

Psychobiology and Crime: ADHD

Nadia Wager examines the link between Attention Deficit Hyperactivity Disorder and criminality.

The actions and intentions of the eugenics movement in the latter part of the nineteenth century have left many social scientists with a sense of foreboding when contemplating the notion of biology playing an aetiological role in criminality. Whilst it is clear there cannot be a single criminal gene, since the definition of criminal acts differ between historical epochs and cross-culturally, there are some universal norms concerning acceptable and unacceptable behaviour. For example, the murder of one's kinsman tends to be universally proscribed. In contrast to traditional biological theories, more modern perspectives on criminality are less likely to purport to hold the answer to the grand question of 'what causes criminality?'.

Rather they are concerned with answering questions such as, 'what biological factors contribute to some people becoming persistent offenders?'.

Moffitt's 'Developmental Taxonomy of Criminals' (1993, in Piquero and Moffitt, 2004) helps explain the relatively small group of offenders whose behaviour is amenable to a biological explanation of causation. She categorises the general population in terms of their propensity towards criminal offending, distinguishing between three distinct groups of people.

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The first group consists of a relatively small number of 'abstainers' who refrain from all forms of criminal activity throughout their lives. The second group is the 'adolescent-limited' offenders whose delinquent/criminal behaviour is restricted to the time period between the onset of puberty and the attainment of adult social roles. More socially inclined explanations, such as the maturity gap and peer associations, are best befitting this group of offenders. Finally the third group are the 'life-course persistent' offenders. Again these represent only a small proportion of the total population, between five and eight percent; however, they are the most prolific in their offending.

Unlike the previous typology these individuals are characterised as generally operating as lone offenders and are more likely to engage in violent crime. Importantly, there are a number of individual differences evident quite early in the childhood of life-course persistent offenders that are predictive of later offending, which are not evident for adolescent-limited offenders. These include cognitive deficits, hyperactivity and a difficult temperament.

Moffitt argues that this group's elevated risk of criminality originates from their inherited or acquired neuropsychological constitution. However, Moffitt does not promote pure biological determinism; rather she contends that environmental conditions have the potential to either exacerbate or ameliorate the risk conferred by the individual's biological constitution. Environmental factors associated with exacerbating the negative

effects of an impoverished constitution include; neglectful, harsh or inconsistent parenting, disrupted family bonds, lone-parenting, maternal psychiatric history, poverty, low birth weight and poor relationships with peers and teachers.

Both the cognitive deficits and the environmental risk factors highlighted by Moffitt as associated with life-course persistent offenders are strongly reflected in the temperament and life experience of children suffering from Attention Deficit with Hyperactivity Disorder (ADHD). This neuropsychological disorder has been linked with antisocial behaviour, juvenile delinquency and adulthood criminality (see Pratt *et al*, 2002). One source of evidence for this association comes from an examination of the rate of ADHD in the offending population. Estimates of the American prison population suggest that between fifty and eighty percent of inmates have ADHD. Pratt *et al*'s meta-analysis of studies examining the relationship between ADHD and delinquency/criminality demonstrates that overall, the size of the effect of ADHD on later criminality is substantial, and thus warrants serious consideration and intervention.

The Diagnostic and Statistical Manual of Mental Disorders (DSM) distinguishes between three distinct subtypes of Attention

Deficit and Hyperactivity Disorder: that which is characterised predominately by inattention, the hyperactive-impulsive type and the combination of inattention, hyperactivity and impulsivity. The combined subtype is the most common manifestation of the disorder and the hyperactive-impulsive type occurs least frequently. According to the assumptions inherent in Moffitt's taxonomy, it is the latter two sub-types that would confer the greatest predisposition for criminality. Interestingly, gender differences in the prevalence rates for males and females indirectly support this. Whereas, the overall prevalence rate is between one and five percent of the population, males are five times more likely to receive a diagnosis of ADHD. However, more recent research suggests that this might be an artefact of the differences in the way the disorder typically manifests in boys and girls. It has been suggested that females may be under-diagnosed due to the presentation of atypical symptoms. In particular, females have been found to exhibit the inattentive sub-type twice as often as males and are far less likely to display hyperactive-impulsive subtype symptoms, which appear to be more closely associated with criminality. Thus, gender differences in the prevalence and presentation of ADHD appear to reflect the male dominated trend in offending behaviour.

With regard to the mechanisms through which ADHD potentiates risk for criminality, a number of possibilities have been identified. For example, Pratt *et al* (2002) contend that the offending mechanism is the debilitating effect of ADHD upon self-control. Whilst there are a number of criminologists (e.g.



Gottfredson and Hirschi) who have alluded to the criminogenic properties of low self-control, most have attributed the origins of this risk factor to ineffective parenting. That is, parents failing to monitor and dissuade against antisocial or deviant behaviour exhibited by their offspring. However, earlier research conducted by Pratt and his colleagues demonstrates that the link between delinquency and ADHD persists even after controlling for parental management. Thus, indicating that low self-control might not arise merely as a consequence of unfortunate social experiences, rather the origins might be biological or genetic. This is not to say that parenting factors have a benign effect, rather they could have an exacerbating effect upon children who already possess a biological predisposition.

The biological or neurological correlates of ADHD are numerous and include premature birth, low birth-weight, prenatal exposure to toxins such as alcohol and nicotine, obstetric complications and head trauma etc. Causal explanations too are numerous. However, there is one in particular that suggests impairment in the capacity for self-control. This is known as the 'frontal lobe hypothesis'. The frontal lobe is analogous to a car's braking system; processing within this region permits the individual to assess the consequences of their behaviour before acting and prevents initiation of the action, if that intended is considered inappropriate. Several different research methodologies have lent support to this hypothesis. For example, electroencephalographic (EEG) research measuring the electrical activity of the brain's cortex, PET scans assessing cerebral blood flow and functional magnetic resonance imaging techniques have all highlighted compromised functioning and diminished size of the frontal cortex in individuals with ADHD compared

to 'normal' controls. Whilst many ADHD sufferers are prescribed stimulant medication (e.g. Ritalin) to treat the disorder, which might confound some of the findings, in many cases the ADHD participants selected for the studies were non-medicated. Furthermore, cognitive performance tasks designed to assess frontal lobe functioning have demonstrated that non-medicated adolescent males exhibit marked deficits in relation to their non-ADHD contemporaries (Aman *et al* 1998). It has been proposed that the diminished functioning of the frontal lobes is mediated by a problem with the transmission of the electrical impulses to and from this region of the brain, which is attributed to the dysfunction of neurotransmitters, or chemical messengers, such as dopamine, serotonin and nor-adrenaline.

Whilst this evidence may appear to be very convincing, it must be remembered that a causal relationship cannot be established in such cross-sectional studies. Whilst it could be assumed that cerebral abnormalities/dysfunction is a precursor to, and a causal factor in, the development of ADHD, it is equally likely that the behavioural traits of individuals exhibiting ADHD-type behaviour compromise neuronal development, which results in the observed aberrations in the brains of ADHD sufferers. The latter argument is considered somewhat plausible in light of evidence of brain plasticity in young children. Plasticity implies that should a functionally specific brain region sustain damage through injury or disease, other unaffected areas will compensate for the lost functions. This has been demonstrated in studies of children who have undergone hemispherectomy (the removal of one whole side of their brain), yet who have retained all normal cognitive and motor abilities.

Additionally, it has been indicated that there is a strong

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hereditary basis to ADHD. Family studies indicate a concordance rate for ADHD between parents and children of about 70%. Furthermore, twin studies suggest a concordance rate of 92% between identical twins. Whilst there is a plethora of criticisms of both of these types of study (see Joseph, 2000), a possible gene has been identified which modulates dopamine activity in the frontal lobe, known as D4 receptor gene (Levy *et al.*, 1997) which substantiates the notion of a genetic basis.

In conclusion, whilst a biological approach to understanding crime is not applicable to the majority of adolescent perpetrated crime, its potential for understanding life-course persistent crime, which is often more serious in nature, is noteworthy. Whilst controversy reigns over the medicalisation of, and pharmacological intervention with, disorders such as ADHD, the biological approach may ultimately shed light on preventative measures that are not as abhorrent as those proposed by the eugenicists.

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