

Choudhury, S., **Charman, T.** & Blakemore, S. J. (2008). Development of the teenage brain. *Mind, Brain and Education*, 2, 142-147.

Development of the teenage brain

Suparna Choudhury¹, Tony Charman² and Sarah-Jayne Blakemore³

¹Division of Social & Transcultural Psychiatry, McGill University, 1033 Pine Avenue West, Montreal, Quebec, H3A 1A1, Canada

²Behavioural & Brain Sciences, UCL Institute of Child Health, 30 Guilford Street, London WC1N 1EH and

³UCL Institute of Cognitive Neuroscience, 17 Queen Square, London WC1N 3AR, UK

Email: s.blakemore@ucl.ac.uk

Abstract

Adolescence is a time characterised by change - hormonally, physically and mentally. We now know that some brain areas, particularly the frontal cortex, continue to develop well beyond childhood. There are two main changes with puberty. Firstly, there is an increase in axonal myelination, which increases transmission speed. Secondly, there is a gradual decrease in synaptic density, indicating significant pruning of connections. These neural changes make it likely that cognitive abilities relying on the frontal cortex, such as executive functions and social cognitive abilities, also change during adolescence. Here we review recent research that has demonstrated development during adolescence of a variety of social cognitive abilities and their neural correlates.

Introduction

Although it has long been recognised that adolescence is a time when children undergo very significant social and cognitive development, until recently little consideration was given to any role that brain maturation might play in these developments. However, research has begun to suggest that the human brain may be adaptive to the behaviourally demanding social environment during adolescence. In recent years, there has been an increase in research into adolescent behaviour and cognition. This may in part be due to new data about brain development during this period and, on the other hand, may relate to broader social issues concerning reports of relatively high rates of psychiatric morbidity during adolescence evidenced epidemiologically and anecdotally and documented in popular media (Green et al. 2004). While adolescence presents a period of maturation in terms of cognitive control, reaction speed, reasoning and decision-making skills, compared with childhood, it also marks a period of increased rates of depression, substance abuse, suicide, eating disorders and other 'risky' behaviours (Dahl, 2004; Spear, 2000). It is likely that this paradox arises due to developmental trajectories of cognitive skills that do not occur in synch. For example, certain abilities, such as emotion regulation may develop later than others, such as motor control, during adolescence. What can knowledge about brain development tell us about the underlying reasons for strengths in certain abilities and vulnerabilities of others and the possibilities for interventions in the clinic, in the classroom and in general? Indeed, social, cultural and family life become increasingly complex in late childhood and adolescence and are likely to present new and challenging contexts for adolescents and a sensitive developmental period. It is likely that interactions between social as well as neural influences underpin behaviours linked to emotional and cognitive control that emerge in adolescence. The role of brain development in particular, however, remains relatively understudied.

In the past few years, several pioneering experiments have investigated the development of the brain and cognitive processes during this period of life. Even though brain adaptation can

occur throughout the lifespan, the maturational phases during early life, that is, during foetal development, childhood and adolescence are thought to be the most dramatic (Toga et al., 2006). Recent brain imaging studies suggest that cortical plasticity during adolescence is associated with cognitive development, particularly in executive function and social cognition. The first section of this paper summarises the cellular studies that first demonstrated anatomical brain developments during adolescence. We then go on to describe how recent brain imaging techniques have supported these findings and have shed some light on the trajectories of maturational processes in the brain during adolescence. The final section discusses research on social cognitive processing in adolescence and suggests that brain maturation may play a role in at least some of these changes, which in turn are likely to be linked to the large shifts in social behaviour that characterise this period. We suggest that this brain development may facilitate socio-cultural learning over the lifespan.

Brain development during adolescence

Histological studies of the adolescent brain.

The notion that the brain continues to develop after childhood is relatively new. Experiments on animals, starting in the 1950s, showed that sensory regions of the brain go through ‘sensitive periods’ soon after birth, during which time environmental stimulation appears to be crucial for normal brain development and for normal perceptual development to occur (e.g. Hubel & Wiesel, 1962). It was not until the late 1970s that research on post-mortem human brains revealed that some brain areas continue to develop well beyond early childhood, which in turn suggested that sensitive periods for the human brain may be more protracted than previously thought. It was shown that cellular events take different trajectories in different areas of the human brain (Huttenlocher, 1979). Synaptic density in the visual cortex reaches a peak during the fourth postnatal month, and is followed by the elimination of synapses and the stabilisation of synaptic density to adult levels before the age of four. In contrast, the structure of the prefrontal cortex

(PFC), the area of the brain associated with understanding other minds (social cognition) and coordinating thoughts and behaviours (executive function), undergoes significant changes during puberty and adolescence. Two main changes were revealed in the brain before and after puberty. As neurons develop, a layer of myelin is formed around their extension, or axon, from supporting 'glial' cells. Myelin acts as an insulator and massively increases the speed of transmission (up to 100 fold) of electrical impulses from neuron to neuron. Whereas sensory and motor brain regions become fully myelinated in the first few years of life, although the volume of brain tissue remains stable, axons in the frontal cortex continue to be myelinated well into adolescence (Yakovlev & Lecours, 1967). The implication of this research is that the transmission speed of neural information in the frontal cortex should increase throughout childhood and adolescence.

The second difference in the brains of pre-pubescent children and adolescents pertains to changes in synaptic density in the PFC. Early in postnatal development, the brain begins to form new synapses, so that the synaptic density (the number of synapses per unit volume of brain tissue) greatly exceeds adult levels. This process of synaptic proliferation, called synaptogenesis, can last up to several months or years, depending on the species of animal and brain region. These early peaks in synaptic density are followed by a period of synaptic elimination (or pruning) in which frequently used connections are strengthened and infrequently used connections are eliminated. This experience-dependent process, which occurs over a period of years, reduces the overall synaptic density to adult levels.

However, histological studies of monkey and human PFC have shown that synaptogenesis and synaptic pruning in this area has a particularly protracted time course. These studies show that there is a proliferation of synapses in the subgranular layers of the PFC during childhood and again at puberty, followed by a plateau phase and a subsequent elimination and reorganisation of prefrontal synaptic connections after puberty (Huttenlocher, 1979; Woo et al., 1997). According to these data, in the human brain, synaptic pruning occurs throughout adolescence and results in a net decrease in synaptic density in the PFC during this time.

MRI studies of adolescent brain development

The scarcity of post-mortem child and adolescent brains meant that knowledge of the adolescent brain was until recently extremely scanty. However, since the advent of Magnetic Resonance Imaging (MRI), a number of brain imaging studies, using large samples of participants, have provided further evidence of the ongoing maturation of the cortex into adolescence and even into adulthood. In the past few years, several MRI studies have been performed to investigate the development of the structure of the brain during childhood and adolescence in humans (cf. Paus, 2005). The brain areas that undergo pronounced development during adolescence, in particular medial PFC and parieto-temporal cortex, form parts of the “social brain,” that is the network of brain regions involved in understanding other people.

Grey matter and white matter in the adolescent cortex

One of the most consistent findings from these MRI studies is that there is a steady linear increase in white matter (WM) in certain brain regions during childhood and adolescence. This changes has been highlighted both in frontal and temporo-parietal regions (Sowell et al., 1999). Myelin appears white in MRI scans, and therefore the increase in WM and decrease in grey matter (GM) with age was interpreted as reflecting increased axonal myelination in frontal cortex. The increased WM and decreased GM density in frontal as well as temporal and parietal cortex throughout adolescence has now been demonstrated by several studies carried out by a number of different research groups with increasingly large groups of participants (e.g. Giedd et al., 1996; 1999).

While the increase in WM in certain brain regions seems to be linear across all brain areas, the changes in GM density appear to follow a region-specific, non-linear pattern. MRI data demonstrate that changes in the frontal and parietal regions are similarly pronounced (Giedd et al., 1999). The study by Giedd and colleagues showed that the volume of GM in the frontal lobe

increased during pre-adolescence with a peak occurring at around 12 years for males and 11 years for females, followed by a decline after adolescence. Similarly, parietal-lobe GM volume increased during the pre-adolescent stage to a peak at around 12 years for males and 10 years for females, again followed by decline during post-adolescence. GM development in the temporal lobes was also non-linear, but the peak was reached later at about 17 years. While frontal and parietal cortex development is relatively rapid during adolescence (Sowell et al., 2001), GM volume in the superior temporal cortex, including superior temporal sulcus (STS), steadily declines during adolescence and across the lifetime, following an inverted U-curve and reaching maturity relatively late (Gogtay et al., 2004; Toga et al., 2006).

In a study of participants aged between four and 21, frontal lobe maturation was shown to occur in a back-to-front direction, starting in the primary motor cortex (the precentral gyrus), then extending anteriorly over the superior and inferior frontal gyri (Gogtay et al., 2004). The PFC was shown to develop relatively late. In the posterior half of the brain, the maturation began in the primary sensory area, spreading laterally over the rest of the parietal lobe. Lateral temporal lobes were the last to mature. It has been proposed that those brain areas associated with more basic cognitive skills, such as motor and sensory functions matured first, followed by brain areas including parietal cortex linked to spatial orientation and attention, and finally the regions related to executive function and social cognition (PFC).

The MRI results demonstrating a non-linear decrease in GM in various brain regions throughout adolescence have been interpreted in two ways. First, it is likely that axonal myelination results in an increase in WM and a simultaneous decrease in GM as viewed by MRI. A second, additional explanation is that the GM changes reflect the synaptic reorganisation that occurs at the onset of and after puberty. Thus, the increase in GM apparent at the onset of puberty might reflect a wave of synapse proliferation at this time. In other words, the increase in GM at puberty has been interpreted to reflect a sudden increase in the number of synapses. At some point after puberty, there is a process of refinement such that these excess synapses are eliminated

(Huttenlocher, 1979). It is speculated that this synaptic pruning is reflected by a steady decline in GM density seen in MRI (cf. Blakemore & Choudhury, 2006).

What do these developments of cortical structure mean for cognitive development? Some of the areas that undergo pronounced development during adolescence, in particular medial PFC and STS, are involved in social cognition, that is, understanding and interacting with others. Recent behavioural and neuroimaging studies have investigated the development of social cognition during adolescence. This research points to continued development of certain social cognitive processes during this period of life. This research is described in the following section.

Social cognitive development during adolescence

Behavioural and functional imaging studies consistently demonstrate maturation in executive functions during adolescence. These studies show improvements in reaction speeds, accuracy and changes in functional activation in associated frontal regions of the brain with age in tasks that involve attentional control, cognitive flexibility and problem-solving (e.g. Anderson et al., 2001; Casey et al., 1997; Tamm et al., 2002; see Blakemore & Choudhury, 2006 for review). There is less empirical data, however, relating to the association between social development and brain maturation in adolescence. Indeed, the period of adolescence involves new social encounters with peers and heightened awareness and interest in other people. The importance of evaluating other people may be associated with increased attention to socially salient stimuli, particularly faces, and the processing of emotional information and mental states. There is a rich literature on the development of social cognition in infancy and childhood pointing to step-wise changes in mentalising (that is, the attribution of mental states) during the first five years of life (Frith & Frith, 2006). However, while the early development of mentalising has been well studied, there has been surprisingly little empirical research on social cognitive development beyond childhood. In the following section, we summarise some of the key findings from studies investigating the implications of brain development for social cognition. There have been a relatively small

number of experiments investigating the development of social cognition during adolescence, most of which have focussed on face processing and mentalising.

Development of face processing during adolescence

Some of the earliest empirical studies on cognitive development during adolescence focussed on the effect of puberty on face recognition. In one study, female participants aged 6 to 16 years performed a face recognition task (Carey et al. 1980). While performance improved steadily during the first decade of life, this was followed by decline at around age 12. Puberty is implicated in this decline: a later study showed that mid-pubertal girls performed worse than prepubertal or postpubertal girls matched for age (Diamond et al. 1980). A more recent study also found evidence of a pubertal dip on a match to sample task in which emotional faces had to be matched to emotion words (McGivern et al. 2002). Participants ranged in age from 10 to 17 years. A 10-20% increase in reaction time on the match-to-sample task occurred in the children of pubertal age (10-11 year old girls and 11-12 year old boys) compared with younger children. Performance then improved, reaching its earlier level by about 16-17 years.

There is some indication that, for face processing tasks, activity in the frontal cortex increases between childhood and adolescence and then decreases between adolescence and adulthood. In an fMRI study of adolescents aged 13 to 17, the perception of happy faces compared with neutral was associated with significant bilateral amygdala activation (Yang et al., 2003). The effect of age was addressed by Thomas and colleagues (2001) in their investigation of amygdalar response to fearful facial expressions in two groups: a group of children (mean age 11 years) and adults (mean age 24). Adults relative to children demonstrated greater amygdala activation to fearful facial expressions, whereas children relative to adults showed greater amygdala activation to neutral faces. It was argued that the children perceived the neutral faces as more ambiguous than the fearful facial expressions, with resulting increases in amygdala activation to the neutral faces. Sex differences in amygdala-mediated cognitive development have

also been reported to occur during adolescence. In an fMRI study that investigated the recognition of fearful faces, female (but not male) participants showed increased activation in dorsolateral PFC in response to fearful faces between childhood and adolescence (Killgore et al. 2001). Another study reported increased activity in PFC (bilaterally for girls; right sided for boys) in response to fearful faces between age 8 and 15 (Yurgelun-Todd & Killgore, 2006). In a recent study of face processing, when attention was directed to a non-emotional aspect of fearful (relative to neutral faces) activity was found to increase in orbitofrontal cortex in adolescents compared to adults (Monk et al. 2003).

Development of mentalising during adolescence

fMRI studies of mental state attribution also show decreases in activity between adolescence and adulthood. A recent fMRI study investigated the development of communicative intent using an irony comprehension task. A group of 12 adults (6 male), ranging in age from 23 to 33, and a group of 12 children (6 male), ranging in age from 9 to 14, were scanned (Wang et al. 2006). Children engaged medial PFC and left inferior frontal gyrus more than did adults in this task. The authors interpreted the increased medial PFC activity in children as reflecting the increased processing demands of this task (compared to adults) of needing to integrate several cues to resolve the discrepancy between the literal and intended meaning of an ironic remark.

A similar region of medial PFC was more highly activated by children than by adults in a recent fMRI study that involved thinking about one's own intentions (Blakemore et al. 2007). Thinking about your own, or someone else's intentions to act, requires mental state representation, so this task was expected to activate the mentalising network. A group of 19 female adolescents (age range 12 to 18) and a group of female adults (age range 22 to 38) were presented with scenarios about causes and effects. In both adults and adolescents, the intentional causality relative to the physical causality condition activated the classic mentalising network including medial PFC and posterior STS. However, the interaction between group and condition

resulted in differential activity within the mentalising network. Medial PFC was activated more by adolescents than by adults when thinking about intentions relative to thinking about physical causality. Conversely, a region in right STS was more active in adults than in adolescents when thinking about intentions relative to thinking about physical causality. These results suggest that the neural strategy for thinking about intentions changes between adolescence and adulthood. Although the same neural network is active, the relative roles of the different areas change, with activity moving from anterior (medial PFC) regions to posterior (STS) regions with age.

While as yet there have only been a handful of developmental neuroimaging studies of social cognition, there does seem to be some consistency with respect to the direction of change in frontal activity. Overall, the studies reviewed above have found that activity in PFC decreases during adolescence. This might be because adolescence represents a time in which the frontal cortex is being fine-tuned by synaptic pruning. Thus, with age, less activity (as seen in fMRI) is associated with carrying out the task in question. An alternative, or additional, explanation is that there is a change in the cognitive strategy for mentalising, which will result in different brain regions being recruited.

Whichever explanation turns out to be the correct one, the data might have implications for learning and behavioural during adolescence. It is proposed that synaptic pruning in early development fine-tunes neural circuitry in an input-dependent manner. Synaptic pruning is thought to underlie sound categorisation, for example. Learning one's own language initially requires categorising the sounds that make up language. New-born babies are able to distinguish between all speech sounds. Sound organisation is determined by the sounds in a baby's environment in the first 12 months of life – by the end of their first year babies lose the ability to distinguish between sounds to which they are not exposed (see Kuhl, 2004). The ability to distinguish certain speech sounds depends on being exposed to those distinct sounds in early development. Before about 12 months of age babies brought up in the USA can detect the difference between certain sounds common in the Hindi language, which after 12 months they

cannot distinguish (Werker et al. 1981). In contrast babies brought up hearing the Hindi language at the same age become even better at hearing this distinction because they are exposed to these sounds in their language. This fine-tuning of sound categorisation is thought to rely on the pruning of synapses in sensory areas involved in processing sound.

It is unknown whether the pruning of synapses in frontal cortex during adolescence is similarly influenced by the environment. If this turns out to be the case, it would have profound implications for the kinds of experiences and environments that are optimal for teenage brain development.

Conclusion and implications for teenagers

In this paper, we have reviewed evidence that certain brain regions undergo substantial development during adolescence. Recent studies of social cognitive development, coupled with neuroanatomical findings, during this period of life suggest that there is a peak in activity within PFC during early adolescence, which reflects the peak in synaptic density and grey matter in this region. It has been suggested that the functioning of PFC is temporarily perturbed by the wave of synaptogenesis that occurs at puberty. Only later in adolescence does synaptic pruning render this region more efficient, such that less neural activity will correlate with the same task performance.

Research into the cognitive implications of continued brain maturation beyond childhood may be relevant to understanding the social development and educational attainment of adolescents. Further studies are necessary to reach a consensus about how axonal myelination and synaptic proliferation and pruning impact on social, emotional, linguistic, mathematical and creative development. Several important questions deserve to be investigated: Which skills undergo perturbation? Which undergo sensitive periods for learning? How does the quality of the environment interact with brain changes in the development of cognition? Whether greater emphasis on social and emotional cognitive development would be beneficial during adolescence

is unknown but research will provide insights into potential intervention schemes in secondary schools.

References

- Anderson, V., Anderson, P., Northam, E., Jacobs, R., Catroppa, C. (2001). Development of executive functions through late childhood and adolescence in an Australian sample *Developmental Neuropsychology*, 20,385-406
- Blakemore, S.J., Choudhury, S. (2006). Development of the adolescent brain: implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry*, 47, 296-312.
- Blakemore SJ, den Ouden H, Choudhury S, Frith C. (2007). Adolescent development of the neural circuitry for thinking about intentions. *Social Cognitive and Affective Neuroscience*, 2,130-139
- Carey, S., Diamond, R., & Woods, B. (1980). The development of face recognition – a maturational component. *Developmental Psychology* 16, 257-269
- Casey, B., Trainor, R., Orendi, J. (1997). A developmental functional MRI study of prefrontal activation during performance of a go-no-go task *Journal of Cognitive Neuroscience*, 9, 835-847.
- Dahl, RE. (2004). Adolescent brain development: a period of vulnerabilities and opportunities. *Annals of the New York Academy of Sciences*, 1021, 1-22.
- Diamond, R., Carey, S., & Back, K. (1983). Genetic influences on the development of spatial skills during early adolescence. *Cognition* 13, 167-185.
- Frith, C.D., & Frith, U. (2006) The neural basis of mentalizing. *Neuron*, 50, 531-4.
- Giedd, J.N., Blumenthal, J., Jeffries, N.O., Castellanos, F.X., Liu, H., Zijdenbos, A., Paus, T., Evans, A.C., Rapoport, J.L. (1999). Brain development during childhood and adolescence: a longitudinal MRI study. *Nature Neuroscience* 2, 861-863.

Giedd, J.N., Snell, J.W., Lange, N., Rajapakse, J.C., Kaysen, D., Vaituzis, A.C., Vauss, Y.C., Hamburger, S.D., Kozuch, P.L., Rapoport, J.L. (1996). Quantitative magnetic resonance imaging of human brain development: ages 4-18. *Cerebral Cortex* 6, 551-560

Gogtay, N., Giedd, J.N., Lusk, L., Hayashi, K.M., Greenstein, D., Vaituzis, A.C., Nugent, T.F. 3rd, Herman, D.H., Clasen, L.S., Toga, A.W., Rapoport, J.L. & Thompson, P.M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Science, USA* 101, 8174-8179.

Green H, McGinnity A, Meltzer H, Ford T, Goodman R. *Mental Health of children and Young People in Great Britain*, (2004.) ONS, HMSO: London.

Hubel, D.N. and Wiesel, T.N. (1962). Receptive fields, binocular interactions and functional architecture in the cat's visual cortex. *Journal of Physiology* 160, 106-154.

Huttenlocher, P.R. (1979). Synaptic density in human frontal cortex – developmental changes and effects of aging. *Brain Research* 163, 195-205.

Killgore, W.D.S., Oki, M., Yurgelun-Todd, D.A. (2001). Sex-specific developmental changes in amygdale responses to affective faces. *Neuroreport* 12, 427–433.

Kuhl, P.K. (2004). Early language acquisition: Cracking the speech code. *Nature Reviews Neuroscience* 5, 831-843

McGivern RF, Andersen J, Byrd D, Mutter KL & Reilly J. (2002). Cognitive efficiency on a match to sample task decreases at the onset of puberty in children. *Brain and Cognition*. 50, 73-89.

Monk CS, McClure EB, Nelson EE, Zarahn E, Bilder RM, Leibenluft E, Charney DS, Ernst M, Pine DS. (2003). Adolescent immaturity in attention-related brain engagement to emotional facial expressions. *Neuroimage* 20, 420-428.

Paus, T. (2005). Mapping brain maturation and cognitive development during adolescence. *Trends in Cognitive Sciences* 9, 60-68.

Sowell, E.R., Thompson, P.M., Tessner, K.D., Toga, A.W. (2001). Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex: Inverse relationships during postadolescent brain maturation. *Journal of Neuroscience* 21, 8819-8829.

Sowell, E.R., Thompson, P.M., Holmes, C.J., Batth, R., Jernigan, T.L., Toga, A.W. (1999). Localizing age-related changes in brain structure between childhood and adolescence using statistical parametric mapping. *Neuroimage* 6, 587-597.

Spear, L. (2000). The adolescent brain and age-related behavioural manifestations *Neuroscience and Biobehavioural Review*, 24, 417-463.

Tamm, L., Menon, V., Reiss, A. (2002). Maturation of brain function associated with response inhibition *Journal of the American Academy of Child & Adolescent Psychiatry*, 41, 1231-1238

Thomas, K.M., Drevets, W.C., Whalen, P.J., Eccard, C.H., Dahl, R.E., Ryan, N.D., & Casey, B.J. (2001) Amygdala response to facial expressions in children and adults. *Biological Psychiatry* 49, 309-316.

Toga, A.W., Thompson, P.M., & Sowell, E.R. (2006). Mapping brain maturation. *Trends in Neurosciences*, 29, 148-159.

Wang, AT, Lee, SS, Sigman, M, Dapretto, M. (2006). Developmental changes in the neural basis of interpreting communicative intent. *Social Cognitive and Affective Neuroscience* 1, 107-121.

Werker JF, Gilbert JH, Humphrey K, Tees RC. (1981). Developmental aspects of cross-language speech perception. *Child Development* 52, 349-55.

Woo, T.U., Pucak M.L., Kye C.H., Matus C.V. & Lewis D.A. (1997). Peripubertal refinement of the intrinsic and associational circuitry in monkey prefrontal cortex. *Neuroscience* 80, 1149-1158.

Yakovlev, P.A. & Lecours, I.R. (1967). The myelogenetic cycles of regional maturation of the brain. In A. Minkowski (Ed.), *Regional development of the brain in early life* (pp. 3-70). Oxford: Blackwell.

Yang, T.T., Menon, V., Reid, A.J., Gotlib, I.H., Reiss, A.L. (2003). Amygdalar activation associated with happy facial expressions in adolescents: a 3-T functional MRI study. *Journal of American Academy of Child & Adolescent Psychiatry* 42, 979-985.

Yurgelun-Todd, D.A., Killgore, W.D. (2006). Fear-related activity in the prefrontal cortex increases with age during adolescence: a preliminary fMRI study. *Neuroscience Letters* 406, 194–199.