The adolescent brain: implications for understanding young offenders

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The following article discusses the many complex factors that influence the development of the human brain, highlighting specific aspects during adolescence, how risks to brain development and function affect the behaviour and maturity of adolescents, and implications for juveniles and those who come into contact with them in the criminal justice system.

**The criminal legal setting**
A guiding principle in the Children (Criminal Proceedings) Act 1987 is that “children who commit offences bear responsibility for their actions, but, because of their state of dependency and immaturity, require guidance and assistance”.1 A range of developmental experiences, discussed below, may have a negative impact on brain development that may result in wide variations in maturity and behaviour in adolescents of the same age and consequently on their degree of criminal responsibility.

**Normal brain development during adolescence**
Adolescence, the period between childhood and adulthood (12 to 21 years) is a time of rapid growth and development across neurological, hormonal, physical, cognitive, and social-emotional domains. Functional magnetic resonance imaging (fMRI) of the brain’s functional architecture during different stages of the lifespan has identified early adolescence as a significant period for brain development.2 Integration and co-ordination between subcortical and cortical connections become more refined and efficient during adolescence, resulting in a reduction in irrelevant connections and strengthening of connections supporting goal-directed actions.3 Simultaneously, myelination of axons, strengthening and consolidation of highly used connections and pruning of unused synapses and receptors improve efficiency of brain processing. This development unfolds in an ecological system comprising family, school, peers, and the wider community.

Adolescence is a paradoxical developmental period characterised by increasing autonomy, strength and resilience, but also heightened expression of mental illness that often has its origins in early childhood adversity. Adolescents are simultaneously capable of rational behaviour and mature decision-making, and heightened emotional reactivity and reckless, impulsive behaviour, which underpin increases in preventable morbidity and mortality.

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1 Section 6(b).
4 Myelination is the process whereby a myelin (i.e., fatty white substance) sheath (an electrically insulating layer) forms around the axons of nerve cells to improve the conduction of nerve impulses.
mortality. This highlights both the biological vulnerability and neuroplasticity of this stage of development.6

Protective factors such as consistent maternal presence, warmth and support and adverse factors (see next section) alter the structural architecture of the brain in ways that enhance or adversely affect adolescent development.7 These environmental effects occur in the context of normative brain developmental processes, including the imbalance between rapidly developing limbic structures associated with affect and motivation and the slower developing prefrontal cortex responsible for cognitive control that makes adolescence a time of higher risk for difficulties in emotion regulation, risk-taking, psychopathology and the onset of addictive behaviours.8

Adolescents also process incentives differently from adults. Reward sensitivity is a neglected but important aspect of adolescent brain development that is highly susceptible to environmental contingencies. Prosocial behaviour results in pro-social rewards, while anti-social rewards that accrue to delinquency, substance abuse, gangs, and criminal behaviour result in further anti-social behaviour if prosocial rewards are not available.9 This heightened reward sensitivity, associated with increased dopamine levels that peak during adolescence in response to pubertal status (not chronological age), affect decision-making, novelty/ fun-seeking, risky behaviour (eg, substance use, reckless driving, unprotected sex) and decreased harm avoidance. Dopamine is critical because of its role in cognitive and affective behaviours and the pathogenesis of psychopathology during adolescence.10 There is wide variability in the degree of risk-taking during adolescence, which has a neurobiological basis in the variable impact of dopamine on incentive-driven behaviours.

Brain development and early trauma

The brain is vulnerable to prenatal, postnatal and early life stress. Exposure to early adverse childhood experiences (ACE) and stress triggers a neuro-inflammatory response that alters neuro-immune development which underlies some psychiatric illnesses.11 The recent work of the Royal Commission into Institutional Responses to Child Sexual Abuse has highlighted the impacts of child sexual abuse on enduring emotional distress and later psychiatric morbidity.12 The quality of parent-infant attachment exerts a profound influence on children’s developmental trajectories,13 which often have their full expression during adolescence.14 Adverse childhood experiences (ie, emotional, physical, or contact sexual abuse; household dysfunction; alcohol/substance abuse, mental illness, violent treatment of a mother or stepmother, criminal behaviour in the household, and parental separation or divorce) change the functioning of brain circuits in the amygdala which mediate fear and pleasure responses; the hypothalamic-pituitary-adrenal (HPA) axis that regulates stress; the prefrontal cortex that regulates mood and emotional and cognitive responses; and the hippocampus, a stress-sensitive brain structure involved in learning and memory15 responsible for emotion regulation. Even moderate forms of life stress, such as inter-parental conflict16 and maternal stress17 increase negative infant emotionality through changes caused by stress to functional brain networks. Stress simultaneously inhibits neurogenesis (ie, the capacity of the brain to continue to grow new neurons into adulthood) and increases stress hormone (ie, cortisol) production which in turn deregulates the HPA system. Stress-provoked interference with other chemical circuits in the brain (eg, epinephrine, norepinephrine and serotonin) results in mood dysregulation and deficits in social attachment.18 Abused children with Post-Traumatic Stress Disorder (PTSD) have increased circulating epinephrine and cortisol and increased resting heart rates compared with non-abused children.19 When abuse is severe and
prolonged, hippocampal development is impaired. Permanent dysfunction and/or reductions in the size of critical brain structures such as the hippocampus, amygdala, medial prefrontal cortex, and other limbic structures associated with emotional regulation20 and attachment have far reaching consequences for emotional (eg, depression, anxiety, panic, impaired attachments, suicidality), behavioural (eg, sexual dysfunction, early sex, promiscuity, substance abuse, aggression, hyperactivity, and eating disorders) and physical health in later life (eg, cardiovascular disease, hypertension, obesity, cancer, type 2 diabetes). The amygdala is particularly sensitive to ACE during pre-adolescence. ACE results in larger amygdala volume, which in turn is associated with heightened aggression.21 Abnormal functioning of the amygdala is also implicated in anxiety disorders, autism, depression, and PTSD. Thus, impaired brain development as a result of ACE mediates the association between ACE and later vulnerability to mental illness and emotional and behavioural dysregulation.22

ACE, particularly repeated maternal separations, are strongly implicated in violent and antisocial behaviours. The causal factors of child temperament (behavioural disinhibition, fearlessness, high negative affect), intergenerational transmission (genetic predisposition and neural and behavioural epigenetics23) and family socialisation history (insensitive, harsh, hostile, rejecting, over-controlling or neglectful parenting, and inter-parental conflict) have now been supplemented with findings that show that chronic severe stress, whatever its source, is associated with dysregulated neurobiological functioning. This is expressed in oppositional behaviours, conduct problems, self-control difficulties or ADHD that manifest as early as two to three years of age, thus setting up a potential trajectory for violent, antisocial and criminal behaviour during adolescence.24 It is noteworthy that the two major types of human aggression — reactive and instrumental — produce neuronal dysfunctions specific to type.25

### Exposure to toxic substances
Add to the above influences on brain development prenatal or postnatal exposure to and/or later self-administration of toxic substances. Prenatal exposure to nicotine increases the risk of ADHD and externalising problems in offspring.26 The adolescent brain is highly sensitive to nicotine; its use impairs neural connectivity and specific brain regions (prefrontal cortex and amygdala) which persist into adulthood, amplifying addiction-prone behaviour.27 Prenatal methamphetamine exposure is associated with deficits in attention, memory, and visuomotor integration.28 Prenatal alcohol exposure results in foetal alcohol spectrum disorders (FASD) that impair neurogenesis with devastating consequences for physical development, cognition and behaviour.29 Alcohol abuse during adolescence has specific effects on neurodevelopmental and neuroanatomical pathways including accelerated grey matter reduction in the cortical lateral, frontal and temporal lobes, which may be irreversible. These epigenetic changes in cortical and subcortical brain structures are associated with increased alcohol use, impulsivity, and externalising disorders.30 Adolescent marijuana use impairs executive function, complex attention, memory, processing speed, and visuospatial functioning, with early onset continuing users showing more impairment in cognitive function and structural damage to the cortical architecture.31

### The adolescent brain and implications for the criminal justice system
Key issues for young offenders in the criminal justice system are their understanding (comprehension of the purpose, nature and consequences of the legal proceedings), reasoning (capacity to converse with legal representatives and to process information) and appreciation (ability to assess their situation coherently and rationally). The juvenile justice system places considerable emphasis on rehabilitation.32 Rehabilitation

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22 Ibid.
23 Epigenetics is the study of changes in genes in response to external stimuli (ie, gene-environment interactions) and whether such changes modify behaviour, cognition, personality and mental health over the lifespan. Epigenetics studies how nurture shapes nature.
potential tends to be greater in younger offenders. The application of alternative justice processes may prove effective as a rehabilitative process in diverting less serious young offenders from a life of crime.  

A large number of studies show that the younger the offender, the greater their deficits in fitness-related capacities. Youth aged 15 and younger consistently perform worse than adults in legal settings. There is a decreasing linear association between age and fitness. However, age alone is not sufficient to determine criminal responsibility because of the different timings of maturation and development and the variability in cognitive functioning among young people of the same chronological age. Younger offenders with lower cognitive functioning, particularly in verbal intelligence, are most disadvantaged in the legal setting. Adults with intellectual disability are rarely considered fit to plead or stand trial. Young people may evince similar deficits because of immaturity even though they test above the critical cut-off for a diagnosis of intellectual disability (IQ<70). Young offenders have IQ scores with much higher prevalence in the ranges of low (31%), borderline (27%) and intellectually disabled (15%) compared with community samples. Low IQ scores that fail to meet criteria for intellectual disability in young people with brain damage due to foetal alcohol exposure, substance use, or mental illness are likely to place the young person’s actual function in the range of intellectual disability, rendering problematic any assessment of fitness to plead, stand trial or give evidence based on IQ alone. Indigenous status confers further risk of disadvantage in the criminal justice system. Adaptive functioning (ie, conceptual, practical and social skills) has not been shown to be reliably associated with a young person’s understanding, reasoning and appreciation of the nature of legal proceedings.

Child maltreatment is highly prevalent in the early histories of young offenders (60% report ACE), who demonstrate all of the adverse consequences to brain development and mental health outlined in the sections above. Psychological morbidity (ie, substance abuse, externalising disorders, depression, and PTSD, a diagnosis often missed among young offenders) is also highly prevalent but the extent to which mental illness impairs young offenders’ culpability and fitness (if fitness is raised as an issue) is complicated and contested, and depends on the nature of the mental illness (eg, psychosis, personality disorder, PTSD, depression) and other factors such as cognitive function and history of ACE.

The construct of psychosocial immaturity, while difficult to define, is becoming more intelligible in the context of increasingly detailed mapping of brain development during adolescence. Figure 1 summarises the psychosocial processes required for the achievement of maturity.

Figure 1. Model of proposed temporal and causal relationships resulting in psychosocial maturity (©DTKenny)

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36 The law conclusively presumes that a child under 10 cannot be guilty of an offence (s 5, Children’s Criminal Proceedings Act 1987); between 10 and 14 the prosecution must rebut the presumption of “doli incapax” and prove beyond reasonable doubt the offender understood that what they were doing was wrong. The law recognises the potential for the cognitive, emotional and/or psychological immaturity of a young person to contribute to their breach of the law. Allowance will be made for an offender’s youth and not just their biological age: R v Heane (2001) 124 A Crim R 451 at [25]; KT v R (2008) 182 A Crim R 571 at [23].


43 Ibid.

44 P Johnstone, above n 33.
This construct unites the roles of IQ (cognitive capacity), relationship and life history (attachment security and ACE), and the component skills needed in a legal setting (eg, time perspective, delay of gratification, goal setting, self-reflection, perspective-taking, emotion regulation) to assist in determining a young offender’s fitness to plead and/or capacity to comprehend court procedure. Failure of emotion regulation is implicated in both psychiatric morbidity and offending during adolescence. Environmental demands and biological/hormonal changes during adolescence amplify emotional reactivity at this stage of development because of the incomplete integration and co-ordination between subcortical (emotion) and prefrontal cortical (cognitive) connections.45

The “immature” brain of adolescents is consistent with observed behaviour in legal settings — eg, poor decision-making, preference for immediate gratification over future welfare, desire to please others or protect one’s friends rather than act in one’s own best interest — orientations that might prove highly detrimental to the young offender in legal proceedings. Emotional dysregulation and poor decision-making are strongly associated with psychopathy, which in turn is associated with structural abnormalities in the frontolimbic and temporal regions of the brain.46 Studies on young people with so-called callous-unemotional traits indicate a neurodevelopmental trajectory for psychopathy.47

Conclusion
Adolescence is a sensitive period for brain development during which the environment exerts strong effects on neurological development and behaviour. Factors such as “dependency and immaturity”, adverse childhood experiences, cognitive function, mental health, early and continuing exposure to toxic substances,48 as well as age, are relevant to determining questions of criminal responsibility,49 fitness to plead and to stand trial, bail decisions and the disposition of criminal proceedings, including diversion and penalties.

48 Where an offender is addicted to drugs at the time of the offence, the age at which he or she became addicted may be a relevant factor to take into account in mitigation at sentence: R v Todorovic [2008] NSWCCA 49 at [58]; SS v R [2009] NSWCCA 114; R v Henry at [273]; Judicial Commission of NSW, Sentencing Bench Book, at [10–485].
49 Mental health concerns or low IQ may be relevant to the question of whether the prosecution has rebutted the presumption of doli incapax: R v AN [2005] NSWCCA 239 at [22]–[32].