Annual Research Review: Towards a developmental neuroscience of atypical social cognition

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Abstract: As a starting point for our review we use a developmental timeline, starting from birth and divided into major developmental epochs defined by key milestones of social cognition in typical development. For each epoch, we highlight those developmental disorders that diverge from the normal developmental pattern, what is known about these key milestones in the major disorders affecting social cognition, and any available research on the neural basis of these differences. We relate behavioural observations to four major networks of the social brain, that is, Amygdala, Mentalizing, Emotion and Mirror networks. We focus on those developmental disorders that are characterized primarily by social atypicality, such as autism spectrum disorder, social anxiety and a variety of genetically defined syndromes. The processes and aspects of social cognition we highlight are sketched in a putative network diagram, and include: agent identification, emotion processing and empathy, mental state attribution, self-processing and social hierarchy mapping involving social ‘policing’ and in-group/out-group categorization. Developmental disorders reveal some dissociable deficits in different components of this map of social cognition. This broad review across disorders, ages and aspects of social cognition leads us to some key questions: How can we best distinguish primary from secondary social disorders? Is social cognition especially vulnerable to developmental disorder, or surprisingly robust? Are cascading notions of social development, in which early functions are essential stepping stones or building bricks for later abilities, necessarily correct? Keywords: Social cognition, autism spectrum disorders, theory of mind, empathy, brain development.

Introduction

The area of social neuroscience has flourished in recent years. In surveying the existing literature it is easy to find useful review articles on the brain basis of social cognition (e.g., Beer & Ochsner, 2006; Kennedy & Adolphs, 2012), either broadly or narrowly defined. There are also articles on brain development at various stages (e.g., preschool, Brown & Jernigan, 2012), and multiple reviews focusing on a single developmental disorder (most notably autism; e.g., Pelphrey, Shultz, Hudac & Van der Wyk, 2011). What we believe to be lacking is a developmental perspective on the topic of social neuroscience, informed by a cross-disorder perspective on atypical development. In this review article we use the chronology of typical social development as a framework to discuss what is known about the cognitive and neural bases of disorders affecting social interaction. While it is clear that social functioning is very important in intellectual disability, we will focus here only on those conditions in which social interaction is markedly discrepant from other areas of functioning. These notably include autism spectrum disorder, conduct disorders, social anxiety, and specific genetic syndromes such as Williams Syndrome. Psychiatric disorders with their usual onset in adulthood (e.g., Lee, 2013) and acquired brain damage (see Anderson & Beauchamp, 2012) are beyond the scope of this review. We also will not address here the question of gender differences, as this is a major topic in its own right with potential links to atypical social cognition. Finally, this review does not extend to the genetics of atypical social cognition, an important and emerging field requiring its own cross-disorder review.

The social brain

The capacity human beings have for social interaction and communication is constantly surprising even when compared with the ingenuity and inventiveness of our dealings with the physical world. A wide range of mental mechanisms are devoted to social information processing. A definitive taxonomy of these mechanisms has yet to be made (cf. Beer & Ochsner, 2006), but we attempt to provide a sketchy map of social cognitive processes towards the end of this review. Neuroscience has tried to illuminate some of the underlying mechanisms, driven largely by combining paradigms from experimental psychology and neuroimaging studies, in both neurotypical and atypical individuals, so far mainly in adults. For example, a meta-analysis of neuroimaging studies comparing social versus nonsocial cognitive tasks with both autistic and nonautistic groups yielded seemingly robust information on the brain regions involved (Gotts et al., 2012).

One recent rendition of the social brain in adults that builds on earlier work from neuropsychological/lesion studies and neuroimaging (e.g., Adolphs, 1999; Brothers, 1990), is provided in Kennedy and Adolphs (2012) review of the field, shown in Figure 1.
Such a figure can only be a rough approximation. Nevertheless, the following regions have come up repeatedly in many different studies of social cognition: medial prefrontal cortex, superior temporal sulcus, temporoparietal junction, temporal poles, amygdala and insula. Kennedy and Adolph suggest four major social processing networks identified by function/anatomy across these regions, which they label the Amygdala, Mentalizing, Empathy and Mirror networks. They are listed below, as we will refer to them throughout this review. It is striking that these networks appear to be separable, in terms of neurological underpinnings, from general-purpose adaptive functions.

**Amygdala network**: amygdala and orbitofrontal regions involved in threat detection, emotional evaluation and emotion regulation.

**Mentalizing network**: medial prefrontal and superior temporal regions implicated in the automatic attribution of mental states.

**Empathy network**: insula and amygdala regions involved in automatically detecting and responding emotionally to others’ distress.

**Mirror network**: parietal and prefrontal regions containing neurons that are responsive both to observed and executed actions.

This may be a workable summary of the adult social brain based on current knowledge, but how does the infant brain arrive at this point? We do not know, although some authors have begun to sketch interesting accounts. Johnson et al. (2005; Johnson 2011a,b) have suggested that the adult ‘modular’ brain architecture is achieved neither by maturation...
alone, nor by learning alone, but by a middle way, termed 'interactive specialization', leading to increasingly specific and focal brain activity. This may parallel the shorter-term brain changes that are associated with development of expertise: large regions of (especially frontal) cortex being employed at early learning stages, with more focal (often more posterior) regions being associated with proficient performance (Sakai, Ramnani & Passingham, 2002). It is tempting to relate this to cognitive-level descriptions of development of expertise, in terms of changes from reliance on multipurpose novel problem solving to use of established routines and stored knowledge.

A pattern of increasingly specific and focal brain activity with age has been reported for several areas of social cognition (e.g., Gweon, Dodell-Feder, Bedny & Saxe, 2012). However, developmental changes in brain activity do not always follow this pattern (for discussion see Poldrack, 2010). In some cases the opposite, that is, increases in area of brain activity with age has been found (Golarai et al., 2007). Another view suggests that the adult brain is marked by more efficient connectivity, based on both growing and pruning of synapses, and this may be a mark of any developing mammalian brain (Workman, Charvet, Clancy, Darlington & Finlay, 2013). In line with this proposal changes in functional connectivity in specific brain networks have been demonstrated in children undergoing puberty (Klapwijk et al., 2013).

The evolutionary history of the human brain suggests that social mechanisms are very ancient and shared with other mammals. This supports the notion that innate predispositions, as start-up kits for learning, play a critical role in the development of the social brain. If the putative networks of the social brain are in essence already mapped out at a very early stage, then this would provide a general explanation for the specificity of developmental disorders, where typically not all cognitive functions are equally vulnerable to neuro-genetic faults. This is not to deny the massive contribution of learning to development (e.g., Karmiloff-Smith, 2010). Indeed, learning from others (both near and distant, as in cultural traditions) is a major task of the developing social brain (e.g., Leppänen & Nelson, 2009).

We know much more about the development of social behaviour than we know about underlying brain processes, and this is apparent in the following overview where we summarize what we know of social development in terms of a broad chronology based on typical milestones. The ages mentioned are, of course, approximate in most cases and of little real importance. Individual and cultural differences may apply. Clearly, many social abilities emerge early in some form but continue to show changes and developments across childhood. To minimize repetition, we will adopt a strategy of discussing major aspects of social cognition under the age category where most empirical work has been conducted. We will mention studies of brain function as relevant to the social abilities in question, but with the proviso that these are usually based on studies with adults.

A chronology of social development. The newborn

Here, we highlight the neonate’s extraordinary capacity for the detection of social agents (associated with superior temporal sulcus), mimicry (associated with prefrontal and parietal regions) and mutual affiliation (associated with the oxytocin transmitter system, see below). We are impressed with a nativist account of these abilities, not because they are present from birth, but because they are seen across many species. Thus, they may have ancient evolutionary roots and are likely to be part of the large set of instincts, that is, genetically endowed species-typical behavioural programmes, which do not require trial and error learning. Such abilities can appear at different stages of development, even beyond adolescence. We acknowledge that instincts do not arise in a vacuum, likely require triggering stimuli and calibration, and can potentially be overridden by top-down control processes. We also acknowledge that there is an alternative non-nativist tradition that posits a strong learning-based account for any higher level cognitive ability including social abilities.

There is evidence that neonates come equipped with a preference for patterns in the world that will direct them to social entities. Orientation to the constellation of three dots in a face-like array appears to be present at birth in human and nonhuman species (see Johnson, 2011a,b; Johnson et al., 2005). This preference has been demonstrated robustly in naturalistic tracking studies in human newborns and in chicks. Newborns also show sensitivity to eye gaze, looking longer at faces with open than closed eyes, and direct versus averted gaze (for a review, see Itier & Batty, 2009).

The preference for face-like stimuli is striking because a learning account for visual stimuli prebirth does not work. By contrast, newborns’ preference for their mother’s voice appears to reflect experience of sound in utero, as tested for example with newborn infants of mono- versus bi-lingual mothers (Byers-Heinlein, Burns & Werker, 2010). In adults, human voices are processed in highly specialized auditory brain regions located in anterior superior temporal sulcus. Remarkably, these regions are already active in the first few months of life (Grossmann & Friederici, 2012). Blasi et al. (2011) using fMRI with 3- to 7-month-olds report greater activation of anterior temporal cortex to voices versus nonvoice sounds, and differential activation of orbitofrontal cortex and insula to sad versus happy/neutral vocalizations. We do not know exactly how early in life this differential brain activation is present, as studies are not available to answer this question.
Face processing and, to a slightly lesser extent, voice processing, have been extensively studied in infants, particularly through electrophysiological methods (see review by Righi & Nelson, 2012). According to Grossmann and Johnson (2007), existing neuroimaging and electrophysiological data suggest that most of the brain areas or mechanisms involved in face processing in adults can be activated in the first few months of life, although the infant system may show broader response properties, not being as finely tuned as the adult system.

Preferential attention to biological motion – typically tested with point-light displays – has also been demonstrated in newborns (Simion, Regolin & Bulf, 2008). A specialized system may underlie processing of biological motion, which is robust in the face of degraded input and impairment in processing other types of motion (Jordan, Reiss, Hoffmann & Landau, 2002). The superior temporal sulcus is the area typically activated during perception of biological motion, showing progressive tuning to this stimulus class over development (Lichtensteiger, Loenneke, Bucher, Martin & Klaver, 2008).

Beyond orienting responses, neonates can also copy social models. For example, the newborn shows raised likelihood of tongue protrusion when seeing an adult doing this, and of mouth opening to an adult opening his mouth (for review and discussion see, for example, Suddendorf, Oostenbroek, Nielsen & Slaughter, 2013). The interpretation of this infant response has been debated, with alternative explanations proposed. Ray and Heyes (2011), for example, review the literature on imitation up to age 2 and come down in favour of the view that infants’ interactions with the world drive the cognitive development underlying imitation, specifically through associative sequence learning, rather than explanations in terms of complex innate mechanisms postulated to be supported by the Mirror network (prefrontal and parietal regions; e.g., Marshall & Meltzoff, 2011).

The neonate is part of a social and reciprocal process of affiliation and relationship-formation with the mother or other caregivers – a process in which neurochemicals including oxytocin and vasopressin appear to play an important role (Carter, Williams, Witt & Insel, 1992). The interaction of mother and baby is not merely marked by contingency but by mutuality – that is the strong feedback between emotionally rewarding signals between two social agents. The attachment between mother and child has often been posited as a major source of later social-emotional development. We will discuss this topic under the section on 6–18 months, since much of the research relevant to atypical development has been performed within this age bracket.

Atypical development. Premature birth: Premature birth (from approximately 24–37 weeks gestation, with low birth weight by definition) challenges the developing brain at a stage when cortical expansion and gyrification is at its peak. Studies of the ‘connectome’ suggest significant lack of structural and especially functional connections in the brains of premature infants, at term and beyond (Ball et al., 2013). In particular, connections between the thalamus and the frontal cortices, supplementary motor areas, occipital lobe and temporal gyri are reduced in preterm infants. Children born premature often suffer from additional perinatal hazards and have a threefold likelihood of a psychiatric diagnosis at 11 years, compared with their full-term peers (Johnson et al., 2010). As well as intellectual disability (with a particular academic deficit in maths), Attention Deficit/Hyperactivity Disorder (ADHD) (primarily of the inattentive type), and anxiety/depression (which may be mediated by victimization), problems of social and communication development are common (e.g., social initiation and joint attention; Landry, Smith, Miller-Loncar & Swank, 1997), with around 5% meeting diagnostic criteria for autism (Johnson et al., 2010). De Schuymer, De Groote, Desoete and Roevers (2012) recently reported gaze aversion in preterm infants, which appears to be part of more general problems in visual attention. Socio-emotional difficulties in this population at school-age have been related to regional cerebral development at term (Rogers et al., 2012). The wide-ranging effects of prematurity are perhaps not surprising if premature birth disrupts connectivity, and/or limits the ability to compensate for any additional difficulty (e.g., due to reduced executive control).

The first 3 months

Here, we highlight developments in social reciprocity related to both contingency recognition and increasing interaction.

A major cue to the presence of a social agent is whether its action is contingent on our own action. Over the first 3 months of life, typically developing infants show the unfolding of a rich repertoire of social reciprocity, or mutual contingency. From around 4 weeks, infants show sensitivity to the contingencies of close one-to-one interaction as manifest in the classic ‘still face’ paradigm (see Walle & Campos, 2012 for a recent review). In this paradigm, the adult interacts (e.g., via video) with the infant in a naturalistic manner, and then freezes and ceases to respond for a set period. Infants show distress to this interruption and may attempt to re-engage the adult, suggesting sensitivity to interaction contingencies. There are of course individual differences in the precise responses, and it has been suggested that these predict, for example, attachment security at 12 months. Feldman (2012) has reviewed typical parent-infant synchrony (both behavioural and physiological) in the first 3 months and possible later sequelae. The emergence of the
interactive social smile at around 6–8 weeks after full gestation, appears to be robust to differences of environment (e.g., visual impairment, cultural differences; for review see Messinger & Fogel, 2007). The development of the infant as a social agent is also facilitated by the emergence of different cry types reflecting different internal states and prompting different responses from caregivers. Reddy, Markova & Wallot (2013) discuss infants’ ability to engage with others’ intentions in interaction. For example, infants as young as 3–4 months may tense their body and arch their back in preparation for being picked up, which may be interpreted as cooperating with a perceived parental intention.

**Atypical development.** One challenge in analysing atypicality in developmentally disordered groups is distinguishing mere delays from distinct difficulties. Only hindsight can tell if it matters whether critical social behaviours do not emerge at the expected time. Some conditions can be recognized early, and here it is possible, in theory, to gauge the extent to which social processes either lag behind or are missing. **Down Syndrome,** which is typically identifiable from birth, attracted a great deal of research attention in the 1970s and 1980s, with a primary focus on development in the first months and years of life. Indeed, in their review of this work, Cebula and Wishart (2008) highlight the need for research on social cognition in later childhood and adulthood. The early studies, and some more recent research, suggest that many aspects of social cognition are in line with developmental level in Down Syndrome. For example, sensitivity to disruption of mother’s interaction during the ‘still face’ paradigm appears broadly in line with developmental level (Moore, Goodwin & Oates, 2008). While there are developmental delays in looking at a caregiver’s face and making eye contact in the first months, prolonged looking has been reported by the middle of the first year, when looking has begun to decrease in typically developing infants. Greater looking at people seems to persist into the early years, with some corresponding difficulty in alternating gaze between person and object, and knock-on effects on joint attention (in some contexts). At least some studies have suggested that imitation skills are relatively good in Down Syndrome, in line with Langdon Down’s own observations. Evidence regarding emotion recognition is somewhat mixed, with some apparent deficits disappearing with methodological improvements. Since there is some evidence that the temporal limbic system is disproportionately reduced in volume and complexity in Down Syndrome, further investigation into emotion processing across development would seem warranted.

Disorders such as **Angelman, Cornelia de Lange and Cri-du-chat-syndrome,** are now detectable with genetic tests very early in life. Children with these conditions show social impairments, and these are beginning to be studied in detail to differentiate them from each other and from autistic disorders (Moss et al., 2013). These studies may in the future provide links between specific social impairment and specific genetic perturbations.

By contrast with these types of genetic/medical conditions diagnosable at birth, conditions with a purely behavioural diagnosis typically lack good information about the very first months of life. For many years, often based on retrospective parental report, it was believed that children with **autism spectrum disorder (ASD)** were noticeably different in their social orienting and reciprocity (e.g., social smile, attachment) from birth. However, over the last decade, the study of genetically at-risk infants (the siblings of children with ASD), has not confirmed this; indeed, in social behaviour there appears to be nothing in the first 3 or even 6 months that indicates a future ASD diagnosis (Ozonoff et al., 2010). While electrophysiological markers (e.g., ERP, EEG) show some abnormalities from the second half of the first year of life, the specificity of these for later ASD is unclear; comparison clinical groups are needed.

Nevertheless, we note that some behaviours already evident in typical development in the first months have been found to be affected in older individuals with ASD. Biological motion perception appears to be impaired in ASD; with, for example, reduced attention to biological motion in 3- to 7-year-olds with ASD (Annaz, Campbell, Coleman, Milne & Swettenham, 2012), and reduced sensitivity to biological motion even when this is not confounded with global form perception (Cook, Saygin, Swain & Blakemore, 2009). Reduced activation of superior temporal sulcus while watching point light biological motion stimuli has also been reported in older individuals with ASD (e.g., Kaiser et al., 2010). Face processing has been extensively investigated at older ages in ASD, with debate concerning the atypicality or otherwise of associated neural activity (in fusiform face area); this literature is beyond the scope of the current paper and the reader is instead referred to recent reviews (e.g., Campatelli, Federico, Apicella, Sicca & Muratori, 2013).

**3–6 months**

Here, we highlight early capacities of emotion processing, and sensitivity to ostensive signals, chiefly through gaze processing. Although information on neural substrates is lacking, Amygdala and Empathy networks (see Figure 1) may be implicated in these developments.

Walle and Campos (2012) provide a review of the development of children’s response to emotions; for example, habituation paradigms suggest infants as young as 4 months can discriminate two emotional displays of opposite valence and even different negative emotions, provided facial and vocal information is given. However, the full range of differen-
tial behavioural affective responses may not be manifested until later in development. At this early age children are already sensitive to ostension – behaviours intended to get the other’s attention, usually for the purpose of communication. For example, 4-month-olds prefer to listen to their own rather than other names (Mandel, Jusczyk & Pisoni, 1995). Adult fMRI studies have suggested activation in medial prefrontal cortex and right inferior frontal gyrus to own name versus familiar or personally salient names – and the medial prefrontal cortex has been linked to personally relevant information more generally (e.g., Tacikowski, Brechmann & Nowicka, 2013). Ostensive stimuli appear to activate brain regions involved in the Mentalizing network (Kampe, Frith & Frith, 2003).

Direct gaze is a powerful ostensive signal (for review of development of response to eye gaze see Itier & Batty, 2009). By 4 months, ERP studies show enhanced processing of faces with direct versus averted gaze (marked by increased amplitude of the N290). At the same age, infants’ object processing is facilitated by adult gaze, as reflected in activation of similar but less specialized cortical circuits in infants and adults (Senju, Csibra & Johnson, 2008). Three-month-olds have been reported to smile more when an adult alternates visual attention between the infant and an object – perhaps marking a precursor of joint attention (Striano & Stahl, 2005). A meta-analysis of almost 60 neuroimaging studies exploring eye movements, shifts of attention and gaze perception, suggested the involvement of the amygdala, the superior temporal gyrus/sulcus, some ventro-temporal regions involved in face recognition (fusiform face area,), as well as a frontoparietal circuit for gaze (Grosbras, Laird & Paus, 2005).

Atypical development. The literature on social cognition in Williams Syndrome (WS) has been comprehensively summarized by Järvinen-Pasley et al. (2008) and more recently by Tager-Flusberg and Skwerer (2011). These reviews provide evidence of ‘hypersociability’ in WS combined with puzzling social impairments. Thus, infants and toddlers with WS have been observed, in case and group studies, to look longer and more intently at adults’ faces during naturalistic interactions. However, the overwhelming tendency to look at faces is accompanied by a tendency to ignore toys that another person may wish to introduce to the child, giving rise to delayed joint attention. Furthermore, young WS children spend more time gazing at unfamiliar than familiar faces and later in life show indiscriminate approach to strangers and remarkably little social anxiety. Poor ability to recognize facial affect has also been identified, and this highlights the specificity of brain networks underlying face perception (fusiform face area, FFA) and emotion perception in facial expressions (amygdala, insula, depending on the emotion expressed).

Moebius Syndrome presents an interesting case where facial expression is specifically compromised from birth due to bilateral facial nerve paralysis. Despite this, recognition of emotional expressions is reported to be normal, as are other aspects of social cognition (Bogart & Matsumoto, 2010). This suggests that intact facial expression is not vital for intact emotion recognition, perhaps arguing against a crucial role for, for example, mirror neurons in the development of emotion recognition. It will be important to establish whether empathic and other social cognitive processes are also intact in Moebius Syndrome.

Developmental prosopagnosia, a condition in which face recognition fails due to brain pathology with a genetic origin, appears to leave other aspects of social cognition unaffected (Duchaine, Murray, Turner, White & Garrido, 2009). Presumably there are many different types of information feeding into the recognition of individuals, so that failure in one modality is not disastrous. It is important to bear this in mind when trying to trace, for instance, the social impairments in ASD to any narrow deficit in social perception. Weigelt, Koldewyn and Kanwisher (2012) reviewed behavioural studies of face recognition in ASD and concluded that there is some impairment in facial processing, especially when memory is involved. Campatelli et al. (2013) provide a comprehensive review of face processing in ASD, including the mixed and complex findings from fMRI and ERP methodologies.

It has been suggested that emotion reading might be the primary deficit in ASD (Hobson, 1986). However, reviews and meta-analyses have suggested that any emotion recognition difficulties become modest when groups are well-matched for verbal ability and publication bias is taken into account (for a review see, e.g., Jones et al., 2011). A few studies of children later diagnosed with ASD suggest reduced attention to social scenes at 6 months (e.g., Chawarska, Macari & Shic, 2013) but, in general, social deficits this early in infancy have been difficult to demonstrate (Ozonoff et al., 2010).

Older individuals with Turner Syndrome (Elgar, Campbell & Skuse, 2002), as well as those with Fragile X Syndrome (Garrett, Menon, MacKenzie & Reiss, 2004) or Fetal Alcohol Syndrome (Bishop, Gahagan & Lord, 2007), have been reported to show impairments in processing direct gaze. Clearly, gaze processing can be impaired for a variety of reasons, and these may differ across these groups (e.g., putatively, social anxiety in Fragile-X but not Fetal Alcohol Syndrome). In Turner Syndrome, a genetic disorder affecting approximately 1 in 2000 women, in which there is complete or partial absence of an X chromosome, problems processing emotional expressions have been reported. Particular difficulties with negative emotions have been reported, but it is important to note that studies do not always account for the differential discriminability of
negative versus positive emotional displays. There is also some (albeit inconsistent) evidence of enlarged amygdala volume in women with Turner Syndrome, and significantly reduced volume of structures heavily connected with the amygdala, such as the right hippocampus, orbitofrontal cortex and superior temporal sulcus (Burnett, Reutens & Wood, 2010; Hong, Dunkin & Reiss, 2011). It remains to be established whether there are distinct neural and cognitive bases for the difficulties with gaze or emotion processing in these different clinical groups.

6–18 months

Social behaviour in this period has been intensely studied in recent times, using ingenious experimental techniques that have revealed many previously unsuspected abilities. Here, we review the development of a hugely important group of social capacities that are all characterized by strong reciprocity and sharing of mental states with a social partner: joint attention, social referencing, attachment and implicit mental state attribution (Mentalizing network in Figure 1). Studies at the end of this period also provide evidence for another, possibly separate, strand of social cognition that relates to self-awareness and identification, mirror recognition and in-group/out-group distinctions.

Joint attention begins to be evident early in this period. While gaze-following to visible targets may be seen around 6 months, tracking of attention from eyes alone continues to develop and may not be fully reliable until the second year. Infants begin to direct others’ attention using pointing from around 9–12 months (see Tomasello, Carpenter & Liszkowski, 2007; for a review). Little is known about the brain basis of joint attention before about 9 months, but studies with adults suggest the involvement of the dorsal part of the medial prefrontal cortex and posterior superior temporal sulcus (Frith & Frith, 2006; Redcay, Kleiner & Saxe, 2012); important parts of the Mentalizing network (Figure 1). Elison et al. (2013) recently reported that 6-month-olds’ fractional anisotropy in the right uncinate fasciculus, a white matter fibre bundle connecting the amygdala to the ventral-medial prefrontal cortex and anterior temporal pole, predicts individual differences in responding to joint attention at 9 months. Infants at 14 months have been reported to show more EEG coherence during joint attention.

Advances in joint attention during this period of development come together with increasing discrimination of emotional displays (involving the Amygdala network in Figure 1). For example, electrophysiological studies suggest that at 7 months, but not earlier, infants show relatively enhanced visual and attention-sensitive event-related potentials, as well as enhanced cardiac orientation response and increased attention-dwell time, to fearful compared with neutral and happy expressions (for review see Leppännen, 2011; Leppänen & Nelson, 2009).

Together, these emotion processing and joint attention capabilities allow ‘social referencing’ (Camras & Shutter, 2010). By 12 months, infants can use an adult’s emotional reaction to a novel object to inform their own response to the referent; for example, avoiding an object to which their mother expresses fear (Vaish & Striano, 2004). However, it may not be until considerably later that infants can understand the emotion as an internal state that helps predict future intentional (e.g., object-directed) actions (Gergely, Egyed & Kiraly, 2007).

Many studies of attachment have been conducted during this developmental period (for review, see Zeanah, Berlin & Boris, 2011). Around 7–9 months, selective attachment becomes evident in the onset of stranger wariness and distress at separation from parents/carers. By 9–18 months infants show a hierarchy of attachment figures. With increasing mobility from around 12 months, the balancing of ‘secure base’ (exploratory) and ‘safe haven’ (returning to or checking in with the caregiver) behaviours becomes especially important. From this age, Ainsworth’s ‘Strange Situation’ is often used to assess attachment security. Minagawa-Kawai et al. (2009) explored the neural substrates underlying social and emotional attachment, measuring mothers’ and infants’ prefrontal activation using near-infrared spectroscopy. They report increased activations in anterior orbitofrontal cortex in mothers viewing their own infant, and infants viewing their mother’s smile.

Reddy (1991) has highlighted naturally occurring ‘teasing’, a contingent and affiliative behaviour that begins around 9–12 months, and includes, for example, showing off, playfully blocking the parent, teasingly offering and withdrawing an object, and simple tricking. She suggests that these early infant behaviours involve engaging with the adult’s mental states, such as expectation and intention.

While success on explicit ‘theory of mind’ or mentalizing tasks, such as the classic false belief paradigm, comes in the third or fourth year, recent work has suggested some ability to track mistaken beliefs as young as 7 months, from gaze behaviours and habituation methods. This work, reviewed by Baillargeon, Scott and He (2010), reveals the automatic tendency to take account of another person’s mental states. In fact, the paradigms used show that it need not be another person, but can be a cartoon character, or even a faceless brush that moves contingently. A number of studies now demonstrate that babies from the second half of the first year display this ability if measured by anticipatory eye gaze or prolonged gaze in scenarios that violate the expectation built on another creature’s inner state. Kovacs, Teglas and Endress (2010) have shown that adults too show evidence of automatic responding that exactly parallels infants’ performance, using reaction times for adults where looking time is used...
for infants. This perhaps suggests that the automatic mentalizing system does not become obsolete once flexible and explicit mentalizing is acquired (by about 4 years in neurotypically developing children; see review by Wellman, Cross & Watson, 2001). Yamaguchi, Kuhlmeier, Wynne and vanMarle (2009) have reported that looking time measures at 12 months correlate with behavioural tests of theory of mind at 4 years.

A rare longitudinal study of infant neural processes related to distinct aspects of prosocial behaviour was conducted by Paulus, Kühn-Popp, Licitra, Sodian, Meinhardt (2012). Resting state brain activation asymmetries on electroencephalogram (EEG) at 14 months were related to two types of prosocial behaviour tested 4 or 10 months later: greater left frontal activation was associated with comforting behaviour (tested at 24 months), while greater right temporal activation was associated with instrumental helping behaviour (tested at 18 months). If replicated, these findings suggest that it might be possible to show an early differentiation of Empathy and Mentalizing networks, each associated with different aspects of social reciprocity.

Atypical development. At risk infant-sib studies suggest that it is around 12 months that many children later diagnosed with ASD begin to show behavioural signs of social impairment. These include reduced imitation, lessened response to name, lower social interest and social smiling, and atypical eye contact (Zwaigenbaum et al., 2005). However, none of these deficits is universal. It is notable that, for example, no major differences were found in ERP responses to faces or biological motion up to and at 12 months in siblings at-risk for ASD (Luyster, Wagner, Vogel-Farley, Tager-Flusberg & Nelson, 2011; for a recent study using functional near infra-red spectroscopy, see Fox, Wagner, Shrock, Tager-Flusberg & Nelson, 2013). While some ERP differences towards the end of the first year have been replicated in at-risk sibs, these have emerged in nonsocial (e.g., attentional) paradigms and surprisingly little support has been found for intuitively plausible accounts of ASD in which social orienting is the primary specific deficit. The literature on imitation in infants and children with ASD is reviewed by Vanvuchelen, Roevers and De Weerdt (2011), who conclude that imitation deficits may not be as robust or universal a part of the phenotype as may have been thought.

The long-term follow-up of children adopted from the severe social deprivation of Romanian orphans under Ceausescu, suggests that lack of any proper parental-like care leave surprisingly little trace if restricted to the first 6 months of life (see, e.g., Rutter, Kumsta, Schlotz & Sonuga-Barke, 2012). This may call in to question at least some accounts of social development, in which the establishment of attachment in the very earliest months is fundamental to the unfolding of good relationships in later life. On the other hand, length of institutionalization beyond this time has been reported to predict attachment status and later internalizing and externalizing disorders (O’Connor & Rutter, 2000). Timing of foster care for children in the Bucharest Early Intervention Project has also been found to relate to EEG measures, which have in turn been linked to ADHD in this sample of institutionalized children (Vanderwert, Marshall, Nelson, Zeannah & Fox, 2010). A meta-analysis by Colonnesi et al. (2011) of 46 studies in typically developing populations also suggests that insecure attachment (typically assessed at or after 12 months) is moderately related to anxiety, measured as a disorder or trait, in later childhood and adolescence. Finally, in relation to ASD, it has long been known that attachment as assessed in standard paradigms is in line with developmental level (Rutgers, Bakersmans-Kranenburg, Van Ijzendoorn & van Berckelaer-Onnes, 2004).

18–36 months

Here, we briefly review the leap in language acquisition, pretend play and emotion recognition that occurs during the early childhood period. Children at this age show an abundance of spontaneous cooperative behaviour and social learning via ostensive signals (natural pedagogy). These social accomplishments are likely facilitated by implicit mental state attribution (see Mentalizing network, Figure 1).

After 18 months of age big changes occur in development, marked by leaving behind the label ‘infant’. Children’s physical body shape changes, so that their proportions are more like those of older children. Their motor development likewise makes big strides in allowing secure walking and running, with fine motor development seen in more advanced play and manipulation of objects. In terms of social development, 2-year-olds have a lot to learn. They seek the company of other children but engage in parallel rather than interactive play. Compared with the first year, they show increasing independence and wilfulness. They copy others freely and show frustration when thwarted in their attempts to emulate others’ more skilled behaviour.

During this time there is a huge leap in language acquisition, at the same time as pretend play spontaneously emerges (Leslie, 1987). This timing does not appear to be coincidental. The leap in language acquisition is thought to occur through exactly the same mechanism as the understanding of make belief play: ostension and mental state attribution. An act of ostension, such as pointing and naming, demonstrates an intention to communicate something. Bloom (2000) has reviewed the literature on the key importance of recognizing speaker’s intentions in word learning. For example, Baldwin and colleagues showed that infants learn
words from adults only if they see that the adult is deliberately addressing them (vs., e.g., being overheard on the phone; work reviewed in Sage & Baldwin, 2010). Csibra and Gergely (2009) showed that children in this age group can learn the meaning of new words after just one trial in such circumstances—supporting their notion of ‘innate pedagogy’.

Emotion processing and labelling is a vital skill that continues to develop at this stage (Hariri, Bookheimer & Mazziotta, 2000), for both basic and social emotions. For instance, jealousy, often directed at a younger sibling, is part of the growing repertoire of emotional expressions. Jealousy can be measured in infants and emerges most intensely between 11 months and around 2 years in situations designed to provoke this emotion (Masciuch & Kienapple, 1993). At the end of the second year, children are protective of their own possessions and find it hard to share with others. Children from the middle of the second year show empathy by responding to others’ distress, and appear to understand the difference between a partner who is unwilling versus unable to cooperate (see Empathy network in Figure 1).

Imitation and cooperation are facilitators of social learning and have been studied extensively during this period. Evidence of deliberate imitation can be found as early as 6–18 months (see review by Jones, 2009), but deferred imitation of a model shown on TV a day later is significantly stronger at age 18 months than at earlier ages (Barr, Muentener & Garcia, 2007). The longitudinal links between imitation, pretend play and mirror self-recognition have been explored by Nielsen and Dissanayake (2004), who examined these behaviours in 98 infants seen every 3 months from 12 to 24 months. Deferred but not synchronic imitation appeared to be a prerequisite for later pretend play and self-recognition skills. Automatic copying has been postulated to reflect the activity of mirror neurons (neurons responsive to matching observed and executed actions; Heiser, Iacoboni, Maeda, Marcus & Mazziotta, 2003; Rizzolatti & Craighero, 2004). Deliberate copying, on the other hand, needs to be considered separately, as it is strongly affected by top-down processes; inhibition of copying is often a necessity, and may be more closely linked to perspective taking than imitation per se (Santiesteban, Banissy, Catmum & Bird, 2012; Santiesteban, White, Cook, Gilbert & Heyes, 2012). Over and Carpenter (2013) draw together evidence from apparently contradictory studies of sometimes faithful copying, sometimes highly selective imitation by showing that imitation in childhood is modulated by a range of social, including intergroup, influences.

Around 18–24 months children begin to pass the classic mirror self-recognition, or ‘rouge’, test; touching their own forehead where they see a mark in the mirror. The status of this as a test for self-recognition has been debated (e.g., Ray & Heyes, 2011). However, success at this age in personal pronoun use and simple perspective taking tasks supports the notion that children’s awareness of self is developing importantly at this time. That infants at this age already represent some aspects of self, however implicitly, may also be inferred from studies of the in-group/out-group distinction. Young babies make automatic distinctions between people like themselves/family/neighbours and people who are different. Mahajan and Wynn (2012) suggest that, well before they acquire language, children make this distinction on the basis of appearance, in particular similarity to self, regardless of familiarity. Thus, girls tend to spontaneously copy girls and women, and boys spontaneously copy boys and men. The in-group/out-group distinction is further discussed in the 3–5 year age group where a range of behavioural paradigms have been used.

This age also finds the emergence of spontaneous helping and cooperation. Cooperative behaviour requires joint attention and thrives on the tendency to copy others but, as Warneken, Gräfenhain and Tomasello (2012) showed, it also requires implicit mentalizing. Children aged 21 and 27 months continued to engage the partner even when this was not strictly necessary to reach the goal, demonstrating that the partner was valued not simply as a tool, but (implicitly) as an intentional agent. Spontaneous helping has been reported in toddlers (Buttellmann, Carpenter & Tomasello, 2009), who appear to recognize others’ intentions and restrictions in simple, naturalistic paradigms.

Atypical development. The symptoms of ASD have, according to current diagnostic manuals, to have been present by age 3, and it is very common for parents to become concerned about their child’s communication delay and/or social difficulties towards the end of the second year of life. Notable signs in this period are reduced or absent pretend play (with instead repetitive, often part-focused manipulation of objects, e.g., spinning the wheels of a toy car), joint attention and communication. The common denominator in these cognitive abilities is likely to be implicit mentalizing. The automatic tracking of others’ mental states appears to be disrupted in ASD; for example, unlike typical toddlers, young children with ASD do not show the easy and natural coordination of intentions in a cooperative play paradigm (Liebal, Carpenter & Tomasello, 2008).

Rett Syndrome is a severe X-linked neurodevelopmental disorder, which typically becomes evident after 2 or 3 years of apparently unremarkable development, with profound regression of speech and hand use and progressive physical and intellectual impairment. During the regression phase, social withdrawal is not uncommon. However, social disruption appears to be transient in most cases, and Rett Syndrome is probably not a specific disorder of
social cognition; social skills are not out of line with other adaptive functioning. Recent work using eye tracking with a large group (N 49) of girls and women with Rett Syndrome (aged 1.5–25 years) suggests the typical weighting of attention towards social stimuli—with, for example, more looking at eyes than mouth or nose (Djukic & McDermott, 2012).

3–5 years

Here we highlight the development of explicit mental state attribution (Mentalizing network in Figure 1) and the implications of this capacity for ‘social policing’ (e.g., taking responsibility and justifying behaviour in terms of moral principles and fairness). We revisit the importance of in-group/out-group distinctions and their possible link to changes in self-awareness and identification (Amygdala network in Figure 1). All these functions are, of course, heavily influenced by cultural learning.

By around 4 years typically developing children pass the classic version of false-belief tasks designed to tap theory of mind or mentalizing. As reviewed above, earlier success on infant habituation tasks, toddler anticipatory gaze tasks, and use of mental state language in everyday life, strongly suggest some capacity to track mental states at much younger ages. Whether the delay in passing explicit tests reflects additional task demands (e.g., for executive functions), or the existence of two systems for representing mental states remains hotly debated. Although this has yet to be tested by neuro-imaging methods (see below), it may be that there are two brain systems that serve mentalizing: an early maturing implicit system and a late maturing explicit system (Apperly & Butterfill, 2009; Low & Watts, 2013).

Prosocial behaviour, moderated by the perceived morality of the adult-to-be helped, is strongly evident at this age. For instance, Vaish, Carpenter and Tomasello (2010) found that 3-year-olds were less inclined to help an adult who harmed (or unsuccessfully tried to harm) another adult than a neutral adult. Children’s prosocial behaviour was apparently mediated by the implicit recognition of the adult’s intentions, irrespective of outcome.

Typically developing children are hyper-vigilant to instances of injustice, and around this age children begin to succeed first on third-person tests and eventually on first-person tests of fairness. Recent infant studies have suggested that babies around 20 months expect an experimenter to divide two things equally between two people, but not between two objects, and also expect rewards to be given fairly. While the first glimmers of conscience are considered to emerge in toddlers, by age 3–4 years children show evidence of moral emotions (e.g., guilt), and complying with and internalizing adults’ rules (Kochanska, Gross, Lin & Nichols, 2002).

The preference for in-group versus out-group persists and perhaps intensifies at this age. For example, Kinzler, Corripio and Harris (2011) showed that 4- to 5-year-olds trust speakers who share their native accent over those who have a different accent. Older children and adults can be induced to form such groupings with minimal stimuli (Biglieri & Liben, 2006). They behave prosocially to members of their in-group, and show hostility to members of an out-group. Thus, Schug, Husterman, Barth and Patalano (2013) showed that 5-year-old treated identical observations of generosity and stinginess differently for members of an in-group versus out-group. The in-group/out-group distinction is critical for discriminate social learning since it is essential to align oneself only with trustworthy others (e.g., nearest kin). Comparative studies show that the in-group/out-group distinction is not human specific. This suggests that ancient brain systems are involved in this categorization of social stimuli. The action of oxytocin appears to promote sociality and affiliation within in-group, but at the same time increases hostility to out-group members (Staufen, De Dreu, Shalvi, Smidts & Sanfey, 2012).

The in-group/out-group distinction, selective copying and helping those like oneself, may have a reciprocal relationship with aspects of self-representation and social identity. Around 5 years, a range of tasks tapping social insight related to self-awareness begin to be passed, including recall of own mistaken belief, discrimination of known versus guessed information and reflex versus intended actions (e.g., Williams & Happé, 2010).

Atypical development. The ability to track others’ mental states in standard, explicit false belief tasks presents a major challenge to children with ASD. Happé’s (1995) secondary analysis of data from ASD and neurotypical participants across a wide range of verbal mental ages suggested that children with ASD pass false belief tasks around 5 years later (in terms of verbal mental age) – perhaps suggesting compensatory learning. Nevertheless, in adulthood many ASD individuals no longer fail these explicit tests. In a behavioural study, Senju, Southgate, White and Frith (2009) have shown that ASD adults who show excellent performance on a large battery of explicit theory of mind tasks, nevertheless fail to show automatic anticipatory eye gaze based on tracking false belief in a simple nonverbal task presented on video. It is tempting to conclude that the implicit mentalizing system remains faulty in ASD. On the existing evidence from a wide range of tasks, individuals with ASD (mostly adults) also show atypical activation in the Mentalizing network (see Figure 1). Marsh and Hamilton (2011) used an ingenious reaching task to separate activation of the Mirror neuron and Mentalizing systems. As in previous studies, the ASD group showed atypicalities in the Mentalizing system (cingulate, fusiform and medial...
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prefrontal regions). In contrast, there were no atypical activations in parietal mirror neuron regions.

One might have expected individuals with ASD to show activity in atypical brain regions when they perform mentalizing tasks in the scanner. This is not the case. Instead patterns of over- or under-activation in the Mentalizing network are found. In addition, differences in functional connectivity between components of the network are consistently reported. These findings apply to both adults (e.g., Castelli, Frith, Happe Frith, 2002) and children with ASD (Carter, Williams, Minshew & Lehman, 2012; Wang, Lee, Sigman & Dapretto, 2007). Thus, atypical activation of the Mentalizing system in ASD groups appears to be not only a highly replicable, but also a sensitive measure of the clinically assessed difficulty with on-line attribution of mental states in everyday life. These difficulties are often hard to capture in behavioural tests of mental state attribution under laboratory conditions, especially in high-functioning adults with ASD.

While most studies of mentalizing in ASD address attribution of mental states to others, rather few have investigated the ability to reflect on one’s own mental states (e.g., a prior false belief). ‘Reading one’s own mind’ also appears to be challenging for many individuals with ASD (e.g., Williams & Happé, 2010). Neuroimaging studies interrogating self-processing in ASD (Lombardo, Barnes, Wheelwright & Baron-Cohen, 2007; Lombardo et al., 2010) also show atypical responses in the Mentalizing network.

Fragile X syndrome (FXS) is a monogenic X-linked disorder, the most common inherited cause of intellectual disability, and the most common genetic condition linked to ASD. Individuals with FXS are reported to show social difficulties and characteristic social anxiety and gaze avoidance. A recent study by Losh, Martin, Klusek, Hogan–Brown and Sideris (2012) suggests that only individuals with FXS plus ASD show deficits in theory of mind and pragmatics (as in ASD without FXS); thus FXS alone does not appear to compromise these aspects of social cognition.

It remains to be tested whether different neural processes underlie different types of in-group/out-group stereotyping; Santos, Meyer-Lindenberg and Deruelle (2010) report reduced racial but unaffected gender stereotyping in children to Williams Syndrome, who are also reported to have reduced fear linked to social threat, related with diminished amygdala reactivity and prefrontal regulation (Amygdala network in Figure 1). There is suggestive evidence that in adults with Williams syndrome the capacity for mentalizing is spared, and this may yet prove to be the case also for adults with Prader-Willi Syndrome, who show a general rather than a specific mentalizing impairment (Tager-Flusberg, Boshears & Baron-Cohen, 1998).

Studies of the social phenotype of Williams syndrome (see reviews by Järvinen-Pasley et al., 2008; Tager-Flusberg & Skwerer, 2011) strongly support a dissociation between different social abilities in this syndrome. For example, two studies have observed empathic responding during a simulated distress paradigm, and find greater expressions of empathy (e.g., comforting behaviour) by children with Williams Syndrome (from as young as 4 years) compared with controls (including those with Prader-Willi Syndrome). As Williams Syndrome children give indiscriminate attention to familiar and unfamiliar others, it would be interesting to see whether this response is shown as much to members of an ingroup as to an outgroup. If so, this would be an atypical response, since adults at least, show empathic responses to members of their ingroup only (see Gutsell & Inzlicht, 2012; for an EEG study using alpha oscillations, that might be applied to children).

Middle childhood

Here, we consider the extended development of mental state attribution, emotion processing and related development in moral judgement (implicating all four major networks in Figure 1). Explicit teaching likely amplifies the effects of learning, and cultural differences might be expected to be manifest in behaviour as well as brain function.

During middle childhood, attribution of mental states develops further with experience of a range of social situations. Around 6 or 7 years children pass explicit tests of second-order theory of mind, tracking for example that John doesn’t know that Mary knows the train has been rescheduled (see Miller, 2009 for a review). This ability underpins important everyday social skills, such as distinguishing whether a speaker is lying, joking or being ironic (critically distinguished by the speaker’s intent regarding the listener’s belief).

So far, most neuroimaging of mentalizing has been performed in adolescents and adults, however, recent work by Saxe and colleagues suggests that the same brain regions (temporo-parietal junctions; precuneus; medial prefrontal cortex) are active when children (aged 5–11) and adults engage in tasks requiring attribution of mental states. Age-related changes are seen in specificity of neural response, to children increasingly showing selective activation of temporo-parietal junction in response to mentalizing tasks only (Gweon et al., 2012). The authors note that degree of selectivity in this region (right temporo-parietal junction) correlated with ability to provide explanations in a theory of mind test, although other relevant functions such as inhibitory control were not tested. Executive function and IQ were controlled in a study by Sabbagh, Bowman, Evrare and Ito (2009), who used EEG to measure neural changes correlated with mentalizing task performance in 4-year-olds. Amplitude and coherence of resting alpha waves (considered to reflect functional maturation) in dorsal medial prefrontal...
cortex and right temporo-parietal junction correlated with false belief test performance.

Emotion recognition and labelling develop further at this time. Self-conscious and social emotions such as guilt, embarrassment and shame begin to be understood and recognized. Surprisingly, perhaps, situational (cause and consequence) information seems to be more helpful for emotion discrimination/attribution than facial expression to children in this age range (Widen & Russell, 2010). Decety and Svetlova (2012) review the literature on emotion processing in relation to empathy, including their own cross-sectional studies of response to observed intentional or accidental pain-causing, with participants aged 7–40 years, suggesting that in response to others’ physical distress, younger participants recruited the posterior portion of the insula plus the amygdala and medial orbitofrontal cortex more than adults – perhaps suggesting more direct and visceral perception of others’ distress at younger ages. The anterior cingulate cortex and anterior insular cortex are implicated in adult neuroimaging studies of perception/imagination of others’ pain (e.g., Bird et al., 2010).

During this period, children’s moral judgements begin to take into account not only outcome but intent, a change that is hypothesized to involve a conceptual reorganization (Cushman, Shekoff, Wharton & Carey, 2013), aided by cultural context, as reflected in explicit teaching. Judging intention clearly involves elements of the cognitive and neural system for mentalizing, but moral development also depends upon emotional responses. Reflecting these two components, neuroimaging and lesion studies (reviewed by Moll et al., 2005) suggest moral cognition involves the ventromedial prefrontal cortex, medial prefrontal cortex, anterior cingulate cortex, insula, amygdala and posterior superior temporal sulcus. Decety, Michalska and Kinzler (2012) report decreases in amygdala and insula activity, which they relate to emotional saliency, and increases in medial and ventral PFC activity, related to decision making/evaluation, in adults compared with children watching and making moral judgements about short videos of actions. These results can be seen as broadly complementary to the cognitive work suggesting that judgement of intention becomes more important, and reaction to outcome less overwhelming, with age.

Atypical development. With the increasing sophistication of emotional experience in middle childhood, a range of conditions putatively rooted in abnormal emotional processing become evident.

Alexithymia refers to deficits in reflecting on, discriminating and labelling one’s own emotions. This trait, which occurs at elevated rates in a range of disorders (including eating disorders, anxiety and ASD), has mainly been studied in adults. However, self-report questionnaires for alexithymia appear to be valid in adolescents, and recently an informant-report measure has been validated for children from 5 years (Riefke, Oosterveld & Meerum Terwogt, 2006). The study of alexithymia in children is likely to be important for understanding social deficits in several diagnoses; Bird et al. (2010; Bird and Cook 2013) have suggested that emotion-recognition and empathy impairments reported in some studies of ASD are best explained by associated alexithymia, found in around half this population. The developmental origins of alexithymia have not been established, but a recent study suggests a link with early emotional neglect in some cases. Aust, Alkan-Härtwig, Heuser and Bajbouj (2013) report a significant positive correlation in adults between self-report questionnaire measures of alexithymia and early emotional neglect (but not to physical or sexual abuse). Within the high alexithymic group, self-reported emotional neglect was related to significantly increased emotional dysfunction even after controlling for alexithymia – perhaps suggesting a differentiation between ‘neglect’ and ‘nonneglect’ subtypes of alexithymia. It should be noted, of course, that the correlation reported need not represent a causal effect of neglect on alexithymia; it is also possible that, for example, parents with alexithymia are less emotionally available to their offspring, who also inherit a genetic predisposition to alexithymia (i.e., passive gene-environment correlation).

Problems in discriminating and/or responding to others’ (and perhaps even own) emotions of distress (fear, sadness) are thought to be fundamental to conduct disorder in those children showing callous/unemotional (CU) traits (identified as psychopathy in adulthood; Crowe & Blair, 2008). Several studies to date report that boys with conduct disorder and CU traits show reduced amygdala reactivity to fear images, when compared with typically developing controls, children with ADHD, or other children with conduct disorder without CU traits (e.g., Jones et al., 2009; Marsh et al., 2008; Viding et al., 2012). Schwencck et al. (2012; in line with Jones, Happé, Gilbert, Burnett and Viding 2010) explored cognitive versus emotional empathic skills in boys with ASD, typical development or conduct disorder with or without CU behaviour (e.g., lack of remorse). Data from three tasks (emotion recognition, perspective taking, emotional affect induced by another person’s situation) suggested that the ASD group was impaired in mentalizing in contrast with participants with conduct disorder plus CU, who were impaired in emotional empathy. The contrast between ASD and children with CU on mentalizing and emotional empathy tasks suggests that knowing what others think and caring how others feel are fully dissociable. Thinking of some types of conduct disorder as deficits of emotional processing might open the way to new interventions; it is interesting, for example, that a single administration of intranasal oxytocin improved fear recognition in typically developing
participants (Fischer-Shofty, Shamay-Tsoory, Harari & Levkovitz, 2010).

The label ‘pathological demand avoidance’ has been proposed by Newson, Le Maréchal and David (2003) for a group of children, some of whom may be on the autism spectrum, who show extreme and apparently socially manipulative refusal of ordinary, everyday requests or demands. The children described are often excluded from school, despite apparently average intellectual and language abilities, because they never comply, identify with the teachers rather than pupils, and show shocking behaviour with apparently little regard for others’ feelings or opinions. Whether this label marks out a special subgroup, or a troubling behaviour that may be found in many different diagnostic groups, is as yet unclear. There is scarcely any research (see O’Nions, Viding, Greven, Ronald & Happé, in press), but these very challenging children lead us to reflect on why typically developing children do comply; presumably social cognitive processes including recognition of hierarchy, affiliation, social identification with peers, and herd behaviour contribute. It is interesting to note that children with ASD do appear to absorb society’s stereotypes and make in-group/out-group distinctions of salient categories, such as race and gender (Hirschfeld, Bartmess, White & Frith, 2007). At the same time they do not, for example, appear to show reputation management (Chevallier, Molesworth & Happé, 2012) or those aspects of in-group behaviour that perhaps require mentalizing.

Attention Deficit/Hyperactivity Disorder typically begins to be diagnosed in this period, although the onset of significant inattention/hyperactivity may be several years earlier. Social cognitive impairments have been reported in young people with ADHD, including deficits in emotion recognition in face and voice, and possibly reduced theory of mind and empathy. Nijmeijer et al. (2008) reviewed the literature on social dysfunction in this group, and Uekermann et al. (2010) provide a comprehensive review of work on possible underlying neural substrates. Fronto-striatal pathways are implicated (orbitofrontal cortex, caudate nucleus, the ventral striatum) as well as the cerebellum in Soliva et al.’s (2009) review. An important question is whether social difficulties in individuals with ADHD are purely secondary to executive dysfunction; performance on facial affect tasks has been reported to correlate with sustained attention and inhibitory performance (Sinzig, Morsch & Lehmkuhl, 2008).

Children with Specific Language Impairment show social difficulties that spread beyond direct communicative or even pragmatic problems, to more general peer interaction even in nonverbal (e.g., cooperation) assessments (see e.g., Marton, Abramoff & Rosenzweig, 2005). Whether these represent downstream effects of language difficulties that disrupt peer relationships and hence social experience, or whether there are core socio-cognitive difficulties in at least some children with SLI remains uncertain.

Children with complete or partial callosal agenesis are typically described as socially naive and vulnerable. While in utero absence or partial formation of the corpus callosum is often part of a genetic condition with accompanying intellectual disability, in some individuals only the corpus callosum is affected, and IQ and adaptive functioning can be in the average range. Social skills form the main focus for concern in many such cases, and deficits can sometimes be seen on tests of theory of mind or emotion recognition – although the presentation is more of delay than abnormality (Booth, Wallace & Happé, 2011).

Adolescence

This developmental period could justify a whole article to itself. Here, we can touch on only a few striking changes in social abilities, cultural conformity, and accompanying brain activity and structure. We highlight the increasing importance of self-awareness and emotion regulation in relation to new peer groups and other culturally salient factors. ‘Social policing’, reputation management and monitoring of information about individuals, all become increasingly important at this time. These various sophisticated functions are supported to a greater or lesser extent by all four networks shown in Figure 1. Of course, social and neural development does not cease after adolescence, but a discussion of changes in adulthood and old age is beyond the scope of this article.

During adolescence major changes in peer relations occur and, arguably, conformity with in-group peers becomes more important than ever before. In a recent review, Crone and Dahl (2012) argue that, rather than an explanation in terms of sex hormone levels and immaturity in the frontal network, increased risk taking and dangerous behaviour at this time has its roots in changes in social and affective processing. These changes are not only associated with vulnerability, but also confer greater flexibility in adjusting goals and motivation in line with the changing social context. During adolescence social interactions become more complex as shifts in belonging to new in-groups can shatter older structures. This necessitates new responsibilities in ‘social policing’. Likewise different roles for the self must be managed, often in competition with same and opposite sex peers, with parents and sibs, and hence changes in social-identity and self-awareness are perhaps inevitable. Greater cognitive control (involving e.g., working memory, or inhibition of action) is achieved throughout childhood and adolescence, as shown in fMRI studies (reviewed by Crone & Dahl, 2012), with downstream effects on social functioning.

As adulthood advances it is increasingly vital to monitor and maintain reputation, which facilitates
the choice of partners (Tennie, Frith & Frith, 2010). The automatic tendency to cooperate, which is already evident in early childhood, is increasingly modulated by other considerations, including in-group status and social hierarchy. In many nonhuman primate species, this tracking of social hierarchy is a key aspect of social cognition, modulating cooperative versus competitive interaction. Human cooperation and reciprocity, as explored in the lab through economic games (e.g., Nowak & Sigmund, 2005), are also highly dependent on moment to moment assessment of a partner’s intentions and good will. This therefore becomes a central task for the brain’s mentalizing system. However, assessment and evaluation of the partner’s intentions is not enough. The dynamics of cooperation require advertising one’s own good intentions too and a degree of forgiveness to solve social dilemmas (Van Lange & Joresman, 2008). What we learn about others and what we remember about our interactions with them has to be kept ‘on file’ in what we tentatively label ‘individuals’ information store’. It is likely that this knowledge is increasingly important over a lifetime.

Clearly, a whole range of social cognitive skills, from face processing, emotion processing and decision making, to in-group allegiance, are undergoing major changes during adolescence (for a review see Burnett, Sebastian, Cohen Kadosh & Blakemore, 2011). For example, performance on difficult tasks involving taking another person’s perspective, continues to develop into late adolescence (Dumontheil, Apperley & Blakemore, 2010). Medial prefrontal cortex and the temporo-parietal junction, the most commonly found regions of the Mentalizing network, change radically in their relative weighting over the adolescent period (Burnett, Bird, Moll, Frith & Blakemore, 2009). Greater activation of temporo-parietal junction is typical of the adult brain, while greater activation of medial prefrontal cortex is typical of the younger brain.

The ability to represent increasingly complex mental states may play a part in the emergence of ‘cognitive reappraisal’ to regulate emotions (McRae et al., 2012). In adults, reappraising distressing and negative experiences to come to a more positive construal, is a commonly used emotion regulation strategy, and individual differences in reappraisal are associated with greater positive affect and well-being, and reduced negative affect and depressive symptoms. McRae et al. (2012) compared children (10–13 years), adolescents and adults on a task involving emotional reactivity and reappraisal and found age-related increases in left ventrolateral prefrontal cortex, previously identified in adult reappraisal, and a u-shaped curve for activation with age in regions associated with mental state attribution (medial prefrontal cortex, posterior cingulate cortex, anterior temporal cortex). These findings support the notion that, as well as requiring various executive functions linked to prefrontal regions maturing during this period (working memory, generation of alternative interpretations, selection and monitoring), cognitive reappraisal of emotions requires introspection on own mental states. Goddings, Burnett Heyes, Bird, Viner and Blakemore (2012) found that pubertal stage in females had an effect on brain activity during introspection on social emotions in contrast with basic emotions, again mainly implicating the Mentalizing network (medial prefrontal cortex, posterior superior temporal sulcus). Greater age was associated with lower medial prefrontal cortex activity, while increased hormone levels, regardless of age, were associated with higher activity in anterior temporal cortex.

We now know that changes in bodily appearance and social and emotional behaviour in adolescence are accompanied by structural changes in the brain to a greater degree than had previously been suspected (for a review see Blakemore, 2008). A study of 288 healthy individuals between the ages of 7 and 30 (Mills, Lalonde, Claesen, Giedd & Blakemore, 2012) found that the main parts of the social brain (medial prefrontal cortex, posterior superior temporal sulcus and temporo-parietal junction) at first increase in volume, with a peak around age ten, and then decrease through to the mid-twenties. Against this knowledge it will be possible in the future to compare atypical development in clinical groups.

**Atypical development.** A great many psychiatric conditions arise during adolescence. Paus, Kesha- van and Giedd (2008), reviewing the evidence and possible causes, conclude that this is likely related to anomalies or exaggerations of typical adolescent maturation processes acting in concert with psychosocial (e.g., school, relationships) and/or biological environmental factors (e.g., pubertal hormonal changes, drugs of abuse)’ (Paus et al., 2008; p. 953).

**Social anxiety disorders** peak in adolescence but can occur at any age. Neuroimaging studies suggest hyperarousal of amygdala and prefrontal regions in response to social threat, and a recent (albeit small) longitudinal study found a predictive relationship between neural reactivity to angry faces in middle childhood and adolescent social anxiety (Battaglia et al., 2012). It is thought that attentional biases to negative information, and mistaken cognitions about self and about others’ thoughts (e.g., ‘I look so silly and flushed – everyone is laughing at me’), may be important in the origins and/or maintenance of social anxiety. Here, some remarkable progress in cognitive behavioural therapies and attention-training interventions has been shown (e.g., Holmes & Mathews, 2010).

**Substance abuse and eating disorders** are also considered to appear at high frequency in adolescence. These disorders might involve problems in attention and self-control. Since voluntary actions and inhibition of impulsive actions depend heavily
on the maturity of the prefrontal cortex, the huge restructuring of prefrontal regions during adolescence (Dumontheil, Burgess & Bakemore, 2008) might well be a relevant risk factor. In addition, for substance abuse the increased novelty seeking and risk taking, along with heightened sensitivity to reward, may play a role (Fareri, Martin & Delgado, 2008). A recent review by Luciana, Wahlstrom, Porter and Collins (2012) suggests that increased incentive motivation in adolescence, and subsequent decline in adulthood, may be modulated by dopamine.

Schizophrenic disorders, which frequently begin in adolescence or early adulthood and have been the subject of huge research activity in relation to social cognition, are beyond the scope of the present article and the reader is instead referred to recent reviews (e.g., Green et al., 2008).

Sketching a map of social cognition

Our review highlights a wide range of functions involved in social interaction as they develop and change over a lifetime. Here, we attempt to synthesize this overview and to map out some putative relations between different aspects of social cognition, tagged by behaviour that is key to each aspect. Figure 2 presents a sketch using the format of network mapping. To sketch this map we have attempted to use information about typical social development, and especially about dissociations between aspects of social cognition that are apparent in atypical social development.

This procedure is necessarily tentative and only intended to provide a starting point for discussion and empirical test. While the format of network mapping by definition distinguishes elements and by implication separate cognitive components, these elements are certainly assumed to be highly interdependent and interconnected. Ideally a map such as this would be animated to show network changes through development, but much more information is needed before this could be attempted.

A long-term aim might be to superimpose a map showing which parts of the system are affected in different developmental disorders. It may never be possible to superimpose neatly a map of the neural processes involved in each node and network, because the mappings are far from one-to-one; indeed any brain region will subserve many functions and each cognitive function will require the coordinated activity of a network of regions.

In keeping with the structure of this review, we briefly discuss the major nodes of social cognition suggested in Figure 2, in an approximate order of emergence in development and when they are most studied. Relevant findings from neuroscience, using...
the regions and networks indicated in Figure 1, and relevant neurodevelopmental disorders are mentioned for each putative node.

1. **Agent identification** has been linked to superior temporal sulcus in particular. The subnodes of this system include biological motion detection, face and voice processing, contingency detection (Blakemore et al., 2003) and eye-gaze tracking (Carlin & Calder, 2013). All relate to a network of regions encompassing superior temporal sulcus in particular, but also medial prefrontal and parietal regions. Neuroimaging studies reveal an overlap with the network involved in mental state attributions whenever the task requires that attention is directed towards processing the intentions of agents (marked as Mentalizing network in Figure 1). In Williams Syndrome, the intense orientation to faces and voices may suggest some up-regulation of parts of this system. In ASD the opposite may be the case. Reduced ability in some of these functions (e.g., biological motion detection) has been reported from as early as age 2 years in ASD (Klin, Lin, Gorrindo, Ramsay & Jones, 2009), and has also been found in the nonautistic infant siblings of ASD individuals (Kaiser et al., 2010). It remains to be seen whether these impairments are primary and foundational to other social impairments. **Developmental prosopagnosia**, which clearly affects only the subnode of face identity recognition, does not prove devastating to social interaction, as it leaves intact the ability to recognize and individuate agents based on other sources of information.

2. **Emotion processing** critically involves the Amygdala network of Figure 1. Impairments in emotion processing in ASD have been reported, especially in the recognition of emotional expressions. However, a recent meta-analysis concluded that any group differences are at most marginal (Uljarevic & Hamilton, 2013). The regulation of emotion is another matter and this prominently involves fronto-striatal pathways (Ochsner, Silvers & Buhle, 2012). This complex process may well be problematic in individuals with ASD (Bird et al., 2010; Samson, Huber & Gross, 2012), and is likely to be implicated in disorders of social anxiety.

3. **Emotional empathy** is a key node, which has been linked to regions including the anterior insula, amygdala and anterior cingulate (Empathy network in Figure 1). Age related changes (from middle childhood to adulthood) in brain activation have been reported during tasks requiring reflecting on one’s own emotional response to others’ emotional expressions (Greimel et al., 2010). The most striking disorder of empathy is psychopathy, which in children is diagnosed as conduct disorder with callous/unemotional traits. In addition, individuals with alexithymia by definition have marked difficulty in identifying their own emotional state, but also show deficits in empathy and recognizing emotions in others.

4. **Self-processing** is an important hub which includes sub nodes for self-awareness of own emotional and bodily state (anterior insula), and social identity (medial prefrontal cortex; part of the Mentalizing network in Figure 1). It is interesting to speculate that the distorted self-perception seen in social anxiety disorder may relate to (genetic or environmentally-acquired) problems in this system. An abnormality in the neural basis of self-representation and self-referential cognition has been reported in fMRI studies with individuals with ASD on the basis of a poorly functioning Mentalizing network (Lombardo et al., 2007, 2010).

5. **In-group/out-group** categorization and stereotyping of social groups has a different neural basis from categorization of other semantic categories, being linked to activity in medial prefrontal cortex, posterior cingulate, bilateral temporoparietal junction and anterior temporal cortex (Contreras, Banaji & Mitchell, 2012). This node has key links to the major node of affilia-tion, as well as the related nodes of compliance and herding, and automatic mimicry/copying (linked to the Mirror network in Figure 1). While oxytocin (and vasopressin) has traditionally been linked with bonding and affiliation, it may have wider relevance for all these processes; administration of intranasal oxytocin both increases preference for in-group members and hostility towards an out-group (Stallen et al., 2012). From existing behavioural studies (e.g., stereotyping by participants with ASD according to gender and race; Hirschfeld et al., 2007) we have little reason to suspect specific impairments in these nodes in ASD, where attachment, for example, is as expected for developmental level. Instead impairments might be associated with conduct disorders of the oppositional defiant level. Children with Williams Syndrome might be hypothesized to have abnormal up-regulation in some aspects of affiliative behaviour or down-regulation of sensitivity to social threat (see below).

6. **Social hierarchy mapping** is another important node in our sketch. Its neural basis might lie in amygdala and hippocampus (Kumaran, Melo & Duzel, 2012). It is linked to kin recognition (which involves e.g., anterior cingulate and medial prefrontal cortex when using facial resemblance; Platek, Krill & Kemp, 2008). We see it connected strongly to the in-group/out-group node. Very speculatively, children receiving the label ‘pathological demand avoidance’ (Newson et al., 2003) may have problems in social identity and specifically knowing how they fit into a social hierarchy with expectations of compliance.
7. Mental state attribution and related nodes (communication and ostension, pedagogy, reputation management) have been robustly linked to a network consisting of medial prefrontal cortex, temporo-parietal junction and basal temporal regions (Mentalizing network in Figure 1). The few neuroimaging studies of children to date suggest that very similar brain regions to those seen in adults are activated in mentalizing tasks from around age 5 years (Gweon et al., 2012). **ASD** is the clinical diagnosis most clearly linked to deficits in automatic mental state attribution and related processes including intentional imitation. Against initial expectations, it appears that ASD is unlikely to involve impairments in automatic imitation/copying (Hamilton, 2013), processes linked with the Mirror system in Figure 1.

8. **Social policing** has subnodes involved in monitoring fairness, detecting cheaters and promoting altruistic punishment. The underlying neural network is associated with compliance and punishment and involves orbitofrontal cortex (Spitzer, Fischbacher, Herrberger, Gron & Fehr, 2007). Another subnode relates to judging relative trustworthiness, with a neural basis in the amygdala (Adolphs, Tranel, & Damasio, 1998). This in turn links, via monitoring moral behaviour, to reputation management and to the Mentalizing network of Figure 1. Moral judgement appears to involve both mental state attribution processes involved in judgement of intent (ventromedial prefrontal cortex), and neural circuitry underlying negative evaluation and even disgust (amygdala, insula; Decety et al., 2012). Findings of reduced neural and physiological activity in response to social threat might suggest that individuals with **Williams Syndrome** have deficits in processes related to social policing, resulting in dangerous degrees of trust and approach.

9. Both of the preceding nodes have essential links to a putative node we label **Individuals’ information store**. Here, information about recognized individuals is stored and retrieved. This updating record for each person includes, for instance, their status, their interests, dispositions and influence. Others’ standing in relation to ourselves and others needs to be updated continuously, based on episodic memory, to track such things as on what terms we parted, what debts might be owed or what amends should be made. Information about people, as opposed to other classes of things, has been suggested to involve lateral prefrontal and temporo-parietal regions (Satpute, Badre & Ochsner, 2013). How this information is represented in different neurodevelopmental disorders, and what qualitative changes might occur with age, are as yet unknown.

**New questions**

This review has taken as a starting framework the chronology of typical social development. This has allowed us to map out some core aspects of social cognition as they emerge in development, and to examine these in relation to a range of developmental disorders that may affect social development. We have attempted to reflect in our review what is known about the brain basis of the social functions discussed, with special focus on atypical development. We have resisted the more usual structure of reviewing each developmental disorder separately, trying instead to draw out cross-disorder similarities or differences. For convenience we have divided the timeline into chunks, from birth to childhood to adolescence. This has been somewhat ambitious, in which we have tried to include all aspects of social cognition at least in brief. It serves, perhaps, to highlight areas where we know rather little, and where the reader may be encouraged to address these open questions, ideally through cross-disorder comparisons. In this final section we pick up some themes that emerge from looking across development, domains and disorders. In particular we ask: how can we distinguish primary from downstream types of social cognition? Given the wide range of developmental disorders including a social aspect, should we conclude that social cognition is particularly delicate and highly vulnerable? Should we accept uncritically notions of ‘cascading’ social and neural development, with vital early building blocks underpinning later emerging abilities?

We focused on the neural basis of social cognition, defined widely as the many processes by which the mind understands the socio-emotional world. It is clear, however, that everyday social competence relies on not only core social cognitive processes, but a range of domain-general functions, many falling under the umbrella of ‘executive function’. Cognitive control in childhood, for example, appears to predict a range of desirable outcomes, not only in social functioning, but also in individual differences in academic achievement and even health (Moffitt et al., 2011). It may therefore be useful to distinguish primary disorders of social cognition from secondary or ‘downstream’ effects on social functioning due to primary nonsocial difficulties. While ASD is thought to represent the prototypical case of a primary social disorder, it may be that problems of social interaction in, for example, ADHD represent downstream or knock-on effects of executive or behavioural dysregulation on peer interaction. Social difficulties in children with SLI may also be of this downstream type.

How can we distinguish downstream from primary social deficits? At least four possible pointers, of different weights, can be suggested. First, when social deficits are downstream effects it would be...
expected that they will not be universal to that diagnostic group, since the cascade of effects will be open to many intervening factors. Second, downstream effects may be more likely to remit over time. Third, in principle, the downstream social difficulties may show a (longitudinal) correlation with the severity of the causal, upstream nonsocial deficit. Fourth, intervention studies may indicate that, when the upstream problem is reduced, the social deficits ameliorate, although again such developmental processes may need to be studied over a long period of time.

While general aspects of development, such as sensory, motor, memory, attention and language functioning all contribute to social functioning, it is remarkable that social development can withstand problems in many of these areas. For example, the existence of good social skills in those with developmental prosopagnosia or Moebius Syndrome suggest that specific difficulties with face recognition or with facial emotional expression are not a bar to normal social functioning. These examples might suggest that social development is not precious and delicate, but vital and robust.

If this is the case, why do so many developmental disorders appear to disturb social competence? One possibility is that social functions are not disproportionately affected, but rather that we as observers are more sensitive to perturbations of social skills than of other functions (e.g., motor, articulation). This might be plausible given the proximal effects of social skills on reproductive fitness, and hence evolutionary pressure on recognition of small individual differences in this area. This is one possible explanation for why it might appear that individual differences are more marked in social than in other skills (Kennedy & Adolphs, 2012); we would argue that individual differences in for example, executive functions or IQ are at least as great, but that humans have not evolved to be acute detectors of differences in the latter domains. There may also be particular sensitivity to social deficits because, in contrast to say phonological impairments in dyslexia, others’ social skills directly affect our own actions and interactions.

An alternative view is that social competence is often affected in developmental disorders because social interaction is the most complex of our species-typical skills, necessitating many component functions, any one of which when disturbed is sufficient to disrupt interaction. Indeed, so many brain regions appear to work in concert to subserve social cognition, that is might seem unsurprising that social deficits often result from abnormal brain development. However, we do not accept uncritically the notion that social stimuli are the most complex in our environment. This would clearly depend upon defining some metric of complexity. It is important, especially in the context of this review, to note that there are many conditions (e.g., Down Syndrome, Prader-Willi Syndrome) in which social functioning is not specifically impaired, but is in line with general intellectual and adaptive functioning. It is also the case that social cognition is not necessarily the first function to suffer when environmental insults occur; for example, long-term follow-up of children from Greek orphanages suggests that elevated rates of ADHD are the most striking outcome (Vorría & Ntouma, 2012).

We followed a developmental framework as it seems intuitively appealing to think that disruption of early and basic mechanisms will derail the later development of more complex functions. However, we found surprisingly few well-worked and clear examples supporting this intuition. Perhaps one of the greatest surprises in recent years has been the failure to find clear social deficits in very young infants later diagnosed with ASD. The earliest markers of later ASD, in attention and electrophysiological measures, currently appear to be nondeterministic and shown also by many non-ASD siblings.

The claim that early social processes are necessary for the development of later social skills should not, perhaps, be taken for granted. We believe that it is possible to test the claim through greater focus on individual differences. Little is known about otherwise typically developing children who fail to show one or more of the key socio-cognitive milestones on which we focus so much attention in developmental disorders. For example, a recent study of a large population-based sample showed that many children who fail to imitate at 2 years (by parental report and in a semistructured parent-infant task), go on to develop perfectly normal social skills (McEwen, 2009). It would be extremely interesting to know whether there are also ordinary children who show abnormal eye gaze, joint attention or pretend play but develop normally in later aspects of social functioning. If cases exist who can ‘skip’ these normal functions, it may indicate that these are not ‘stepping stones’ to typical social cognition, but rather (optional) manifestations of underlying social development, perhaps following separate streams. More evidence is needed concerning longitudinal relationships.

In other domains of brain development, there is good evidence of sensitive periods, and age of acquisition effects, such that insults/stress to the system at early time points have long-lasting effects (e.g., Heim & Binder, 2012). In the area of social development, there has been a strong expectation of similar effects (e.g., on attachment). However, the evidence to date does not overwhelmingly support the assumption of critical periods in social development and irreversible cascading effects. Nor does it fully support the idea that early abnormalities re-route development so that later intervention can only work through compensatory mechanisms. Doubt has been cast on these traditional ideas by the remarkable finding by Bird and colleagues on the possible reversibility of Rett Syndrome. Guy, Gan, Selfridge,
Cobb and Bird (2007) demonstrated that, in a mouse model of Rett Syndrome, the impaired phenotype could be ‘rescued’ even in maturity. This finding is so startling that we must begin to question whether our one-way cascade models always apply. Instead, this work suggests maintained rather than cascading effects, at least in Rett Syndrome, and possibly also in other disorders (notably Fragile-X syndrome). This raises new intervention hopes that some windows of plasticity may be wide open even later in life.

Conclusion

In the Introduction, we noted the existence of many reviews of the social brain, and of specific developmental disorders of social cognition, but none to our knowledge integrating the two. Having completed this review article we are more aware why! We would suggest that the current state of knowledge concerning cognitive and occasionally neural underpinnings of key components of social cognition is good in places, but rather little is known about development and transitions, even in typical development let alone developmental disorders. How does one build a social brain? This question reminds us of the story of a tourist who asked an Oxford college gardener, ‘How do you have such a lovely lawn?’ The gardener replied, ‘I cut it, feed it and water it’. ‘I do all that’, said the visitor, ‘but my lawn isn’t like this one’. ‘Well, you have to do it for 300 years’, said the gardener. So with the social brain – the timescale for individual development must be considered within the context of the evolutionary timescale. Nor do we refer only to the evolution of the ‘hardware’, but also the possible role of cultural evolution in social cognition (C.M. Heyes, C.D. Frith, unpublished data).

While we cannot in this article even begin to sketch the blueprint for building the social brain, we do believe that development and plasticity will be of paramount importance in understanding the neuroscience of social cognition. This is because, unlike many important adaptive functions, an individual’s role and therefore task as a social agent changes qualitatively through development. At different points on this journey an individual will be a naive learner, a co-operator, a competitor, a leader, a follower, a teacher, a carer. Even at any single point in development, an individual may play many different roles in relation to different conspecifics. All of these roles are also subject to change across cultures, requiring the evolved potential for massive flexibility throughout life. The putative socio-cognitive processes sketched in this review provide a starting point for investigating the extraordinary versatility of the developing social brain.

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Key points

- Existing literature covers well the adult ‘social brain’, the behavioural and neural basis of specific developmental disorders affecting social cognition (such as autism), but barely addresses the development of the social brain, nor cross-disorder comparisons.
- Taking typical social development as a chronological structure, we briefly review key social abilities, what is known about their neural bases, and their impairment in developmental disorders.
- A wide range of clinical groups show social impairments, but the distinct pattern of functions affected suggests that social cognition involves a variety of potentially separable components.
- We sketch a tentative map of social cognition, including: agent-identification, in-group/outgroup categorization, mental state attribution, emotion processing, self-processing, social hierarchy mapping and social policing.
- Sketching the wide array of social processes seen in typical development may help clinicians map uneven socio-cognitive profiles in young people with neurodevelopmental disorders.
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