleus accumbens in the midbrain and transmitted largely into the frontal cortices. One hypothesis is that those with higher impulsivity have fewer active dopamine receptors in their midbrain but when stimulated, these neurons are more likely to secrete large quantities of the neurotransmitter, which is then not reabsorbed efficiently (Buckholtz et al., 2010). Serotonin (5H-T) is a neurotransmitter originating mostly in the median and dorsal raphe nuclei in the brain stem. Seretonergic neurons reach up into the nucleus accumbens, the amygdala, and the prefrontal cortex and a depletion of serotonin in the brain is linked to a reduced ability to delay gratification. Studies have shown that serotonin depletion either through pharmacological manipulation or lesion increases response onset to stimuli, as assessed by stop signal reaction time tasks (Winstanley, Theobald, Dalley & Robbins, 2005). Dysfunctional interactions between the serotonin and dopamine systems in the prefrontal cortex are associated with impulsive aggression and depression in adults (Seo, Patrick & Kennealy, 2008).

Much of what we have learned about the links between dopamine and serotonin secretion and impulsivity comes from the study of stimulants on the brain. Stimulant

use increases the tendency to choose a small, immediate (impulsive) reward instead of a larger but delayed reward (known as delay discounting). Rats with a genetic tendency toward impulsiveness, because of a deficiency of dopamine receptors, are more likely than others to self-administer large amounts of cocaine by lever pressing (Dalley et al., 2007). Importantly, it has also been shown that repeatedly injecting any rat with cocaine gradually makes it more impulsive (Simon, Mendez, & Setlow, 2007 see Box 14.4), so an environmental effect of learned impulsivity through repeated behavior is possible as well as the possibility of having a genetic predisposition and passing this on to future generations.

Drug abuse *Impulsive behavior is suggested to be one underlying mechanism of acquisitive offenses. Impulsive traits are common also in drug abusers (Kirby & Petry, 2004). This may explain the high prevalence of acquisitive offenses among drug users.*

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Box 14.4 Cocaine exposure causes long-term increases in impulsive choice

The delay-discounting task is a common method for assessing impulsive behavior. The task involves the choice of two rewards, an immediate one in which the profits are small and a delayed one in which returns are high (Kirby & Petry, 2004). To examine the effect of drug abuse on impulsive behavior male Long Evans rats received injections of cocaine or saline for 14 days. Following three weeks of withdrawal the rats received training on two levers with different

reward outcomes. After three months of treatment the rats given cocaine displayed increased impulsive behavior by choosing the immediate reward. They also exhibited less anticipation prior to the delivery of the reward (Simon, Mendez, & Setlow, 2007). These findings suggest a difficulty of the rat to associate time delay with the reward and the "winning" response even after long periods of drug abstinence. Observations on humans suggest that impulsive traits share a strong correlation with drug abuse, however, the direction of causation between the two variables remains unclear (Kirby & Petry, 2004). Impulsive behavior can have detrimental effects on an individual's life whether this is the short-lived happiness of drug use or the long-term consequence of a criminal offence. Impulsive traits have been associated with orbitofrontal lobe dysfunction (Rudebeck, Walton, Smyth, Bannerman, & Rushworth, 2006).

Recent work indicates a complex interaction between dopamine and 5H-T regarding how they contribute to impulsivity suggesting they should not be studied in isolation (Dalley & Roiser, 2012). The genetic/environmental impact of substance misuse on impulsivity is a cause for concern given the pre-existing increased likelihood of experimenting with substance misuse during adolescence. The young person from an impoverished background, who already may have a more impulsive nature (perhaps inherited structurally via parental substance misuse and exacerbated through chaotic learning experiences in childhood) will redouble this tendency through the recreational misuse of substances.

The Development/Non-Development of Impulse Control Processes

We have seen that an infant can register and orient towards an important stimulus (such as its caregiver) or orient away from a negative stimulus in the early months of life. But when does clear behavioral control begin to manifest itself? There is a great deal of evidence that the ability to delay gratification for greater rewards later, and altruism (reward for others as well) alongside more complex affective decision making (such as learning which deck of cards is more advantageous in the Iowa Gambling Task (IGT)) cannot be done by three-year olds but can by children aged four and five (Prencipe & Zelazo, 2005).

These processes involve the increasing role of the amygdala and orbitofrontal cortex (Crone & van der Molen, 2004) in both decision making and behavior. Several cognitive processes, which are inextricably linked to maturation of the brain begin around age four or five but continue to develop through childhood and adolescence through early adulthood. For example, working memory, which could be seen as the foundation of executive functioning (particularly in choosing to inhibit information deemed unimportant and selectively attending to other types), is evident from early childhood but really takes off from mid-childhood to the late teens, see Box 14.5 for a description of studies in this area.

Box 14.5 The prefrontal cortex during early development in humans

The capability to undertake tasks requiring behavioral inhibition and memory seems to begin between three and six years of age. Popular experimental paradigms that examine the above mechanisms include the A-not-B error (Zelazo, Frye & Rapus, 1996), the appearance-reality task (Flavell, 1993), the go/no-go task (Casey et al., 1997) and the theory of mind and false belief tasks (Fritz, 1991). The above paradigms share similar if not identical results, however, the mostly discussed paradigm is the theory of mind or false belief task. The child in this task is required to *indicate where the agent would think that the object is* as well as *conceal the item's true location*. An object of interest is placed at a pre-defined location while both the agent and the child observe. Then in the absence of the agent the object is relocated. Remarkably a child below three to four years old is unable to inhibit an inclination to say where the object really is or override a stronger impulse even though the child is aware of the fact that the given answer is "wrong" (Fritz, 1991). To successfully perform these tasks children not only need to recall instructions from their memory and acquire an understanding of another's behavior but most importantly *disengage* from a previously rewarded response (where the object was) and engage with a new one. This "error" has two elements to it: location and reward (Luciana, 2001). Cortical regions mediating this task involve the seeking system (Panksepp, 1998), made up by the inhibitory orbitofrontal cortex (Casey et al., 1997), the working memory dorsolateral prefrontal cortex (dlPFC) (Baddeley, 1992) as well as limbic and mesolimbic regions of appetitive responding (MacLean, 1990). Dopamine provides the neural gel that orchestrates successful interaction of the above regions (Luciana, 2001) and deficiencies in this task may not be related so much to the immaturity of the dlPFC but to inconsistent or insufficient signaling of dopamine during the tasks contextual change (Luciana, 2001).

Increases in the ability and functioning of working memory are strongly associated with increases in white matter in the prefrontal cortex (see Box 14.6), especially the dlPFC (Nagy, Westerberg, and Klingberg, 2004).

Box 14.6 Maturation of white matter and its association with the development of cognitive functions

Organized into tracts the white matter of the brain consists in its majority of axons insulated by myelin sheaths. Produced by glia cells myelin is a substance made by fats and protein aiming to accelerate the communication in many vertebrate axons, and unlike other cortical maturation processes, it forms consistently for decades (Benes, Turtle, Khan, & Farol, 1994). Maps of white matter in the

human brain are performed in vivo by an MR method called diffusion tensor imaging (DTI). This technique harnesses the anisotropic diffusion of water deriving from local tissue boundaries (Moseley et al., 1990) and anatomical elements of white matter such as axonal thickness and myelination are quantitatively measured by *fractional anisotropy*. Nagy et al. (2004) investigated a sample of participants aged 8–18 years, white matter maturation and its cognitive impact on working memory performance and reading ability. Findings showed a positive correlation between working memory and fractional anisotropy on two regions situated in the left frontal lobe as well as on a region that stretched between the superior frontal and the parietal cortex. Reading ability showed an increase in myelination and axonal thickness only on the left temporal lobe. The restricted maturation of the white matter on particular brain regions and the improvement of specific cognitive abilities are important parts of child development and brain maturation. Finally, it would be important to stress the fact that structurally related cortical changes develop also according to experience and cognitive practice (Scholz, Klein, Behrens, Johansen-Berg, 2009).

Another crucial element of developing executive control is the ability to detect when something has not turned out the way expected (error detection). This involves an increasing role for the ACC. Although younger children (four to five years old) often know they have made a mistake, they do not appear to have the ability to correct it. It is only in mid-childhood and into adolescence that this function appears (Santesso, Segalowitz, & Schmidt, 2006). As the ACC increasingly monitors activity and decides higher decision making is needed, a reprocessing of rules takes place, associated with both the vmPFC and dlPFC. With this comes a greater capacity to reflect and generate increasingly complex rules (reflecting Luria's rule–use paradigm). Associated with this, another central element to impulse control is the ability to move away from one task to another (more advantageous) one (known as task switching). This ability again begins around the age of five (e.g., being able to switch from one set of rules to another to sort information). The time taken to switch tasks decreases from childhood into young adulthood and stays fairly constant until about 60 years of age (Cepeda, Kramer, & Gonzalez de Sather, 2001). It is clear from a variety of sources that from a neuroscientific (structural) point of view we are not fully developmentally equipped to self-regulate until early adulthood. It is interesting that this perspective fits with the well-established age–crime curve indicating an increase in offending in the early teens and the drop-off in criminal behavior in the early 20s (Farrington, 1986) (see Chapter 21).

All of the developmental processes described above assume reasonable environmental conditions for the normal development of the brain. These might include: sufficient nutrition, rest, and a stimulating and loving environment; ample opportunity to observe prosocial behavior and consistent rewards for the incremental development of self-regulation and empathy for others; a childhood free from physical violence and access to toxic substances. These assumptions cannot be made in relation to the homes and environments of the children likely to become "typical" acquisitive offenders. Lack of exposure to the modeling of effortful control and a lack of opportunity to practice

and be rewarded for delaying gratification and desire are not optimal conditions for the corresponding cognitive, affective, and neurological developments for the development of agency and self-control to take place. As noted several decades ago in the groundbreaking work of Ross and Fabiano (1985), the chaotic environment that many young offenders grow up in naturally fosters a "survival" oriented approach to life that leaves little room for the development of self-regulation and social perspective-taking. Much further work is needed to understand the complex interaction between brain, cognitive and affective function, and behavior, especially under the atypical conditions that deprived children experience, and from a developmental perspective.

Changes in Adolescence that Might Explain the Increase in Offending Behavior

Chapter 21 provides a more detailed account of the neuroscience of adolescence but it is worth making a few points here. As the child progresses into adolescence (the stage at which most offending begins), massive changes in the brain occur in tandem with the desire for increased autonomy and a reduced desire for reliance on parents – in other words, the building of an autonomous self-identity. A natural increase in risk taking (see Chapter 7 for an in-depth discussion of the neuroscience of risk taking), novel sensation seeking and extra-familial social behavior is common in adolescents in a variety of mammalian species as well as humans (Spear, 2000) during this quest for autonomy.

Partially explained from a psychosocial point of view as the pushing of boundaries in order to explore appropriate "future selves" (Oyserman & Markus, 1990), there are strong neuroscientific correlates associated with these behaviors. Although often identified as negative and sometimes dangerous in contemporary society, these adolescent behaviors have positive benefits to the individual, including an increased sense of self-esteem, self-efficacy, and autonomy from parents as a result of skill-building, and praise and acceptance from peers (Spear, 2000). The finding that risk taking is much more common when with peers than alone points further to a social/evolutionary explanation for this behavior (Steinberg, 2007). Box 14.7 contains more information on risk taking in adolescence.

Box 14.7 Risk taking in adolescence

Contemporary neuroscientific research on the unusually risky behaviors undertaken in adolescence takes into account two models: logical reasoning and psychosocial factors (Steinberg, 2007). These models are governed by two brain systems the socioemotional, governed by limbic and paralimbic structures and the cognitive-control network. The risky behavior of adolescence might be explained by the fact that social, emotional and reward related networks are more strongly interconnected. The varying sensitivity of these networks is based on reward magnitude (Nelson, Leibenluft, McClure, & Pine, 2005). In the presence of peers, the socioemotional network is strongly activated and overrides the pre-mature cognitive-control network and its regulatory effects, something that

does not seem to take place when individuals are alone (Chambers, Taylor, & Potenza, 2003). It appears that the presence of peers in a laboratory driving risk-task, more than doubles the chances of adolescence exposing themselves to danger (Gardner & Steinberg, 2005). In addition, neuroimaging studies have suggested that although activation of the socioemotional network is involved in relatively risky decisions, these decisions can also be potentially highly rewarding (Ernst et al., 2005). In contrast to the sensitive socioemotional network, evidence of prematurity in the cognitive network is clear as changes in structure (Casey, Tottenham, Liston, & Durston, 2005) and function (Luna et al., 2001) of the brain still occur for many years afterwards.

In terms of changes to the structure and function of the brain, research points to four major changes. First, the impulsive/reactive system involving the limbic system, amygdala, and orbitofrontal cortex appears to become increasingly sensitive and active in the years approaching puberty (Ernst et al., 2005) with greater attention focused on the rewards that certain behaviors will bring. Second, these changes, coupled with the suggestion that dopamine receptors may become less sensitive during adolescence, may explain the tendency towards novel sensation seeking – including experimentation with alcohol and drugs (Spear, 2000; Williams, 2012) – that is, greater effort/indulgence is needed to feel good.

Third, the more reflective systems in the prefrontal cortex areas of the brain involved in planning, thinking ahead, and self-regulation are developing more gradually over the course of adolescence and early adulthood (Steinberg, 2004; Lamm, Zelazo, & Lewis, 2006). Within this system, logical reasoning abilities reach adult levels typically by around age 16, whereas the more psychosocial capacities, such as the connections in the ACC and the vmPFC that improve impulse control, future orientation, or resistance to peer influence, are lagging behind and continue to develop into young adulthood, that is, young people are simply not equipped with the ability to fully self-regulate until their early 20s, especially when in social situations (Gruber & Yurgelun-Todd, 2006). Notably, these research findings come from samples of typically developing teenagers who are unlikely to have suffered the extra negative impact of various risk factors on cognitive and emotional development in childhood (see Chapter 21, and the effects of substance misuse below).

Finally, the "synaptic pruning" aspect of brain development emerges consistently as an important part of change in the developing brain during adolescence, influencing the number and quality of connections among neurons. This process includes myelination (which increases white matter conduction speed through the growth of the myelin sheath around each neuron), arborization (in which the number of branching connections between neurons increases), and pruning (in which neuronal connections that are not needed are destroyed). Grey matter volume increases to a peak in early adolescence and then decreases, resulting in an "inverted-U" pattern over the course of development (Gogtay et al., 2004).

This pattern of grey matter increase followed by a decrease may be attributable to arborization followed by subsequent pruning of unused synapses (Giedd, 2004).

The upshot is increasingly smaller areas of the brain taking on more sophisticated and discreet functions. Rueda et al. (2005) have noted far less brain exertion in adults when resisting temptation than in four-year-old children in which activity can be seen all over the frontal lobes. In adults, it was focused on a much smaller area of the midline of the frontal cortex. This research again involved typically developing participants and it would be interesting to replicate this work with a sample of persistent offenders and children from less than optimal backgrounds.

It is important to note at this point that a myriad of factors as indicated at the beginning of this chapter will be affecting the individual's ability to master impulse control and self-regulation, for example *ADHD*, *substance abusing parents*, and the experience of *traumatic brain injury (TBI)*, discussed below.

Sadly, it is clear that numerous genetic factors from conception onwards, coupled with the impact of an impoverished attachment and learning environment in which a disadvantaged child is likely to be functioning, are likely to have a negative outcome on the ability to control and resist impulsive behavior. Individual genetic influences and how these eventually play out in the environment (with the help of "epigenetic" mediation) will have a crucial influence on whether this child will become a persistent offender or not. A natural ability to regulate emotion, an ability to control impulsive behavior, to develop theory of mind and be empathetic, to be female, to be of average or higher intelligence, to not have addictive parents, to be free from TBIs in childhood and adolescence and to be born into a cohesive, caring culture may *each* be enough in their own right to protect a child from embarking wholeheartedly on an antisocial route. However, forensic neuroscientific research is relatively rare, and studies using "average" offenders are almost non-existent leaving large areas of enquiry that need to be addressed.

Attention deficit hyperactivity disorder (ADHD)

A disorder strongly associated with impulsivity that is dramatically over-represented in the offending population is ADHD. ADHD is a clinical syndrome defined in psychiatry by high levels of hyperactive, impulsive, and inattentive behaviors beginning in early childhood. The disorder is common in the general population with prevalence estimates in the UK of around 3–4% (Young et al., 2011) and persists into adulthood about 50% of the time. It is highly heritable, though no specific candidate genes have as yet been identified (Williams, Giray, Mewse, Tonks, & Burgess, 2010), may be sometimes caused by TBI in childhood (Max et al., 2005), and up to two-thirds of young offenders and half of the adult prison population screen positively for the disorder in childhood (Young et al., 2011). Further, adults with ADHD account for eight times more aggressive incidents than other prisoners and six times more than those with antisocial personality disorder (APD) (Young et al., 2011). Young et al. have also shown that ADHD was the strongest predictor of violent offending in adult male prisoners, even above substance misuse (Young, Wells, & Gudjonsson, 2010). One hypothesis held by many in the field of ADHD is that the disorder renders children more vulnerable to all of the other risk factors associated with an impoverished environment resulting in a wide range of comorbid treatment needs (many of which are also criminogenic) such as educational and consequent occupational dysfunction, substance misuse, mental illness, and personality disorder (National Institute for Health and Clinical Excellence, 2009).

Neuroscientific explanations of ADHD are emerging and appear to involve many of the same mechanisms as those involved in general impulse control above. However, as well as deficits in executive functioning (particularly in poor inhibitory control), recent theories have placed increasing emphasis on altered reinforcement sensitivity as etiological in the disorder. This is associated with the ventral striatum part of the forebrain, underneath the cortex. Research has shown reduced ventral striatal activation in adolescents with ADHD during reward anticipation, relative to healthy controls. Ventral striatal activation was also negatively correlated with parent-rated hyperactive/impulsive symptoms (Scheres, Milham, Knutson, & Castellanos, 2007).

Luman, Tripp, and Scheres (2010) indicate that the positive reward of reinforcement is larger in those with the diagnosis and fosters a strong preference for options that are immediately rewarding but relatively unfavorable in the long term, even if the short-term reward is smaller. Further, midbrain dopamine dysfunction is also accentuated in sufferers. A lower firing rate in the dopamine neurons in the mesolimbic reward circuits of the brain suggests that reinforcement loses its value when the delay between the desired behavior and the reinforcement increases, making effortful control much harder and resulting in impulsivity (dynamic developmental theory of ADHD, Sagvolden, Johanson, Aase, & Russell, 2005). Interestingly, from a risk factor point of view, children with fetal alcohol spectrum disorders and children exposed to stimulants in utero are at very high risk of developing ADHD (Fryer, McGee, Matt, Riley, & Mattson, 2007; Langlois & Mayes, 2008). This may also account for the increased comorbidity with addiction problems.

Reward versus penalty *Acquisitive offense can be conceptualized as prioritizing "easy" gains over the risk of punishment. It is suggested that abnormal responses of the ventro-medial prefrontal cortex can bias behaviors and decision toward immediate profits over punishment.*

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Research is in its early days in this field however, and new findings are highlighting the complexity of altered reinforcement sensitivity in ADHD (Luman et al., 2010).

Given the impact on offending behavior, developments in this area are particularly welcome. At present pharmacological treatments are the prominent treatment option, in cases where the syndrome has been diagnosed. The UK Adult ADHD Network has been working hard in recent years to put awareness, treatment, and assessment of offenders with ADHD at the heart of the criminal justice system, but their work is in its infancy. Better still would be prevention and early intervention before criminality begins.

The increased likelihood of experimentation with alcohol and substance misuse during the period of adolescent brain development has been noted above and two factors related to this are likely to add to the criminogenic profile of the average offender. First, from an etiological perspective, cohort studies of the development of criminality have indicated that parental substance misuse is a considerable risk factor. Second, from an outcome point of view, having carers that misuse substances and alcohol, and misusing oneself during childhood and adolescence, increases the likelihood of TBI. We will look at the neuroscientific aspects of each of these factors.

Substance misusing parents

Alongside the increased sensation-seeking and risk-taking behavior that is characteristic of the development of self in adolescence, we have seen above how the dopamine reward circuitry in the brain is affected by substance misuse in two ways. First, the dopamine neurons become less sensitive to the effects of substances and second, these neural adaptations may be passed on to future generations. When the fetus is exposed to alcohol and drugs in utero, structural abnormalities in the developing orbitofrontal cortex, the prefrontal cortex and the ACC put the child at much greater risk of poor impulse control, greater emotional reactivity, and difficulty sustaining attention (Langlois & Mayes, 2008; Fryer, McGee, C.,. Matt, G., Riley, E., & Mattson, 2008). These factors, among the many other difficulties they bring, increase the child's own likelihood of substance misuse in childhood and adolescence.

Traumatic brain injury (TBI)

TBI (see Chapter 24 for more coverage on this) occurs when an external force traumatically injures the brain, for instance, as a result of sports injuries, a fall, a fight, or an accident with a vehicle. It is usually associated with loss of consciousness. Typical side-effects after one mild TBI include headache, fatigue, anxiety, emotional lability, and cognitive problems such as impaired memory, attention, and concentration (Hall et al., 2005). Those with ADHD, those with abusive or addictive parents, and those intoxicated with alcohol or substances themselves are at a much greater risk of TBI than the general population. Not surprisingly then, up to 60% of young people in custody have been subject to TBI, (as opposed to 9% of the general population, Williams, 2012).

Moderate (more than 30 minutes loss of consciousness) to severe TBI (more than six hours loss of consciousness) is typically associated with neuropsychological (executive function), behavioral, and social problems as a result (Williams, Cordan, Mewse, Tonks & Burgess, 2010). Several studies have noted a correlation between offending behavior and increased experience of TBIs (Hux, Bond, Skinner, Belau, & Sanger, 1998) and more recent work has suggested an etiological contribution of TBIs to

later offending behavior (Timonen et al., 2002; Williams et al., 2010). Timonen et al. (2002), controlling for a variety of confounding variables, found that TBIs in childhood and adolescence were significantly, positively correlated with mentally disordered offending in adulthood, in a general population cohort of over 10,000 in Finland. Criminality began earlier in those who suffered TBIs before the age of 12. Williams, Potter, and Ryland (2010) noted that even those with mild TBIs (less than ten minutes loss of consciousness – often referred to as concussion) if cumulative (e.g., as a result of incidents with inebriated parents, general physical abuse, or one's own intoxication) could lead to attention and memory problems. Impulsivity and lack of affective empathy (two common characteristics of the typical offender) are also strongly associated with adults with TBI in childhood (Tonks et al., 2009).

Effects of TBI on the brain

A straight impact to the front or the back of the head causes linear acceleration of the brain and is relatively well tolerated, but lateral or up-cutting blows cause rotational acceleration causing much more damage (Blennow, Hardy, & Zetterberg, 2012). Despite the protection of cerebrospinal fluid all around the brain, head injury (even without fracture) can damage fragile brain tissue as it accelerates and decelerates by the tearing of the long axons that interconnect brain regions and upsetting the neurochemistry (known as diffuse axonal injury or DAI). Repeated blows to the head are especially detrimental as the cerebral physiology is disturbed even after mild trauma making it vulnerable to further injury. Moderate and severe TBIs can also result in "focal injuries" including contusion (bruising of the brain as it hits the skull) and intracranial bleeding, which can result in death (McAllister, 2011). Importantly, damage occurs immediately, but continues for an extended period depending how serious the blow to the brain was (e.g., axons continue to degenerate and swell).

In terms of the structures and consequent functions of the brain that are most at risk of damage in TBIs, it is noteworthy that these correspond directly with the regions reviewed above dealing with attention, memory, executive function, emotion regulation, and effortful control of behavior (McAllister, 2011): first, a circuit in the dlPFC (modulating working memory, decision making, problem solving, and mental flexibility); second, in the OFPFC (playing a critical role in the capacity to self-monitor and self-correct in social context, reducing interpersonal impulsivity); and third, a circuit starting in the ACC (modulating reward-related behaviors). Gerring et al. (1998) noted premorbid diagnoses of ADHD in 20% of one sample with severe TBIs, while Max et al. (2005) and saw an *onset* of ADHD symptoms in 15–20%. Those who develop ADHD post TBI are most likely to have damage to the thalamus, basal ganglia, or the orbitofrontal gyrus and are more likely to come from backgrounds of LES and psychosocial adversity (Max et al., 2005).

Conclusions

Acquisitive offenders are relatively common offenders and are likely to make up the bulk of the sentenced population at any one time. There is overwhelming evidence from cohort studies that these offenders are subject to a wide range of risk factors from conception onwards that will affect their neurological, cognitive, behavioral, and

emotional development. We have focused in this chapter on the neuroscientific correlates of the early "impulsive system" in infancy, mostly governed by the somatic marker response of the amygdala and the subsequent development from about three years onwards of the "reflective system". The increasing mediating role of the ACC and the involvement of the prefrontal cortex in executive function, results in the ability to control impulsive responses. The successful development of these processes has predominantly been demonstrated in typically developing samples of children and adolescents.

We have described how the chaotic and impoverished background of most offenders is unlikely to offer the neurological prerequisites or to facilitate functional development. It will instead foster the development of an individual who is impulsive, who is focused on immediate reward, and who is unlikely to reflect on the consequences of their actions either for themselves or those around them. Alongside this heightened impulsivity, there is an increased likelihood that such young people will develop ADHD, will have addiction problems, and will suffer from TBI. Looked at in the round, the odds are stacked against individuals from these backgrounds for developing secure emotional attachments and functioning adequately in education, employment, and in their interpersonal relationships. This has been borne out empirically, in studies of the characteristics of young and adult offenders, over and over again. Once offending, they are ill-equipped in comparison to other young people to find alternative lifestyles and to desist from offending. They are also likely to be serving short sentences, which currently precludes the possibility of any significant attempts at intervention or rehabilitation in standard cognitive-behavioral treatment approaches.

Implications for Forensic Practice

A pressing dilemma emerges from the evidence reviewed in this chapter in relation to acquisitive offenders. While often considered "run-of-the-mill" offenders with (relatively) low risks and needs, the evidence is quite the opposite. Most offenders serving three months, or less, are offered no offender treatment/intervention to deal with their problems (National Audit Office, 2010), even though their rates of recidivism are very high – 58% (Prison Reform Trust (2013).

As testament to the level of problems encountered by this group on release, and as described above, homelessness, unemployment, substance abuse, mental health, and other problems affect short-sentenced offenders more than other prisoners (National Audit Office, 2010) and the cost of incarceration alone is around £300m per year (National Audit Office UK, 2010). The lack of recognition of the factors reviewed above (and in other chapters in this book) and their contribution to offending behavior is disheartening at the very least (Hughes, Williams, Chitsabesan, Davies, & Mounce, 2012) and calls for increased screening and identification of problems and intervention at an early age have increased over recent years (Bradley, 2009; *The Lancet*, 2009; Sainsbury Centre for Mental Health, 2009; Williams, 2012).

Forensic practitioners need to be keenly aware of the neurocognitive dysfunctions underlying many of the entrenched behaviors we see in young and adult offenders and need to be equipped to assess and identify particular anomalies in order to make an informed choice about intervention and support. Some recent moves have been made in this direction and are welcomed, such as the introduction of the

Comprehensive Health Assessment Tool (CHAT) (Offender Health Research Network, 2013), as a result of recent Department of Health strategy for young people in contact with the youth justice system (Department of Health, 2009). It contains a first night reception screen and subsequent measures to assess for risks in physical health, mental health, substance misuse and safety risks, learning disability, autistic spectrum disorders, speech, language and communication needs, and assessment for brain injury. There is a long way to go, however, in terms of comprehensive and effective use of such instruments.

Prevention is better than cure and many of the problems described above could be prevented with better education and socioeconomic support. In the unlikely event of this happening, however, there is evidence that the reciprocally determined (negative) outcomes of brain and environment on development and behavior can be modified, ideally in the early years when brain plasticity is at its greatest (Rueda et al., 2005), though this again assumes early identification of the problem. Tarullo et al. (2009) and Rueda et al. (2005) summarize numerous studies in which the foundations of executive function, emotion regulation, and ultimately impulse control can be modified and improved upon in pre-school and pre-teenage children. However, much more work on the neuroscience of offending behavior is needed, especially using more typical offender populations (most existing work using offender populations looks at more extreme, violent groups). Work on TBI (McAllister, 2011) and homeless children (Obradovic, 2010) has noted increased neurological resilience in some individuals against adversity. Understanding resilience more clearly from a neuroscientific point of view should be a priority and could unlock the differences between children who survive adversity and those who are less fortunate.

Note

1 Including burglary, all forms of theft, fraud, and criminal damage.

Recommended Reading

Liu, J. (2011). Early health risk factors for violence: conceptualization, review of the evidence, and implications. *Aggression and Violent Behavior*, *16*, 63–73. doi:10.1016/j.avb.2010.12.003. *A really thorough overview of the many and varied risk factors involved in the development of criminality.*

Luciana, M., & Nelson, C. (2008). (Eds.), *Handbook of developmental cognitive neuroscience.* Cambridge, MA: MIT press. *For those wishing to truly immerse themselves in the scientific research and development of theory surrounding the way cognitive capabilities such as language, decision making, memory, and visual perception develop in children, from a neuroscientific point of view; this gives you an insight into what should happen in typically developing children, but it devotes a large section to childhood disorders, some of which gives us an insight into what might happen in children from offending backgrounds.*

Rueda, M., Posner, M., & Rothbart, M. (2005). The development of executive attention: contributions to the emergence of self-regulation. *Developmental Neuropsychology*, *28*, 573–594. *A detailed and readable review of research that has contributed to our understanding of the neuroscientific correlates of emerging self-control and regulation in children.*

Tarullo, A., Obradovic, J., & Gunnar, M. (2009). Self-control and the developing brain. *ZERO TO THREE*, *29*, 31–37. *An excellent description of the neurobiological correlates of self-control in the developing child, written with practitioners in mind.*

Williams, H. (2012). *Repairing shattered lives: Brain injury and its implications for criminal justice*. London: Barrow Cadbury Trust. *Provides a succinct account and in an accessible way of the effects of brain injury, written for criminal justice practitioners, also has excellent diagrams of the brain.*

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