Development of the social brain during adolescence

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Adolescence is usually defined as the period of psychological and social transition between childhood and adulthood. The beginning of adolescence, around the onset of puberty, is characterized by large hormonal and physical changes. The transition from childhood to adulthood is also characterized by psychological changes in terms of identity, self-consciousness, and cognitive flexibility. In the past decade, it has been demonstrated that various regions of the human brain undergo development during adolescence and beyond. Some of the brain regions that undergo particularly protracted development are involved in social cognitive function in adults. In the first section of this paper, I briefly describe evidence for a circumscribed network of brain regions involved in understanding other people. Next, I describe evidence that some of these brain regions undergo structural development during adolescence. Finally, I discuss recent studies that have investigated social cognitive development during adolescence.

The first time Uta Frith made an impression on me was when I was 15. That year I was given a copy of her book *Autism: Explaining the Enigma* (U. Frith, 1989), which had recently been published. I knew nothing about autism and found Uta’s book captivating. It inspired me to write to its author and ask if I could do a week’s work experience in her lab. With characteristic generosity, Uta agreed. So in the summer of 1990, I spent a week in the Medical Research Council (MRC) Cognitive Development Unit, where I observed children with autism being tested on the Sally Anne task, and joined in when Uta’s group were generating spoonerisms like Dob Bylan and Himi Jendrix. At the time, I didn’t quite realise that this research had revolutionized what is known about autism and dyslexia. Together with Simon Baron-Cohen and Alan Leslie, Uta had just a few years earlier published the first paper to show that children with autism have problems passing Theory of Mind tasks. If you search for [Theory of Mind in autism] on the web today you get over one million entries! Uta’s work on autism and dyslexia is not just renowned amongst scientists, but has also made a significant impact on teachers, clinicians, carers, and parents. Doing work experience with Uta all those years ago was truly inspirational.

I met Uta again when I was doing a PhD with Chris Frith. In 2000, Uta asked me to help her write a report for the Economic and Social Research Council (ESRC) on the implications of brain research for education. Uta had the vision to realise that this would soon become an important area of science. Indeed, brain and education research is now a flourishing discipline in itself,
with ring-fenced funding, dedicated international conferences, books, and a new journal. While writing the ESRC report, Uta and I were struck by the scarcity of literature on brain research that was aimed at educators. This seemed curious since some areas of brain research are very relevant to education (and in other areas the implications are far from clear). In addition, we came across the substantial market of educational tools that make claims about the brain. Our experience was that there is real interest amongst educators about these claims and the research they are based on. As a consequence, Uta and I decided to write a book on the subject (Blakemore & Frith, 2005). On the basis of Uta’s previous well-received and successful books (e.g., U. Frith, 1989, 2003; Houston & Frith, 2000), her long-time publisher, Blackwell, agreed to publish our book.

One of the areas of brain research that was just starting to take off at the time we wrote our book was development during adolescence. Much is known (mostly from animal research) about brain changes in the early years. In comparison, at the time of writing our book, there was a large gap in knowledge about brain development after early childhood, possibly because the extant research was carried out on animal brain tissue, and, unlike humans, animals do not go through extended periods of adolescent maturity. There were a handful of new magnetic resonance imaging (MRI) studies looking at development of the human adolescent brain, and these were pointing to significant waves of change in several areas of cortex. This was fascinating, not least because several developmental disorders develop during or just after adolescence. For example, schizophrenia is a disorder that usually develops at the end of adolescence. Was it possible that postpubescent cortical sculpting does not proceed normally in people who develop schizophrenia? While there are indications that neuropathology occurs in early development in schizophrenia (e.g., Weinberger, 1987), recently it has been suggested that aberrations in neurodevelopmental processes might also take place during the adolescent years (McGlashan & Hoffman, 2000). When Uta and I were researching our book, there was relatively little research on brain development in typically developing adolescents and even less on adolescents who later develop schizophrenia.

Another field in which Uta’s research has been influential from the start is social neuroscience. Together with Chris Frith and other colleagues, Uta published some of the first papers in this new area. In the next section, I describe evidence for a network of brain regions dedicated to understanding others.

The social brain

It is only in the past decade or two that the search for the biological basis of social behaviour began (Cacioppo & Berntson, 1992). Uta Frith was among the first to study the brain basis of social cognitive processes, in particular Theory of Mind (ToM; or what Uta termed “mentalizing”). Mentalizing is defined as the ability to attribute mental states to other people in order to predict their behaviour (e.g., U. Frith & Frith, 2003; see also Perner & Leekham, 2008 this issue; Sodian, 2008 this issue).

Uta and colleagues published one of the first neuroimaging studies of mentalizing in 1995 (Fletcher et al., 1995). In this positron emission tomography (PET) experiment, subjects read stories that required mental state attribution (ToM stories), stories about physical, natural events that did not require any attribution of mental states (physical stories), and paragraphs made up of unlinked sentences that were unconnected with each other and did not constitute a story. The Theory of Mind (ToM) stories, relative to unlinked sentences, activated the superior temporal sulcus (STS), medial prefrontal cortex (mPFC), precuneus/posterior cingulate cortex, and temporal poles (see Figure 1). Compared with the physical stories, the ToM stories activated the mPFC and precuneus, as well as the right inferior parietal cortex adjacent to the temporoparietal junction (TPJ).

Since this ground-breaking paper, there have been dozens of neuroimaging studies investigating the neural basis of mentalizing, each showing
remarkable agreement with this first study. Here, I briefly review the evidence that mentalizing is associated with activation of the same circumscribed neural network. Recent neuroimaging studies, some by Uta Frith’s group, others by labs all over the world, have used a wide range of stimuli including verbal stories (Gallagher et al., 2000; Saxe & Kanwisher, 2003), sentences (den Ouden, Frith, Frith, & Blakemore, 2005; see Figure 2), words (Mitchell, Heatherton, & Macrae, 2002), cartoons (Brunet, Sarfati, Hardy-Bayle, & Decety, 2000; Gallagher et al., 2000), and animations (Castelli, Happé, Frith, & Frith, 2000; see Figure 3). These studies have replicated the original finding by Fletcher and colleagues (Fletcher et al., 1995) of activation in mPFC, STS/TPJ, and the temporal poles when subjects think about mental states (see C. D. Frith & Frith, 2006; U. Frith & Frith, 2003, for review).

The same brain regions have been implicated in mentalizing from lesion studies. In particular, lesions to the frontal cortex (Channon & Crawford, 2000; Gregory et al., 2002; Happé, Malhi, & Checkley, 2001; Rowe, Bullock, Polkey, & Morris, 2001; Stone, Baron-Cohen, & Knight, 1998; Stuss, Gallup, & Alexander, 2001) and STS/TPJ (Apperly, Samson, & Humphreys, 2005; Samson, Apperly, Chiavarino, & Humphreys, 2004) impair mentalizing performance. One exception is a study that Uta Frith was involved with (Bird, Castelli, Malik, Frith, & Husain, 2004). The researchers studied a patient with damage to much of her frontal cortex, including the whole of mPFC.
Surprisingly (given the neuroimaging literature), the patient showed normal performance on mentalizing tasks. Whether this rules out mPFC as being necessary for mentalizing, or whether this patient’s good performance was because of neuronal reorganization, is unclear. Uta often says that surprising results, results that one would never predict, can be more important than predicted results. This is a clear example of a surprising result, which needs to be considered in theories of mPFC function.

The roles of the different regions in mentalizing are the subject of much debate. The mPFC is activated when subjects think about psychological states even if those states are applied to animals (Mitchell, Banaji, & Macrae, 2005). The mPFC is also activated by tasks that involve thinking about mental states in relation to the self (Johnson et al., 2002; Lou et al., 2004; Ochsner et al., 2004; Vogeley et al., 2001). Games that involve surmising an opponent’s mental states also activate the mPFC (e.g., Gallagher, Jack, Roepstorff, & Frith, 2002; McCabe, Houser, Ryan, Smith, & Trouard, 2001). All of these tasks involve thinking about mental states. One prominent theory of mPFC function is that it is activated whenever subjects reflect on mental states (e.g., Amodio & Frith, 2006). Frith (C. D. Frith, 2007) has proposed that the mPFC is involved in the necessary decoupling of mental states from reality. Activity in mPFC is often observed during rest conditions in comparison with higher demand tasks (including mentalizing; Gusnard & Raichle, 2001). It has been suggested that, during rest or low-demand tasks, participants might indulge in spontaneous mentalizing (Amodio & Frith, 2006).

The STS has been proposed to play a role in the prediction of observed patterns of behaviour in order to surmise the mental states underlying this behaviour (C. D. Frith, 2007; C. D. Frith & Frith, 2006; U. Frith & Frith, 2003). This region is activated during the perception of biological motion (e.g., Allison, Puce, & McCarthy, 2000; Grezes et al., 2001; Grossman et al., 2000), faces and body parts (e.g., Campbell et al., 2001; Chao, Haxby, & Martin, 1999; Puce, Allison, Bentin, Gore, & McCarthy, 1998) and eye movements (e.g., Pelphrey, Morris, Michelich, Allison, & McCarthy, 2005). One possibility is that this region is involved in predicting observed movements (C. D. Frith, 2007).

In summary, a network of brain regions including mPFC and STS/TPJ seems to be involved in many aspects of social cognition. In the next section I review evidence that these brain regions develop over several decades in humans.

Development of social cognition

There is a rich literature on the development of social cognition in infancy and childhood, and here I will not go into any detail (this literature is reviewed in papers by Perner & Leekham, 2008; Sodian, 2008). Signs of social competence develop during early infancy, such that by around 12 months of age infants can ascribe agency to a system or entity (Johnson, 2003; Spelke, Phillips, & Woodward, 1995). The understanding of intention emerges at around 18 months, when infants acquire joint attention skills—for example, they are able to follow an adult’s gaze towards a goal (Carpenter, Nagell, & Tomasello, 1998). These early social abilities precede more explicit mentalizing, such as false-belief understanding, which usually emerges by about four or five years (Barresi & Moore, 1996). While normally developing children begin to pass Theory of Mind tasks by age five, the brain structures that underlie mentalizing (mPFC and STS/TPJ) undergo substantial development well beyond early childhood. These studies are reviewed in the next section.

Cellular development in the brain during adolescence

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 (Hubel & Wiesel, 1962). These experiments suggested that the human brain might be susceptible to the same
sensitive periods in early development. Research on postmortem human brains carried out in the 1970s revealed that some brain areas, in particular the PFC, continue to develop well beyond early childhood (Huttenlocher, 1979; Huttenlocher, De Courten, Garey, & Van Der Loos, 1983; Yakovlev & Lecours, 1967).

Two main changes were found in the brain before and after puberty. As neurons develop, a layer of myelin is formed around their axon. Myelin acts as an insulator and significantly increases the speed of transmission of electrical impulses from neuron to neuron. While sensory and motor brain regions become fully myelinated in the first few years of life, 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 might increase throughout childhood and adolescence.

The second difference in the brains of prepubescent children and adolescents pertains to changes in synaptic density in 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 synaptogenesis lasts up to several months, depending on the species of animal and the brain region. These early peaks in synaptic density are followed by a period of synaptic elimination (pruning) in which frequently used connections are strengthened and infrequently used connections are eliminated. This process, which occurs over a period of years, reduces the overall synaptic density to adult levels. In sensory regions of the monkey brain, synaptic densities gradually decline to adult levels at around three years, around the time monkeys reach sexual maturity (Rakic, 1995).

In contrast to sensory brain regions, histological studies of monkey and human PFC have shown 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 reorganization of prefrontal synaptic connections after puberty (Bourgeois, Goldman-Rakic, & Rakic, 1994; Huttenlocher, 1979; Woo, Pucak, Kye, Matus, & Lewis, 1997; Zecevic & Rakic, 2001). According to these data, 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

Until recently, the structure of the human brain could be studied only after death. The scarcity of postmortem brains in research meant that knowledge of human brain development was limited. Since the advent of magnetic resonance imaging (MRI), a number of brain-imaging studies have provided further evidence of the ongoing maturation of the frontal cortex and other regions, into adolescence and even into adulthood. A consistent finding from both cross-sectional and longitudinal MRI studies is that there is a steady increase in white matter (WM) in certain brain regions, particularly PFC and parietal cortex, during childhood and adolescence (e.g., Giedd et al., 1999; Giedd et al., 1996; Paus et al., 1999b; Pfefferbaum et al., 1994; Reiss, Abrams, Singer, Ross, & Denckla, 1996; Sowell et al., 2003; Sowell et al., 1999). Most studies point to a linear increase in white matter with age (Barnea-Goraly et al., 2005; Giedd et al., 1999; Paus et al., 1999a, 1999b; Pfefferbaum et al., 1994; Reiss et al., 1996; Sowell et al., 1999). In light of histological studies, this has been interpreted as reflecting continued axonal myelination during childhood and adolescence.

While the increase in white matter in certain brain regions seems to be linear, changes in grey matter (GM) density appear to follow a region-specific, nonlinear pattern. Several studies have shown that GM development in certain brain regions follows an inverted-U shape. In one of the first developmental MRI studies, Giedd et al., (1999) performed a longitudinal MRI study on 145 healthy boys and girls ranging in age from about 4 to 22 years. The volume of GM in the frontal lobe increased during
preadolescence with a peak occurring at around 12 years for males and 11 years for females. This was followed by a decline during postadolescence. Similarly, parietal lobe GM volume increased during the preadolescent stage to a peak at around 12 years for males and 10 years for females, and this was followed by decline during postadolescence. GM development in the temporal lobes was also nonlinear, and the peak was reached later at about 17 years. A similar inverted-U shaped developmental trajectory of GM in various cortical regions has been found in several subsequent MRI studies (e.g., Gogtay et al., 2004; Thompson et al., 2000). Most studies show that sensory and motor regions mature first, while PFC and parietal and temporal cortices continue to develop during adolescence and beyond.

In summary, several recent MRI studies have suggested that a perturbation in GM density more or less coincides with the onset of puberty in some cortical regions. This has been interpreted as reflecting the synaptic reorganization that occurs at the onset of puberty (Bourgeois et al., 1994; Huttenlocher, 1979). Thus, the increase in GM apparent at the onset of puberty (Giedd et al., 1999) might reflect a wave of synapse proliferation at this time, while the gradual decrease in GM density that occurs after puberty has been attributed to postpubescent synaptic pruning.

The brain regions that undergo protracted development include PFC, parietal cortex and superior temporal cortex (in some studies this has included STS). As discussed above, these are regions that are implicated in social cognition in adults. The effect of continued neural development in brain regions associated with social cognition is largely unknown. Only a relatively small number of studies have investigated social cognitive function during adolescence.

**Development of social cognition during adolescence**

While there is a wealth of social psychology research on socio-emotional processing in adolescence, there is surprisingly little empirical research on social cognitive development during this period. One area of social processing that has been studied in the context of adolescence is face processing, perhaps because early behavioural studies of face processing found evidence for an interruption at puberty in the developmental course of face recognition (Carey, Diamond, & Woods, 1980; Diamond, Carey, & Back, 1983). In one study the percentage of correct responses in a behavioural face recognition task improved by over 20% between the ages of 6 and 10 and declined by about 10% around the age of puberty (Carey et al., 1980). Performance on the task recovered again during adolescence. In another study, face encoding was found to be worse in pubescent girls than in pre- and postpubescent girls matched for age (Diamond et al., 1983).

Recently, several groups have investigated the neural processing of facial expressions of emotion in adolescents. Thomas et al. (2001) investigated amygdala activation to fearful facial expressions in a group of children (mean age 11 years) and adults. Adults demonstrated greater amygdala activation to fearful facial expressions, whereas children showed greater amygdala activation to neutral faces. Slightly different results were obtained by Killgore, Oki, and Yurgelun-Todd (2001). Results indicated sex differences in amygdala development: Although the left amygdala responded to fearful facial expressions in all children, left amygdala activity decreased over the adolescent period in females but not in males. Females also demonstrated greater activation of the dorsolateral PFC over this period, whereas males demonstrated the opposite pattern. In a recent study, bilateral PFC activity increased with age (from 8 to 15 years) for girls, whereas only activity in right PEC was correlated with age in boys (Yurgelun-Todd & Killgore, 2006).

In a recent study, a group of adolescents (aged 7 to 17) and a group of adults (aged 25–36) viewed faces showing emotional expressions. While viewing faces with fearful emotional expressions, adolescents exhibited greater activation than adults of the amygdala, orbitofrontal cortex, and anterior cingulate cortex (Monk et al., 2003). When subjects were asked to switch their attention
between a salient emotional property of the face (thinking about how afraid it makes them feel) and a nonemotional property (how wide is the nose), adults, but not adolescents, selectively engaged and disengaged the orbitofrontal cortex. These functional MRI (fMRI) results suggest that both emotion processing and cognitive appraisal systems develop during adolescence.

A recent fMRI study investigated the development during adolescence of the neural network underlying thinking about intentions (Blakemore, den Ouden, Choudhury, & Frith, 2007). In this study, 19 adolescent participants (aged 12.1 to 18.1 years), and 11 adults (aged 22.4 to 37.8 years), were scanned using fMRI. A factorial design was employed with between-subjects factor age group and within-subjects factor causality (intentional or physical). In both adults and adolescents, answering questions about intentional causality versus physical causality activated the mentalizing network, including medial PFC, STS and temporal poles. In addition, there was a significant interaction between group and task in the medial PFC. During intentional relative to physical causality, adolescents activated part of the medial PFC more than did adults and adults activated part of the right STS more than did adolescents. 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 prefrontal) regions to posterior (temporal) regions with age.

While face processing is an example of an area of social cognitive development during adolescence that has received attention in recent years, very little is known about how other aspects of social cognition change during the teenage years. It appears paradoxical that the same brain regions involved in social cognition undergo such dramatic development into adolescence, when the functions mediated by these regions (e.g., ToM) appear to mature much earlier. If an ability (such as passing a ToM task) is accomplished by early to mid childhood, it is unlikely that it will undergo dramatic changes beyond that time. One possibility is that neural development during adolescence influences more subtle abilities, such as the capacity to modulate social cognitive processes in the context of everyday life. Another possibility is that tasks that tap into implicit social cognitive processes might be more likely to undergo change during adolescence. We have recently found some evidence for this in the domain of action imagery (Choudhury, Bird, Charman, & Blakemore, 2007, in press). However, in the realm of development of social cognition during adolescence, there is a large gap in knowledge waiting to be filled.

CONCLUSION

After Uta Frith and colleagues’ seminal paper on the neural processing of Theory of Mind (Fletcher et al., 1995), much has been learned about the social brain. There is a general consensus about brain regions activated when subjects think about mental states, though the exact role each region plays in Theory of Mind processing is still debated. Some of these brain regions undergo substantial development during adolescence, which has implications for the development of social cognition, in particular understanding other people. Social cognitive development during adolescence is a new and rapidly expanding field. Many questions remain unanswered. The role of hormones, culture and the social environment on the development of the social brain are unknown. It is possible that changes in hormones and social environment (for example, changing school) interact with neural development at the onset of puberty. Future research is needed to disentangle the contributions of biological and environmental factors to the developing social brain.

REFERENCES


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