RISK TAKING AND THE ADOLESCENT BRAIN

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Introduction

" I think I am going to make this diary private - there are so many things you don't know - I've got girls, I nick things, I swear, I smash windows, I smoke – *oh yes* – I'm a right little tearaway!!"

Journalist Toby Young reads an excerpt from his own teenage diary on Radio 4 (Young, 2012) and though apparently rather proud of his antics in one respect….the adolescent Toby also clearly recognises that his anti-social behaviour is probably best kept just to himself and his peer group.

For around fifteen years, in both popular and scientific literature, there has been plentiful discussion on teenage anti-social behaviour: "Moody, impulsive, maddening" (Dobbs, 2015) ….even "violent", "brain damaged" or "like an alien" (Danessi, 2003; Bradley, 2002)) are frequent media descriptors for teenagers of the Western world. Whilst the idea commonly promoted by newspaper journalists is that the adolescent brain is somehow deficient or partially-functional, like a "race car with no brakes or steering wheel" (themompsych.com), scientists report in academic journals MRI evidence to suggest there may be a firm neural basis for many of the erratic and reckless behaviours seen in this age group (Giedd et al, 1999, 2004; Blumenthal et al, 2002; Steinberg, 2010). Critics of such studies argue the findings are based on a number of 'naïve epistemic assumptions' (Bessant and Watts, 2012; 181) and warn that their academic colleagues are too readily adopting a 'biological reductionist' approach (Males, 2009) and that biodeterminist claims (where behaviour is determined entirely by biological factors) are the 'most profound and potentially dangerous that scientists can make' (*ibid*; 5).

Legal and Policy Discourse

Despite such criticism, the available neuroscience on the adolescent brain is now slowly filtering into legal and policy discourse. Neuroscientific evidence was referred to by the US Supreme Court (Roper v Simmons, 2004) in its decision to abolish the juvenile death penalty, on the grounds that adolescents do not possess the maturity and sense of responsibility that adults do. In particular the fact that adolescents lack self-control and are more susceptible to negative sways and external pressures, most especially peer pressure (Steinberg et al, 2003) were significant factors cited in the summing up of the case.
(http://www.apa.org/about/offices/ogc/amicus/roper.aspxf)

In response to public concern around the issue of anti-social adolescent behaviour, the US and the UK governments have made several attempts to address the problem, primarily through providing educational programmes in schools (Steinberg, 2008; Chowdry et al, 2013). However, little consensus has been reached in the research regarding which interventions are most successful. In 2013, the Department of Education commissioned a report by the Centre for Understanding Behavioural Change (CUBeC) to investigate whether providing information to adolescents can reduce their risk taking behaviours. Amongst the study's key findings were that firstly, a variety of preventative approaches are currently made in the UK, but because of the wide variety of age bands and behaviours targeted, assessment of individual outcomes is challenging. Another key finding was that knowledge and perceptions of potential issues are changed through educational programmes but not behaviour; interactive interventions are more successful in this respect (Choudry et al, 2013).

Researchers on adolescence are calling for further discourse between policy makers and scientists and suggesting that more studies are required to provide further insight into developing more effective intervention programmes for schools (Steinberg, 2008; Blakemore, 2012).

In the following pages, some possibly related factors to risk taking and sensation seeking behaviours are explored, including psychosocial elements, cultural contexts and neurobiological events – all of which could be implicated to some extent.

But first, to define adolescence…..

Adolescence is the intermediary phase of life that lies between childhood and adulthood, a period in which individuals undergo extensive physical and psychological change that will shape their sense of identity as well as develop social awareness and self-consciousness (Sturman and Moghaddam, 2011). Puberty occurs within the adolescent phase but is perhaps most commonly associated with the maturation of the reproductive system and physical conformation (Laviola, 2011).

The point at which adolescence begins and ends is not clear, as it is for puberty. Adolescence can extend from around 12 years old and often will last into the person's twenties, encompassing not only the emotional, hormonal and bodily

changes of puberty but also a period of tremendous neuronal development in the brain. The duration of adolescence in fact appears to be lengthening, with young people now delaying beginning their careers and marrying and having children later in life (Sturman and Moghaddam, 2011; Dahl, 2004).

The focus here will remain primarily on the brain changes that occur during adolescence (Shaw et al, 2008; Giedd et al, 1999; Lebel and Beaulieu, 2011; Ostby et al, 2009). It is such findings that have sown the 'seeds' for the aforementioned 'neuromyths', in which teenagers are characterised as 'faulty' or 'defective'.

A look first however, towards some of the behavioural evidence implicated in the issue.

Behavioural Changes in Adolescence and Risk Taking

During adolescence social behaviour intensifies greatly (Csikszentmihalyi et al, 1977), as does emotional unpredictability (Steinberg, 2005), impulsivity (Chambers et al, 2014; Adriani and Laviola, 2003) and 'sensation seeking' (Adriani et al, 1998; Stansfield and Kirstein, 2006). Teens often experience exceptionally strong motivational drives to seek out new thrills and feel 'the need for varied, novel and complex sensations and experiences' (Zuckerman et al, 1979; 10). Sensation seeking behaviour peaks in early to mid-adolescence and impulse control develops more gradually through mid - late adolescence (Sturman and Moghaddam, 2012). This trend is perhaps unsurprising from an evolutionary perspective, for the struggle for independence associated with risk taking coincides with the need to find a sexual partner whilst at the peak of fertility (Steinberg, 2003).

It may be however, that adolescents in industrialised societies are engaging in unnecessarily risky behaviours as they seek their independence, as clearly was the case with the journalist in my opening statement. Adolescence has become a vulnerable period in our culture, when individuals are more likely to suffer accident, injury or even death from a number of different self-inflicted causes than at any other age (Kavanagh et al, 2007; Blum and Nelson- Mmari, 2004; Williams et al, 2002)*. Risk taking is more likely to occur in in highly emotional situations (Figner et al, 2009) and in the presence of peers, where oftentimes high risk activities are perceived as acceptable and even increase kudos within the group (Steinberg, 2008). Kavanagh (2007; pp.4-6) found with regard to risky driving, that peer passengers invoke more dangerous driving than parent passengers do. Both accidents and dangerous driving were not felt to be embarrassing by Kavanagh's adolescent subjects, in fact risky driving was seen as 'enjoyable' and 'not unsafe'. According to Nelson (2007) the experience of being socially accepted by one's peers may be processed in the brain in similar ways to other non–social rewards. The intersection of neural circuits handling social information and reward processing could therefore aid understanding of why much adolescent risk taking behaviour occurs in the presence of peers (Steinberg, 2008).

**Note 1*

In addition to being at higher risk of accident or injury, adolescents are also more likely to develop mental illness than children or adults. Commonly mood and eating disorders manifest in adolescence as well as addiction and psychotic illnesses such as schizophrenia (Paus et al, 2008; Sisk and Zehr, 2005; Volkmar, 1996). Blakemore (2012; 404) suggests that this therefore may be a particularly sensitive period for the neurobiological functions underlying these conditions.

Developmental scientists have tried to find the possible basis for adolescents' risk taking behaviours for around 25 years and findings have not supported many of the aforementioned popular media beliefs on why they may occur. Contrary to the newspaper headlines, adolescents are not found to be irrational, ignorant or defective in their risk *calculation*; adolescent logical reasoning and cognitive processing skills are measured at a similar level to adults (Steinberg, (2008; 81). Furthermore, adolescents do not view risk and their susceptibility to it all that differently to adults, in fact sometimes risk may be over-estimated by teenagers, for example going to jail, dying young or becoming pregnant (de Bruin et al, 2007).

To investigate further, it is necessary to consider the problem from two perspectives; the neurobiological and the environmental/cultural.

Some of the most significant neural changes to occur in the brain during adolescence are explored in the following section.

Synpatic Growth and Pruning

Synaptogenesis* occurs early on in postnatal development and extends into childhood, at this stage surpassing adult levels (Blakemore, 2012). During adolescence, synaptic pruning takes place (Cragg, 1975; Chechik et al, 1998) where unused neuronal connections are eliminated and regularly used synapses are strengthened (Changeux and Danchin, 1976; Low and Cheng, 2006). The pruning process sometimes continues into the adult's thirties (Petanjek et al, 2011; Shaw et al, 2008). Initially the motor and sensory areas are pruned followed later by the lateral and prefrontal cortices (PFC); regions related to cognitive control and decision making. Synaptic pruning alongside the myelination process described below, enable more efficient connectivity between the PFC and other brain areas and thereby more competent evaluation of risk and reward (orbitofrontal cortex) and maturation of , decision-making (ventromedial prefrontal cortex) and impulsivity (dorsolateral prefrontal cortex) (Giedd et al, 1999; Gogtay et al 2004; Sowell et al, 2001).

**Note 2*

Myelination

Myelination is the process by which glial cells (oligodendrocytes and Schwann cells) generate the fatty insulating material myelin to encase the nerve axons and thus improve efficiency of conduction. The myelin enables the conduction of electrical signals along axons at high speeds (NGIDD, 2010). During synaptic pruning in adolescence, grey matter is reduced, whilst white matter is increased from extensive myelination occurring (Barnea-Goraly et al, 2005; Giedd et al, 1996, 1999, Paus et al, 1999)*. Myelination follows chronological sequence in the brain and the final regions to be insulated are the association areas, the PFC (Vackovlev and Lecours, 1967). Though connectivity becomes more efficient through the myelination process, the brain also becomes less plastic and therefore less receptive to new learning (Giedd, 2011).

So, it can be seen from this evidence that the adolescent brain is undergoing a period of extreme refinement and maturation and the rate at which some regions mature is different to others. White matter develops progressively whilst grey matter is progressive at some points and regressive at others (Blakemore and Choudhury, 2006). It has been revealed that regions associated with basic sensory and motor functioning mature first, whilst higher cognitive areas follow later. Those regions that undergo the most prolonged maturation period are the PFC and the Superior Temporal Sulcus (STS), both involved with executive function and social cognition *(ibid).* The possible implications of this restructuring process are discussed later.

Reorganisation in the dopaminegic system

Adolescents' risky behaviour may also be rooted in how reward is experienced. During adolescence there is a significant reorganisation of the dopaminergic system. Dopamine is an important neurotransmitter* that plays a fundamental role in the experience of pleasure. When a neurotransmitter crosses the neuron's synapse, it is received at a specific site called a receptor (Chudley, 1996). During early puberty, there are significant alterations in both density and distribution of dopamine receptors in the pathways that connect the limbic system (emotion, rewards and punishments) and the PFC (cognitive control, decision making and planning) (Sisk and Foster, 2004; Sisk and Zehr, 2005). Dopaminergic activity within these pathways is highest in early adolescence. As dopamine has a vital role to play in the brain's reward system, the combination of increase, decrease and reorganisation of dopamine receptors, especially between the limbic and prefrontal regions, may lead to an increase in the adolescent's drive to experience risk and sensation (Steinberg, 2008; 84; Steinberg, 2013). Spear hypothesises that adolescents may even experience a mini- 'reward deficiency syndrome', as if they suffered functional dopamine deficits (Spear, 2000; 446-447). However, a number of other studies showing increased reward activity in adolescents' sub cortical brain areas cast some doubt on this hypothesis (Ernst et al, 2005; Galvan et al, 2006).

**Note 3, 4. See Appendix 1*

Changes in Oxytocin Levels During Puberty

Gonadal steroids (e.g oestrogen and testosterone) may also influence social memory and bonding processes (Nelson et al, 2005). They affect the production of receptors in the limbic system (amygdala, nucleus accumbens) for the neurotransmitter (and hormone) oxytocin (Miller et al, 1989; Chibbar et al, 1990). Oxytocin plays an important role in social bonding and also regulates memory for social stimuli (Insel and Fernald, 2004; Winslow and Insel, 2004). The hormonal changes in puberty may therefore be one reason for adolescents' increased emotional arousal (over adults and children), in limbic, paralimbic and medial prefrontal regions to emotional stimuli (Nelson et al, 2005).

Furthermore, this may explain the huge significance adolescents place on other's opinions and the increased self-consciousness commonly experienced until around fifteen years old (Ranking et al, 2004).

Neurobiological Hypotheses for Adolescent Risk Taking Behaviours

Adolescents may take greater risks than adults because of differences in the developmental trajectory of their PFC compared to sub cortical structures (amygdala and ventral striatum) (Somerville and Casey, 2010; Galvan et al, 2006), as discussed in earlier sections. Galvan et al (2006) showed similarity between activity in the nucleus acumbens (reward centre) between adolescents and adults, but less activity in the orbitofrontal cortex, which appeared to be closer to the activity patterns of children. This disparity in maturity levels between the sub cortical systems and the PFC (cognitive control) may explain why teenagers more readily seek out novelty and sensation through high risk activities (Sturman and Moghaddam, 2011). Similarly, the triadic node model (Ernst et al, 2006) contends that activity in three different nodes corresponding to particular brain regions; ventral striatum – 'reward approach', amygdala – 'punishment avoidance' and PFC – 'modulation', are unbalanced in the adolescent brain, so the teen's natural hypersensitivity in the reward system is not sufficiently regulated by PFC activity, leading to a loss of self-monitoring and inhibitory control (Ernst et al, 2009). Steinberg (2008) claims that between adolescence and adulthood, cognitive control over affective drives is increased due to developing connectivity between cortical and sub cortical regions and this occurrence leads to decreased risk taking.

**Note 4*

The key overarching theme clearly, is that the risk taking/ reward seeking behaviours and emotional turbulence of adolescence are partly due to a combination of neurobiological factors:

- Heightened emotional sensitivity
- 'Immature' prefrontal neuronal activity
- Connectivity between cortical and sub cortical structures still developing
- Hormonal changes affecting neurotransmitter levels

However, psychosocial and cultural elements must also be taken into account and these are explored in the next section.

Cultural Factors

If adolescence was universally characterised by biologically driven emotional strife, egocentricity and confrontation, then surely we would see teenagers from every culture acting similarly? This is not the case; Schlegel and Barry (1991) studied 186 pre-industrial societies and found no adolescent psychopathology or anti-social behaviour in 50 % the cultures observed and only mild forms in the other 50%. Whiting and Whiting (1991) ran similar studies and found that teens became troubled only after the introduction of Western culture, in particularly schooling methods and television.

In Western society, do our teenagers often suffer in one of two ways? Whilst some may be overprotected, cossetted even…as Epstein (2007; 58) suggests: "infantilised" and "trapped in a frivolous world of teen culture", others may be neglected, unsupported and left to seek out their thrills in anti-social activity and find 'mentors' in equally troubled peers.

Choudhury (2010) points out that anti-social adolescent features common in the West, for example 'intergenerational conflict' (Blakemore, 2012) are rare in collectivist societies, for such traits are so deeply rooted in individualism.

Blakemore (2012; 404) speculates that the extensive synaptic reorganisation of adolescence may reveal an especially sensitive period for 'experiential input' to the brain. Is it possible that it is also a particularly receptive period for imitation learning? Many scientists claim that the mirror neuron system (MNS) has evolved as an adaptation for social understanding, empathy and theory of mind (Gallese et al, 1996; Rizzolatti et al, 2004), that it constitutes a "mapping mechanism between the observation of action and its execution" (Dinstein et al, 2009; 13). As such, when one individual observes another's action, mirror neurons in their brain activate and evoke a motor response to match the observed action. We know that adolescents in our society are deeply affected by the thoughts and actions of peers…maybe their super sensitivity could be utilised to better aid their development. Could regular, extended contact periods with wiser, older role models as well as their peers, perhaps be beneficial; mentors whose thoughts, actions and impulsivity are no longer as turbulent as their own, whose actions are mature, rational and grounded.

Experiential, imitative learning for adolescents is a central part of tribal life. Bravery, resilience and independence is both expected and celebrated in such cultures. Teenagers spend regular time with older adults and enjoy strong support and guidance from their family elders. They are intentionally and firmly guided through extreme risk and 'sensation' experiences, life threatening experiences even, in elaborate 'coming of age' ceremonies. '*Walkabout*' for adolescent Aboriginal boys (Aboriginal Culture.org) and '*Enkipaata*' for 14 year old males of the African Masai tribe (Masai-Association.org) are examples of intense self-sufficiency and survival challenges.

Perhaps the combination here of guided high risk experience with deep respect, acknowledgement and celebration of the extraordinary journey that adolescence is, could be fundamentally supportive elements in meeting adolescents' needs… and possibly are features absent from the lives of many Western teenagers.

Public Briefing /Conclusion

There is much scientific evidence to show that adolescence is a unique period of life during which extensive growth and development occurs in brain structure and function. The adolescent brain is predisposed towards learning and risk experiences – both fundamental elements to making a positive transition to adulthood. It is now known that the brain continues to develop well into adult life. This is a natural and necessary process that should be expected and embraced by parents of teenagers, not feared for its possible stress and turbulence.

The past fifteen years have provided many MRI studies showing significant adolescent brain changes that may be partly implicated in their risk taking activities. A key notion is that the brain's cognitive control system, responsible for decision-making and forward planning, is in a rapid process of growth and refinement at this age and is therefore not yet fully able to control and regulate typically impulsive, reward seeking behaviours. The control is lost further in the presence of peers due to an 'overlap' between the brain's social processing areas and its reward system. Connectivity, neural signalling and therefore impulse control gradually improve as the individual approaches adulthood.

Just because there are brain changes in this sensitive growth period does not mean the teenage brain is defective, as newspaper headlines might have us believe. The toddler's brain is not malfunctioning when she cannot yet hold an adult conversation and so it is with the teenager who seeks out risk and adventure or acts irrationally. The problem comes when natural teen behaviour becomes anti-social behaviour and that issue, perhaps, is rooted partly in culture and environment; indeed research findings have shown anti-social adolescent behaviour problems are rare in pre-industrialised societies.

More scientific research is needed to find ways of supporting and guiding adolescent risk taking behaviour in the West to reduce possibilities of harm. Perhaps one source of wisdom to be mined in this respect could be these environments. Further discussion between policy makers, neuroscientists and psychologists should take place, so that fresh research on the adolescent brain and behaviour can be utilised to improve teenagers' potential outcomes.

In sum, the teen brain debate could perhaps lose its unconstructive spotlight on 'faulty and troublesome' and instead more positively promote the teens as an age ripe for learning, ready for responsibility and intensely keen for challenge and adventure.

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NOTES

1. A review in the UK of other statistical evidence relating to teenage death or injury through risk taking reveals that with regard to drug taking, the risk to 12 – 24 year olds is in fact less than those in their immediately older age group, except for young people from deprived backgrounds and those just released from prison, who are at the greatest risk. 11- 17 year old adolescents are those most likely to be admitted to hospital for alcohol poisoning, whilst for those older than 16 years, alcohol related injuries and conditions begin to steadily decrease. Road traffic accidents related to drinking are the highest risk factor for older adolescents, with 17 – 19 year olds being the most likely group to drink and drive and road traffic deaths being the most common in 16-24 year olds. The morbidity rate overall for adolescents doubles over that of pre-pubescent children (Dahl, 2004).

2. Synaptogenesis is the configuration of new neurons.

3. Within the brain there exists both grey matter and white matter. The grey matter consists of neuronal cell bodies, neuropil, myelinated and unmyelinated axons, glial cells and capillaries. The white matter has few cell bodies and more myelinated axons.
4. A neurotransmitter is chemical released by neurons to transmit signals.

APPENDIX ONE

Development of white matter tracts alongside changes in volume, density and thickness of grey matter have been described in many studies that relate to childhood and adolescence. For example, Sowell et al (1999) found 14 year olds had increased white matter volume and decreased grey matter volume in both the frontal and parietal cortices, in comparison to 9 year olds. This finding has been repeated in a number of different larger scale studies (Barnea-Goraly et al, 2005; Giedd et al, 1996, 1999, Paus et al, 1999). Additionally, progressive changes in white matter integrity have been revealed using magnetisation-transfer ratio (MTR) and fractional anisotropy (FA) in diffusion tensor MRI, an MRI technique that measures the diffusion of water molecules through body tissues and can reveal important microscopic details about tissue structure such as axonal organisation (Mori and Zhang, 2006).