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The Structure of Social Cognition: In(ter)dependence of Sociocognitive Processes

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ABSTRACT

Social cognition is a topic of enormous interest and much research, but we are far from having an agreed taxonomy or factor structure of relevant processes. The aim of this paper is to outline briefly what is known about the structure of social cognition, and to suggest how further progress can be made to delineate the in(ter)dependence of core sociocognitive processes. We focus in particular on several processes that have been discussed and tested together in typical and atypical (notably Autism Spectrum Disorder) groups; imitation, biological motion, empathy and 'Theory of Mind'. We consider the domain specificity/generality of core processes in social learning, reward and attention, and highlight the potential relevance of dual-process theories that distinguish systems for fast and automatic versus slow and effortful processing. We conclude with methodological and conceptual suggestions for future progress in uncovering the structure of social cognition.

Keywords: Social cognition, theory of mind, autism, imitation, empathy

1. WHY IS ESTABLISHING THE IN(TER)DEPENDENCE OF SOCIOCOGNITIVE PROCESSES IMPORTANT?

Few would deny the functional importance of social interaction, nor the value of scientific study of the processes supporting it. The last few decades have seen a snowballing of interest in the cognitive and neural bases of social processing, much of it motivated by the desire to understand and ameliorate clinical conditions characterised by problems in social interaction. Despite this interest, there is little agreement as to the core socio-cognitive processes or their inter-relation or independence – we call this the 'structure' of social cognition, which constitutes the focus of this paper. The term 'cognition' is used here in the same way as Morton and Frith (1995), to refer to the level of explanation lying between neural processes and behavior, and includes emotion, and we define social cognition as the processing of stimuli relevant to understanding agents and their interactions.

In contrast to the study of intelligence or personality, little work has examined the factor structure of social cognition. Even limited sampling of recent papers shows how differently authors divide up social cognition. Reviewing work on social cognition in non-human animals, Seyfarth and Cheney (2015) propose the core building blocks comprise individual recognition, knowledge of others' relationships (e.g., dominance), and 'Theory of Mind' (understanding of others' mental states; mentalizing). A recent review of social cognition in schizophrenia (Green et al. 2015), focused on "four general social cognitive processes — perception of social cues, experience sharing, mentalizing, and experiencing and regulating emotion". In their comprehensive textbook, 'Social Cognition: from brains to culture', Fiske & Taylor (2013) identify 14 domains of social cognition, ranging from more basic concepts such as social

attention, encoding of social stimuli, and social memory representations, to higher-order social processes such as social decision making, social inference, attitudes, stereotyping, and prejudice. Happé and Frith (2014), reviewing the developmental neuroscience of atypical social cognition, sketched a hypothetical network including at least eight separable components (affiliation, agent identification, emotion processing, empathy, individuals information store, mental state attribution, self-processing, social hierarchy mapping, social 'policing', and in-group/out-group categorization). NIMH's research domain criteria (R-DoC) initiative currently divides the domain of 'Social Processes' (which includes both traditional social psychological processes such as attachment, and socio-cognitive processes such as animacy perception), into four constructs: 'affiliation and attachment', 'social communication', 'perception and understanding of self', and 'perception and understanding of others'. There is also little consensus across authors as to which processes should be distinguished or which are inter-related. For example, R-DoC combines emotions and intentions under the subconstruct of 'understanding mental states', while other authors have claimed these to be dissociable (e.g. Lewis & Todd 2005). R-DoC also separates the understanding of self and others, while other authors have suggested that, for example, representing own and others' mental states require common representational mechanisms (e.g. Carruthers 2009).

Does it matter how we divide the space of social cognition, or whether we decipher its factor structure? It is helpful to note that these are two independent but complementary endeavors. The first relates to the development of a standard taxonomy and vocabulary of sociocognitive processes. At present, different authors use similar terms differently (e.g., 'empathy'), and different labels for ostensibly similar or overlapping processes (e.g. 'motor empathy' and

'imitation'; 'cognitive empathy' and 'Theory of Mind'), leading to misunderstanding and confusion. Loose definitions, and a failure to discriminate distinct processes, will add to problems of non-replication and cause difficulties in mapping cognitive to neurological processes. Which term is used for a particular process, and the dimensions one chooses to group sociocognitive processes, are a matter of taste rather than empirical investigation; all that is required are for these terms (for precise and testable processes) to be standardized and applied consistently by researchers.

The second endeavor involves determining the relationship between different sociocognitive abilities; whether, for example, individual differences in emotion recognition predict individual differences in Theory of Mind (ToM). This question is empirically tractable, and will allow sociocognitive ability to be described in terms of a smaller number of factor scores rather than a multitude of scores across different tests of social ability that may or may not measure distinct processes. Furthermore, the identification of latent factors that contribute to performance across a range of sociocognitive tests (as verbal ability contributes to a range of IQ subtests), is likely to aid in mapping sociocognitive processes to neural networks, and to identifying the genetic contribution to individual differences. Identification of these factors will make it easier to test causal hypotheses that could be vital to developing, for example, interventions for social impairments, or understanding the mechanism of putative treatments. Looking forward, having an agreed taxonomy of social cognitive processes with an understanding of the structure of social cognition would be a starting point for developing a shared protocol of tasks, allowing assessment of specific profiles of ability across sociocognitive processes, and across groups. Again, by analogy with intelligence testing, knowing an individual's peaks and troughs across

subcomponents of social cognition, would allow discrimination of phenocopies (e.g. attachment disorder versus autism), detailed measurement of change (e.g. decline in dementia), test of specificity of treatment effects, and cleaner mapping to neural or genetic bases of social ability.

In what follows we largely address endeavor two, the structure of social cognition, as adding a further idiosyncratic taxonomy of sociocognitive abilities to those already in existence would be of little use to the field. This makes our task harder, however, as very little research has explicitly addressed this question in large samples of neurotypical adults. As a result, we draw heavily on research addressing social processing in neurodevelopmental disorders, particularly Autism Spectrum Disorder (ASD), the archetypal disorder of social cognition. While this is a research area rich with relevant data, it should of course be acknowledged that the structure of social cognition in atypical populations may not reflect that in typical populations – due to compensation for example – but we hope that this research may suggest fruitful methods of investigation in typical individuals (Section 5), as well as other clinical groups (e.g. acquired lesions). A second difficulty relates to the sheer scope of social cognition - the full range of processes that contribute to social ability has never been delineated, as far as we are aware, and if we were to attempt to list them all we would likely have little space to do anything else. Therefore, the range of social abilities we discuss is limited and determined by the availability of evidence relating to their in/inter-dependence (much of which is from social neuroscience), the availability of existing reviews of the relevant literature, and our own fields of expertise. This necessarily means that there is a vast swathe of literature on social ability that we do not address, but several sections (detailed below) refer to general factors bearing on the structure of social cognition that are of relevance to many, if not all, areas of sociocognitive research.

2. AN OVERVIEW OF OUR APPROACH AND THE CURRENT STATE OF PLAY

In what follows we will introduce the sociocognitive processes that are the focus of the paper (Section 2.1), describe five ways in which sociocognitive processes may be related (Section 2.2), and the types of evidence commonly used to establish the relationship between these processes (Section 2.3).

Section 3 takes four of the five ways in which social processes may be related and reviews literature in which this relationship has been empirically tested. Section 3.1 presents research addressing the relationship between imitation, ToM and empathy, in order to determine whether these three social abilities are unitary, distinct, or whether the development of one of these abilities is necessary for the development of the others. Section 3.2 assesses whether 'self-other distinction/control' may be recruited by a number of sociocognitive processes, explaining correlated ability across seemingly distinct social abilities. Section 3.3 presents a possible example of one sociocognitive process constituting a necessary component of another; the abilities to perceive biological motion, and to imitate the actions of others.

Section 4 addresses an issue bearing directly on the question of the factor structure of social cognition; the extent to which social ability relies on domain-specific, possibly modular/modularised (Karmiloff-Smith 1994) processes, versus domain-general processes that are recruited for social and non-social processing alike. Although the domain specificity and factor structure questions are in principle distinct, if social ability were only to recruit general executive or perceptual processes, for example, one might expect much more overlap among

social abilities than if distinct abilities relied on different domain-specific or dedicated modules/ processes. Section 4 focuses specifically on the fifth way in which social abilities may be related; assessing whether factors affecting the speed and/or extent of social *development* are specifically social, or whether they merely recruit general learning, attention, and reward mechanisms.

Section 5 addresses how we might make further progress in determining the structure of social cognition. Section 5.1 outlines the available methodologies to address the question, and highlights their strengths and limitations. Section 5.2 makes general recommendations that could lead to further progress, focusing on conceptual rather than methodological issues.

2.1 Putative Components of Social Cognition

Potentially, any cognitive process may be called into the service of understanding social agents and social interactions. However, as previously described, this paper will discuss only a subset of social abilities to illustrate various ways in which different social abilities may be related. These abilities include those related to:

- Affiliation and social motivation: Factors influencing approach tendency, and hence quantity of an individual's social interaction.
- Agent recognition: Allowing conspecifics to be individuated.
- Biological Motion Perception, Action Recognition and Imitation: Processes underlying the ability to determine which action is being performed by an agent, and the reproduction of that action by the self.
- Emotion recognition: The ability to determine the affective state of another.

- Empathy: When recognition of another's affective state prompts the recogniser to adopt the same state (with the added requirement that one recognizes that the other is the source of one's state under some accounts).
- Social attention: The degree of attention paid to social stimuli either due to a conscious choice (endogenous attention) or as a result of automatic capture of attention (exogenous attention).
- Social learning: Learning from other individuals.
- 'Theory of mind': The ability to represent one's own mental states (propositional attitudes, e.g. beliefs), and those of others.

2.2 Types of Relationship between Components of Social Cognition

There are at least five ways in which sociocognitive processes may be related.

1) They may actually be synonymous, or alternative labels for the same core process. For example, while several authors claim that mirror neurons contribute to 'action understanding' (Gallese & Sinigaglia 2011, Rizzolatti et al. 1996), others have suggested that action understanding is synonymous with either action perception (determining which action has been performed) or ToM (determining the intention driving the action) (Rizzolatti & Sinigaglia 2010).

2) One process may constitute a necessary (sub)component of another. For example, emotion contagion (in which the affective state of another is mirrored in the self) is thought to be a necessary component of empathy under frameworks in which empathy is said to have occurred when the empathizer recognizes that the other is the source of their current emotional state (de Vignemont & Singer 2006).

3) They rely on at least one common process, but also have distinct elements. For example, it has been argued that several sociocognitive processes rely on the ability to distinguish representations of the self and others (See Section 3.2). When empathizing with another, one must be able to distinguish between one's own emotional state and that of the other, and when inhibiting the tendency to imitate another, one must be able to distinguish between one's own motor plan and that of the other. While both imitation inhibition and empathy may require self-other distinction processes, each is likely to recruit additional distinct processes.

4) Two sociocognitive processes may be developmentally associated, due to a direct causal link. This is sometimes referred to as 'cascading' (**Fig. 2C**), where, for example, imitation is proposed to be essential for development of ToM. Such cascades are often referred to in theories of atypical developmental; e.g., deficits in social motivation are hypothesized to cause reduced attention to faces, in turn leading to failure of neuronal and cognitive specialization for face processing (Klin & Jones, 2008).

5) Two processes may be developmentally associated due to a third factor of importance to both. For example, two processes that are learned through social interaction during development (for example imitation and empathy), may develop at the same speed/level as a product of an individual's degree of social attention. An individual who is a good social learner may learn to imitate and empathize quickly and thoroughly (**Fig. 2A&B**), while the opposite may be true of a poor social learner.

2.3 Types of Evidence Currently used to Establish Relationships

Broadly speaking, researchers interested in the relationships between cognitive components of social processing currently refer to 5 types of evidence.

1) Single or double dissociation of abilities in developmental or acquired clinical groups: If Process X is intact but Process Y impaired in one group, and Process X impaired and Process Y intact in another group, then it is concluded that Process X is distinct from Process Y.

2) Neuroimaging data demonstrating overlapping or distinct brain activity during different tasks/processes: Differential activation caused by two different social tasks follows the dissociation logic described above, but common activation of neurological networks by two social processes often prompts the conclusion that the social processes recruit common cognitive mechanisms.

3) Correlations (cross-sectional) between individual differences in two or more socio-cognitive processes: Patterns of co-variation across individuals have been used to support claims of common mechanisms between processes.

4) Longitudinal associations of individual differences: Co-variation within individuals across development has been used to argue for developmental cascading, where the acquisition of one social ability leads to the acquisition of another.

5) Intervention effects: If interventions (psychological, pharmacological, etc.) can differentially affect social abilities then they are seen as distinct.

3. WHAT DO WE KNOW CURRENTLY ABOUT THE LANDSCAPE OF SOCIAL COGNITION?

In the following section we turn to empirical evidence concerning the factor structure of social cognition. Research directly addressing this question is scarce, but we have sought to illustrate four of the five ways in which social abilities may be related using examples from the literature. We address the fifth relationship in Section 4.

3.1 Imitation, Empathy, and ToM: Synonymous, Developmental Cascade, or Distinct?

While research that attempts to determine the factor structure of social cognition is in its infancy, several sets of social abilities have been examined together, typically because they are associated with psychopathological conditions, or because one ability is hypothesised to be either a 'stepping stone' or subcomponent (**Fig. 1**) of the others. All of these motivations underlie research examining imitation, ToM, and empathy together; all three processes have been hypothesized to be impaired in Autism Spectrum Disorder (ASD), and imitation has been proposed as a necessary building block for the development of ToM and empathy. Indeed, the concept of empathy has sometimes been extended to include imitation ('motor empathy'), ToM ('cognitive empathy') and 'affective empathy', and it has been argued that 'empathizing' ability is a primary trait governing individual (and gender) differences in social ability (Baron-Cohen 2009).

Recent evidence, however, does not support a link between these three processes. The idea that imitation leads to the development of ToM and empathy due to the operation of an innate module (Meltzoff & Moore 1977) is not supported by data showing that imitation relies on domain general learning rather than an innate module (Anisfeld 1979, Cook et al. 2014c, Jones 2009, Ray & Heyes 2011). In addition, McEwen et al (2007) found that some typically developing children who were reported at age 2 years to show no imitation, nonetheless had social skills in the average range at age 8 years. Thus, imitation may not be a vital stepping stone to later mental state attribution.

The hypothesized link between imitation, ToM and empathy was bolstered by the discovery of 'mirror neurons', neurons which fire when actions are both executed and observed (di Pellegrino et al. 1992). These cells are thought to support imitation (Catmur et al. 2009, Heiser et al. 2003), and were originally thought to code the 'goal' of an action (Bonini & Ferrari 2011, Rizzolatti & Craighero 2004, Rizzolatti & Sinigaglia 2010), a nonspecific term commonly construed as the intention behind an action, i.e. a mental state. By coding for own or others' intention, mirror neurons were proposed to provide a neural basis for ToM (Rizzolatti & Sinigaglia 2010).

However, more recent evidence calls into question the straightforward interpretation that mirror neurons code the goals of actions, and therefore form a connection between own and others' intentions (see Cook et al., 2014; Cook & Bird, 2013 for a summary). Perhaps most convincingly, a meta-analysis of neuroimaging studies of ToM that set-out to determine the contribution of mirror neurons concluded that "the mirror system is not activated and does not aid the mentalizing system in detecting intentionality" (Van Overwalle & Baetens 2009).

As mentioned above, one important motivation for hypothesizing the inter-dependence of imitation, empathy and ToM, has been the claim that all three social processes are affected in ASD. If ASD is characterized by impairments in all three areas, then a parsimonious explanation is that the three are developmentally linked, or rely on a common underlying process (on-line or developmentally) (Colombi et al. 2009, Eckerman & Whitehead 1999, Hobson 1989, Rogers & Pennington 1991).

Again, recent evidence calls into question the claim that ASD is a condition characterized by deficits in imitation and empathy. Studies of automatic imitation (whereby observation of another's action prompts the tendency to produce an identical action) reveal that individuals with ASD have at least a typical, if not *increased,* tendency to copy simple hand and finger actions (Cook & Bird 2012, Sowden et al. 2016, Spengler et al. 2010), and emotional facial expressions (Press et al. 2010). Where deficits in voluntary, non-automatic, imitation are observed in ASD, these are likely due to non-specific factors such as attentional control, working memory and/or pragmatic language understanding (Leighton et al. 2008). In addition, available evidence is either unable to support mirror neuron deficits in ASD (Hamilton 2013) or suggests that abilities claimed to depend upon mirror neuron function (e.g. action understanding and prediction) is typical in ASD (Hamilton et al. 2007).

Further evidence from clinical groups also suggests that affective empathy and ToM are distinct and demonstrate a double dissociation. While ASD does not seem to be directly linked to problems with affective empathy (Bird et al. 2010), most individuals with ASD show impaired ToM (Happé 1994, White et al. 2009); individuals with high levels of psychopathic traits demonstrate intact ToM but impaired affective empathy (Jones et al. 2010, Lockwood et al. 2013, Schwenck et al. 2012). Furthermore, meta analyses of neuroimaging of ToM and empathy in typical and atypical populations have identified reliable, but non-overlapping networks including the medial Prefrontal Cortex (mPFC), temporoparietal junction (TPJ) and precuneus for ToM (Frith & Frith 2010, Saxe et al. 2006), and anterior insula and anterior cingulate cortex for empathy (Singer & Lamm 2009).

3.2 Self-Other Distinction and Control: A Common Factor?

Although imitation, ToM and empathy appear to be distinct processes, evidence suggests that false belief attribution (a key test of ToM; tracking a character's mistaken belief), empathy and the ability to *inhibit* imitation may call on a common process - that of self-other distinction and control (**Fig. 1**). This proposal was originally made by Brass and colleagues (Brass et al. 2005), who noted that imitation inhibition caused activation of a neural network commonly seen during ToM tasks. They suggested that this activation may reflect a common process, self-other distinction, that is necessary for both imitation inhibition and ToM. It was argued that in order to inhibit imitation it is necessary to distinguish between one's own motor intention and that of another, and, at least in classic false belief tests of ToM, one must be able to distinguish between one's own knowledge states and those of another (inhibiting own true belief to predict behavior based on another's false belief). This explanation was tested in typical individuals and those with ASD (Spengler et al. 2010), who completed a test of imitation inhibition and verbal and non-verbal tests of ToM. Within the ASD group performance on the imitation inhibition test predicted performance on the verbal ToM test and neural activation in the ToM network when completing the non-verbal ToM task. These measures were not associated in the group of typical adults, which in principle could reflect a meaningful difference between the way in which typical individuals and those with ASD complete the tasks, but in this case likely reflects the fact that the tests of ToM were less sensitive to individual differences in typical individuals due to ceiling effects.

The hypothesis of a common self-other distinction process recruited by multiple sociocognitive processes (see Figure 1) was tested using two 'intervention' studies in which individuals were

trained to inhibit imitation (theorized to increase their ability to distinguish and control representations of the self and others), before completing other sociocognitive tests to identify transfer effects. Santiesteban et al (2012b) tested the impact of imitation inhibition training on a visual perspective-taking task. It was predicted that the visual perspective taking task would recruit the same self-other distinction process as ToM and imitation inhibition; in order to represent another's perspective it must be distinguished from one's own. This prediction was fulfilled - performance on the visual perspective taking task was improved by imitation inhibition training but not by imitation training, nor by training on a standard Stroop inhibition task closely matched for difficulty. Using a study with a similar design, de Guzman et al (2016) demonstrated an effect of imitation inhibition training on empathy for pain – thought to be due to the fact that in order to be empathic one must be able to distinguish one's own, non-pain state, from the pained state of the other.

A number of studies using fMRI and/or transcranial magnetic stimulation (TMS) have demonstrated an important role for the TPJ in self-other distinction (Brass et al. 2005, Hogeveen et al. 2015, Santiesteban et al. 2012a, 2015; Sowden & Catmur 2013). In line with this Santiesteban et al (2012a) used transcranial direct current stimulation (TDCS) to excite the TPJ and showed a corresponding *enhancement* of the ability to take another individual's perspective. However, Santiesteban et al (2012a) also showed, in the same individuals, that exciting the TPJ led to a *reduction* in imitation. Santiesteban et al (2012a) therefore suggest that the common process may be self-other *control* rather than distinction, defined as the ability to switch attentional focus between co-activated self- and other- related representations. This ability would

allow the selective enhancement of the self and inhibition of the other, or vice versa, according to task demands.

3.3 Biological Motion and Imitation: Constituent Processes?

As listed in section 2.2, one of the possible ways in which two putative sociocognitive processes might be related is that one constitutes a subcomponent or necessary input to another. An example of such a potentially constitutive relationship is between biological motion processing and imitation; a strong argument can be made that one can only imitate another's action if one can accurately perceive the action. Traditionally, biological motion processing and imitation have been treated as distinct topics of enquiry, however the fact that both abilities are thought to be impaired in individuals with autism has led to their investigation in some depth in this population.

'Biological motion' refers to the movements of other animate beings, and has been studied using a variety of stimuli from animations of moving people (e.g. Pelphrey et al. 2003), to single dots moving with a velocity profile that matches human movement (Dayan et al. 2007). Annaz and colleagues (2012) investigated attention to biological motion in young children with ASD and found that whereas typical children preferentially attended to biological motion, children with ASD showed no such preference. Together with work from other labs (Dawson et al. 1998, Klin et al. 2009) this finding suggests that, unlike typical children, those with ASD do not demonstrate preferential attention to social stimuli. Given that individual differences in some aspects of biological motion processing have been correlated with socio-cognitive abilities (Miller & Saygin 2013, Sevdalis & Keller 2011), it has been suggested that atypical attention to

biological motion from an early age could be part of a developmental cascade resulting in atypical sociocognitive abilities in ASD (Dawson 1991, Klin et al. 2003).

Reduced attention to biological motion from an early age may be causally related to atypical *development* of biological motion processing. Annaz and colleagues (2010) demonstrated that between the ages of 5 and 12 typical children improve in their ability to determine human form from biological motion whereas children with ASD do not (see also Blake et al 2003), while data from Koldewyn, Whitney and Rivera (2010) suggests that atypical biological motion processing in ASD extends into adolescence. Though the ability of autistic adults to process biological motion is a matter of debate (Koldewyn et al. 2010, Murphy et al. 2009, Saygin et al. 2010), Kaiser, Delmolino, Tanaka and Shiffrar (2010) demonstrated that, unlike typical adults, adults with ASD do not exhibit greater visual sensitivity for human motion relative to the motion of a vehicle. Likewise, using stimuli that require only local, not global, motion processing, Cook and colleagues (2009) demonstrated that adults with ASD were less sensitive to perturbations to biological motion compared to typical adults, but equally sensitive to perturbations to gravitational motion.

As discussed in Section 3.1, most studies have reported typical automatic imitation in ASD. However, there are some exceptions – and thinking about the relationship between imitation and biological motion perception may shed light on these. Cook and colleagues (2014a) asked participants to perform horizontal arm movements whilst observing congruent (horizontal) or incongruent (vertical) arm movements conducted by a virtual reality agent with either human or robot form. For typical individuals incongruent arm movements conducted by the human, but not

the robot avatar, interfered with on-going action control. In contrast, individuals with ASD were not affected by human or robot movements.

Imitation involves the activation of motor representations upon activation of a visual representation of action. Atypical imitation could therefore be the result of atypical visual biological motion processing. In line with this, it is notable that paradigms demonstrating typical imitation in ASD tend to have employed stimuli that rely on apparent motion - stimuli in which still images of body positions are presented and the viewer infers the kinematics of movement (as in a flicker book or traditional cartoon). With such stimuli the viewer's inferred kinematics are unconstrained and need not necessarily follow the kinematics of typical biological motion. However, some paradigms constrain participants' representation of movement kinematics by showing videos, or using live stimuli. For example, the stimuli presented by Cook and colleagues (2014a) were animations displayed at a high refresh rate, meaning that the representation of the kinematics of the movement was driven by perceptual input and not inferred by participants. In other words, evidence from the biological motion literature suggests that individuals with ASD may represent the kinematics of movement atypically, and this may have a concomitant effect on imitation if perception of action kinematics is a crucial component of the imitation task.

This literature provides a good example of the importance of considering that some socio-cognitive abilities may comprise a constituent component of other abilities. When imitation and biological motion processing are viewed in isolation it is difficult to explain why imitation appears atypical in some, but not all, situations in autistic individuals. However, if one considers

the extent to which an imitation paradigm constrains biological motion processing then the ambiguity may be resolved.

4. UNIQUELY 'SOCIAL' PROCESSING?

As discussed in Section 2, the extent to which social ability relies on domain-specific, possibly modular/modularised processes, versus domain-general processes that are recruited for social and non-social processing alike, impacts upon the question of the factor structure of social cognition. Although the domain specificity and factor structure questions are in principle distinct, if social ability were only to recruit general processes then one might expect more overlap among social abilities than if distinct abilities relied on distinct domain-specific modules (see Duchaine & Yovel (2015) for review of this issue within the face processing literature).

If one accepts that social ability is, to a greater or lesser degree, learned from others over development, then factors affecting the speed and depth of such social learning are likely to affect social ability. Assuming that social ability is typically a product of learning from others and individual trial-and-error learning (e.g. learning to imitate may rely on observation of others and on individual trial-and-error based learning to control one's own actions), then whether social learning is governed by socially-specific or domain-general factors will impact the interdependence of social processes. If good individual learners are also good social learners because both types of learning are governed by domain-general factors, then these individuals will excel in all social abilities regardless of the degree to which a particular social ability relies on social, rather than individual, learning. In contrast, if factors affecting social learning are domain-specific and distinct from those governing individual learning, then social abilities may

dissociate from one another as a function of the degree to which they rely on social vs individual learning. We therefore provide an overview of research examining the domain-specificity of social learning (Section 4.1), social reward (Section 4.2), and social attention (Section 4.3). Finally, we discuss the potential relevance of 'dual process' accounts to the question of domain specificity of sociocognitive processes (Section 4.4).

4.1 Social Learning

A domain-general view is that all learning, including social learning (i.e. learning from conspecifics), is governed by the operation of a few general learning principles (e.g. associative and instrumental learning; Heyes & Pearce 2015). Heyes (2012a) presents a summary of the evidence supporting a domain-general view of social learning. Perhaps most important is the finding that social learning covaries with non-social learning: in male zebra-finches song complexity (social learning) is correlated with the rate of learning in an extractive foraging task (non-social learning) (Boogert et al. 2008). Such correlations are seen, not just within species, but also across species, such that species that tend to be good social learners are also good non-social learners (Lefebvre & Giraldeau 1996, Reader et al. 2011, Reader & Laland 2002). This correlation between social and non-social learning is consistent with the view that there is just one single set of, domain-general, learning principles. Heyes also notes that if social learning were an adaptation for social living it would not be present in solitary species; however at least two solitary species (the common octopus and the red-footed tortoise) are capable of social learning (Fiorito & Scotto 1992, Wilkinson et al. 2010).

By contrast, theoreticians in the domain-specific camp have argued that living in social groups has specifically favored the evolution of social learning; that social learning is an adaptation for social living (Klopfer 1961, Templeton et al. 1999). Neuroimaging studies have demonstrated that social and non-social learning are associated with activity in dissociable neural networks, raising the possibility of distinct and specialized mechanisms. For example, Behrens and colleagues (2008) used fMRI to demonstrate that learning from individual experiences about reward outcomes was associated with activity in a network of brain regions including the ventral striatum and anterior cingulate sulcus, whereas social learning from an adviser was associated with activity in a distinct network of brain regions including the TPJ and anterior cingulate gyrus. Further evidence for dissociable mechanisms underlying social learning and non-social learning comes from a recent study by Cook et al. (2014b), which demonstrated that social and non-social learning dissociate with respect to their relationship with social dominance. Whereas social dominance predicted social learning ability, it was not related to ability to learn via non-social means. This result is consistent with the domain-specific view that social and non-social learning are underpinned by dissociable mechanisms.

With many questions yet to be addressed, the debate concerning the domain-specificity of social learning continues. For example, with respect to the neural correlates of social and non-social learning, Behrens and colleagues (2009) have argued that although the neural correlates may be dissociable in terms of their spatial location, it may still be the case that the same computational learning mechanisms are employed for both social and non-social learning. This issue has been examined using computational modelling approaches in which formal mathematical models of learning are used to model the learning behavior of real individuals. These studies have shown

that models developed to explain non-social learning can explain social learning (Diaconescu et al. 2014), although in some cases they may need to be modified to adequately explain social behavior (Boorman et al. 2013).

4.2 Social Reward / Motivation

Learning efficiency is affected by reward, and here we review evidence concerning the domain-specificity of social reward and the idea of specific social motivation. A number of theories have argued for domain-specificity in this area; in particular several theories suggest that ASD is characterized by a specific deficit in social motivation. The social motivation theory of autism (Chevallier et al. 2012, Dawson 2008) postulates that the starting point for the socio-cognitive differences in ASD is that social stimuli and activities are intrinsically less motivating for infants with ASD. For example, Van Etten and Carver (2015) have suggested that reduced social motivation explains reported imitation deficits in ASD (but see Section 3.1). Such a theory implies that reward systems have a modular organisation, in which social motivation can be selectively impaired, with the processing of other motivational factors (e.g. food or monetary rewards) being spared. Whether there exists a separable social reward system, however, is still a matter of debate.

Social reward and motivation is subserved by a network of brain regions including the amygdala, the ventral striatum, and orbital and ventromedial regions of the prefrontal cortex (Chevallier et al. 2012). A long-standing debate in the reward processing literature concerns whether primary rewards (essential for the maintenance of homeostasis and reproduction; food, sex and shelter) and secondary rewards (rewards not directly related to survival, e.g. money and power) are

processed in common or distinct brain structures (Schultz 2000). Some have speculated that primary and secondary rewards may be represented in phylogenetically distinct brain regions (Knutson & Bossaerts 2007), but the majority of researchers within the decision neuroscience and neuroeconomics fields have argued that various stimulus types are compared on a common scale in terms of their "decision value" (see Peters & Büchel (2010) for review). This debate can be extended to encompass social reward: is there one common reward processing network, or is it feasible that social reward processing might be subserved by at least partly dissociable neural mechanisms? Sescousse and colleagues (2013) reviewed the human neuroimaging literature concerning the processing of monetary, food and erotic rewards. They demonstrated that a core set of brain regions including the striatum, anterior insula/frontal operculum, mediodorsal thalamus, amygdala and the ventromedial prefrontal cortex were associated with reward processing in an indiscriminate fashion, consistent with the idea of a common "reward circuit". In addition, comparative analyses between rewards revealed that some regions were more specifically recruited by one type of reward compared to the others; for example, the bilateral amygdala, the ventral anterior insula and the extrastriate body area were more robustly activated by erotic than by monetary and food rewards. At face value this result suggests that although all types of reward recruit core reward processing mechanisms, different types of reward may be discriminated on the basis of neural mechanisms outside of the common reward circuit. This result makes it feasible that social reward processing could be subserved by at least partly dissociable neural mechanisms from those related to other rewards. However, it should be noted that it is unclear whether the partially dissociable networks identified by Sescousse et al (2013) are specifically related to reward processing; for example the extrastriate body area activation observed in relation to processing of erotic rewards may simply reflect the fact that these stimuli,

but not money or food stimuli, contained images of bodies. In other words, differences in neural activation may simply reflect different types of input to a common reward system.

4.3 Social Attention

Objects with social importance are prioritized by attention; social stimuli capture attention automatically, rather than requiring deliberate attentional control (exogenously). Numerous studies demonstrate that infants preferentially attend to face-like stimuli rather than to scrambled or inverted faces (Goren et al. 1975, Morton & Johnson 1991). This preference is maintained throughout the lifetime such that, in human adults, attention is rapidly captured by human faces and bodies (Fletcher-Watson et al. 2008, Shah et al. 2013), compared to masked objects masked faces are detected faster and more accurately (Purcell & Stewart 1988), and changes to faces are detected better than to non-face objects (Kikuchi et al. 2009, Salva et al. 2011).

It has been proposed that a subcortical face-detection system, present at birth, underlies this preferential orientation towards faces (Johnson 2005). Critics, however, have argued that humans are simply biased to attend to top-heavy, as opposed to bottom-heavy, stimuli and that faces fall into this top-heavy stimulus category (Simion et al. 2002). More recent research has controlled for "top-heavy" stimuli and still found a significant bias for attending to face-like stimuli in adult participants (Shah et al. 2013, Tomalski et al. 2009). Humans appear to have a specific, and perhaps innate, bias to attend to stimuli that possess the same orientation and polarity as real-life faces.

Deliberate (or endogenous) attention to social stimuli has been much discussed in theories of ASD (Chawarska et al. 2015). Several developmental accounts (e.g. Chevallier et al. 2012, Dawson 1991, Klin et al. 2003, 2015) trace a pathway from a specific reduction in orientating to social stimuli (due to e.g., reduced social motivation, or to problems of attentional disengagement), through reduced exposure to relevant learning opportunities, to poor social cognition (e.g. ToM). Such theoretical accounts underpin a number of prominent intervention approaches for young children with ASD, focused on increasing attention to social stimuli and establishing joint attention.

Perhaps the most pertinent evidence for these cascade theories comes from studies of infants at high genetic risk of autism (those born into families with a child with ASD); to date these show little in the everyday social behavior under 12 months that discriminates those children who will later receive an ASD diagnosis, and in the lab attention abnormalities (evident from c. 6 months) do not appear to be strongly domain-specific (Elsabbagh & Johnson 2016). Claims of sustained abnormalities in attention to social stimuli (e.g. reduced looking to other's eyes, more looking at mouths) in ASD (Klin et al. 2002), did not receive support in a recent review of eyetracking studies in ASD (Guillon et al. 2014), and at least one study suggests that attention to faces in general, and the ratio of eye and mouth fixations, may be differentially affected by ASD and alexithymia (the inability to identify and describe one's own emotional state), respectively (Bird et al. 2011). Interestingly, it may be domain-general properties of ostensibly social stimuli (such as point light displays of biological motion), that determine whether children with ASD pay preferential attention or not; a greater preference for exact predictability or contingency at key stages of development may distinguish ASD from TD children (Klin et al. 2009).

4.4 Dual-process Theories and Social Cognition

A broader issue within the domain specific versus general debate concerns the issue of whether there are two 'types' of social cognition. These types are consistent with classic dual process theories, which posit two systems - one which is cognitively efficient, fast and automatic (System 1 in Kahneman's (Kahneman & Frederick 2002, Stanovich 1999) terminology), while the other is cognitively demanding, slow, controlled, and of limited capacity (System 2). Many instantiations of dual process theory suggest that the fast automatic System 1 is domain-specific, whereas the slow controlled System 2 is domain-general (Evans 2008).

This issue has been discussed extensively within the ToM literature (e.g. Apperly & Butterfill 2009, Butterfill & Apperly 2013). The suggestion of two systems for ToM was prompted by the observation that although typical children below the age of four years of age on average do not pass verbal, explicit tests of ToM (as measured by classic false belief tests), implicit false belief paradigms based on eye gaze behaviour are passed by 18-month-old infants (Onishi & Baillargeon 2005). A dual system view of ToM was supported by the finding that individuals with ASD who were able to pass explicit tests of false belief understanding did not show eye-gaze behaviour consistent with false belief understanding on implicit tasks (Senju et al. 2009).

Several authors have claimed to demonstrate automatic, cognitively efficient ToM in typical adults (where it is often labelled 'implicit mentalizing'), for example Samson et al (2010) introduced the 'Dot Perspective Task' in which participants are presented with an image of a blue room with red dots on the walls. An avatar faces towards one of the walls, and participants

are asked to count the number of dots they can see, ignoring the avatar. Despite this instruction, participants respond faster when the avatar can see the same number of dots they can see. This 'consistency effect' has been interpreted as evidence for automatic mentalizing: that the avatar's visual perspective (i.e. knowledge state) is automatically processed in addition to the participant's own.

The problem with tests of implicit ToM, however, is that it is difficult to establish that the observed effects are a consequence of the automatic representation of mental states (Heyes 2014a,b). For example, Santiesteban and colleagues (Catmur et al. 2016, Santiesteban et al. 2014) demonstrated that the consistency effect could be observed in the Dot Perspective Task when the avatar was replaced with an arrow, a stimulus clearly not appropriate for the attribution of mental states. They argued that the effect observed in the avatar condition was a result of domain-general processes such as attentional orienting, where the avatar's gaze acted a directional cue, rather than the attribution of mental states to the avatar. A similar debate occurred following the publication of another paper claiming that adults automatically represent an avatar's false belief (Kovács et al. 2010). In a replication and extension of this study, Phillips et al. (2015) demonstrated that the effect was due to an experimental confound.

While the debate surrounding the existence of implicit mentalizing continues, the general principle of separating sociocognitive processes into System 1 and System 2 promises to bear fruit. Perhaps all core social abilities could be accomplished via two routes, one being an automatic, cognitively efficient process that relies in part on heuristics/learned associations, and the other a deliberative reasoning process. If it is the case that each aspect of social processing

can be accomplished via either route then the relationship between different social abilities may depend on whether the automatic or deliberative route is used to accomplish a particular social goal (and hence, what type of task provides the relevant evidence on inter-relations). Presumably, any time the rational, deliberative System 2 route is used then performance will, in part, be affected by individual differences in general processes such as working memory, executive function and intelligence, and correlations will be observed between different social abilities. As discussed earlier, if System 1 processes are learned over development then factors that determine learning speed (such as social attention, social reward, and social learning ability) will produce associations in the speed of acquisition or extent of learning in each of these processes. By contrast, if System 1 processes rely on dedicated domain-specific modules, then dissociations between different System 1 social abilities are more likely to be seen.

5. HOW CAN WE MAKE FURTHER PROGRESS?

5.1 Available Methodologies

We began our review of the structure of social cognition by contrasting it with the structure of intelligence. Although many in that field would argue that much is still to be determined, the general methodological approach has been successful. Typically, large numbers of participants complete various tests designed to measure some aspect of intelligence, and statistical techniques such as factor analysis are used to examine the relationships among tests. The result is the identification of a number of factors that explain performance on those tests. Such an approach would be of obvious benefit when it comes to determining the structure of social cognition. For example, in Section 3.2 we hypothesized that a common ability to distinguish and select between representations of the self and others may be recruited by empathy, ToM and imitation

inhibition. One would expect therefore that a factor analysis of tests assessing these abilities would identify a common factor corresponding to this self-other ability. Such techniques could examine the (in)dependence of a large number of tests of social ability, and determine whether there is evidence for factors underlying performance on multiple tests of the sort hypothesized in Section 4, such as social learning ability, social attention, and social motivation.

Such a study, although useful, would not be able to uncover all relationships between different social abilities. One such relationship is where process X is necessary, at a certain developmental stage, in order to develop process Y (the 'cascade' or 'stepping stone' model; **Fig. 2c**). Such a potential relationship could have been uncovered by the study described in Section 3.1 (McEwan et al., 2007) in which imitation was measured at 2 years of age and social ability was measured in the same individuals at 8 years of age. If all of those who could not imitate at 2 years were social impaired at 8 years, then one might conclude that the ability to imitate at 2 is necessary to develop appropriate social ability in later childhood. Of course, we could not make that claim solely based on data from such a cross-lagged design - there may be another factor, process Z, which actually determines social ability in later childhood and which also happens to co-vary with imitation at 2 years of age. Regardless of the inability of cross-lagged designs to demonstrate definitively a causal influence of one process on another, the fact remains that if imitation at 2 years is necessary for appropriate development of other social abilities, then collecting and factor analyzing data from a large group of adults on multiple tests of social ability is unlikely to uncover this developmental relationship; most adults can successfully imitate, leaving little variance in this ability to predict other social abilities.

This problem is an example of a more general problem associated with developmental influences of one process on another. Consider the case of empathy and the recognition of one's own emotions. There are two ways in which the ability to recognize one's own emotion may be necessary for empathy. The first, developmental, account suggests that infants learn to associate the experience of a state, whether pain, sickness, or joy, with the expression of that state in another. For example, the infant falls and is hurt, and caregivers mimic a pained facial expression and vocalise pain. Over repeated painful experiences, learning will result in a link between the feeling of pain in the self and its expression in another. Several theories suggest that this learning is sufficient for empathy (at least for emotion contagion; e.g. Bird & Viding 2014, Heyes & Bird 2007). After these links have been learned, it is possible that recognition of one's own emotion plays no further role in the expression of empathy. Under this model, individual differences in empathy and own-emotion recognition will no longer be correlated, meaning the factor analysis strategy using adult data will erroneously conclude that they unrelated. While potentially true in adulthood, such a conclusion would not capture the necessary role of recognition of one's own emotion in the development of empathy.

In order to postulate a causal connection between two processes such that one can claim that ability in one sociocognitive domain determines ability in another, or that two abilities share common components, one must randomly assign individuals to groups, experimentally increase or decrease social ability in one group and compare this group following the intervention with another who did not receive training. Such studies are not easy to design however; one must be extremely careful in ensuring that the control training is matched in every way with the social cognition training. Although difficult to achieve in practice, such designs are very powerful in

determining causality. They are not a panacea, however, and several factors may limit their use. First, if used in adulthood, they are insensitive to the kind of developmental relationships described earlier (e.g., imitation at 2 years of age relating to ToM in later childhood). Second, unless several such experiments are performed, or extremely subtle (or numerous) control conditions are used, it is hard to determine the process whereby the training is having an effect because it is unclear exactly what is being trained.

Neuroimaging methods, particularly fMRI, have often been used to answer questions relating to the relationship between different socio-cognitive processes. For example, Quirin and colleagues (2013) demonstrated that areas coding for dominance relationships did not overlap with those coding for affiliative relationships. Such evidence of dissociation is powerful if a number of design issues are addressed; given tasks equated for sensitivity and difficulty, reliable dissociations are likely to signal (at least partially) distinct processes. Of course, dissociations cannot be claimed on the basis of one study (a lack of evidence that empathy activates TPJ in one study, is not the same as evidence that empathy does not activate TPJ), but can be made on the basis of multiple studies with appropriate (Bayesian) statistics.

Studies demonstrating *association* between different sociocognitive processes on the basis of shared activation are on less solid ground however. Such studies find that one process activates a network including region A, and another process activates a more or less distinct network which also includes region A. The problem with this logic is that the unit of analysis common in fMRI studies may contain 7-9 million neurons. It is therefore perfectly possible that two processes activate distinct sets of neurons that cannot be resolved with the existing spatial resolution of

fMRI. More promising is a technique known as 'repetition suppression' or 'fMRI adaptation' which takes advantage of the fact that repetition of a particular stimulus, or a particular stimulus class, causes a reduction in the signal measured with fMRI. For example, in order to identify which brain areas encode facial identity one can compare the neural activation elicited by a particular face when it is preceded by the same face, to the neural activation elicited when it is preceded by a different face. If an area shows reduced activation to the repetition of the particular face then it is concluded that the area codes for face identity rather than the mere presence of a face. The cellular mechanisms underlying such reduced activation are at present unclear (Grill-Spector et al. 2006) but the presence of repetition suppression is thought to reflect the activation of the same population of neurons. At present, this technique has been little-used to examine the relationship between different sociocognitive processes but, if the assumption that suppression reflects activation of common neurons holds, it could prove a very powerful technique.

A further class of techniques seeks to find a differential impact of modulators - whether these be drugs, organic or experimental neurological lesions, personality types or neurodevelopmental disorders - with the aim of demonstrating single- or double- dissociations. In general the logic of this approach is simple, if one factor can be shown to modulate sociocognitive process A without affecting sociocognitive process B, and another factor can be shown to modulate process B without affecting A, then we assume A and B are independent. We have already referred to the fact that ASD and psychopathy provide strong evidence for the independence of ToM and empathy; individuals with ASD appear to be impaired at ToM but not empathy, while individuals with psychopathy are impaired at empathy but not ToM (Jones et al. 2010). Such

dissociations may also be observed with organic lesions, where one patient may experience a loss of premorbid ability in a particular social domain while another domain is unimpaired, while another patient may have the opposite pattern of deficits. For example, Calder (1996) reported the case of patient DR who had a specific impairment in the recognition of fear, but was able to recognize facial identity, while Tranel, Damasio and Damasio (Tranel et al. 1995) reported a series of patients with acquired prosopagnosia, a deficit of facial identity recognition, who were still able to recognize emotional facial expressions (including fear) from faces.

While such examples are powerful, their effectiveness rests on the tests of social ability being very finely matched. If one test is speeded and another not, one requires holistic processing and one local, one makes demands on memory and another not, then dissociations may reflect the differential demands of the tests, rather than of the social abilities under test. Furthermore, dissociations observed in patients with psychiatric or neurodevelopmental conditions, or in those with brain lesions, may reflect patterns of compensation (over development or in response to brain injury) within an atypical cognitive system. For example, Brewer et al. (Brewer et al. 2015) demonstrated that emotion recognition and moral reasoning are associated in typical individuals, thought to reflect the fact that moral judgments reflect the combination of emotional processes such as empathy for the victim and the application of socially-agreed rules arrived at through deductive reasoning (Greene et al. 2001, 2004). In individuals with ASD however, these processes were uncoupled, hypothesized to be due to the fact that those with ASD rely less on emotional heuristics in decision-making tasks (di Martino et al. 2008).

5.2 Recommendations for Further Progress

While reviewing the little available literature on the structure of social cognition it became clear that a number of factors may be inhibiting progress in this area. First, the vocabulary of sociocognitive ability is highly variable and nonspecific. Happé and Frith (2013) surveyed a multitude of social abilities, yet this represents only a fraction of the myriad hypothesized social abilities in the literature. The problem is that the relationship between these terms is often not specified, leading to a multitude of terms, which may or may not refer to the same construct. For example, affective theory of mind, emotion contagion, empathy, emotional mirroring, emotion understanding, and emotional resonance all appear to refer to remarkably similar, or the same, processes. It is therefore difficult to integrate all these terms into a factor structure of social cognition when they may be synonyms for a single ability. Adopting an agreed-upon lexicon for aspects of social ability would likely accelerate research in this area, and increase the comprehensiveness and utility of meta-analyses relating to these abilities. Such a lexicon is also likely to increase the consistency with which the results of certain tests are interpreted. For example the Reading the Mind in the Eyes Test (RMET; Baron-Cohen et al. 2001) involves participants being presented with images of the eye region of faces and asked to pick the mental state or emotional term which best describes the image. This task has been claimed to index theory of mind, empathy and emotion recognition, but a clear decomposition of task demands, or evidence of differential relationships to performance on other assays of these processes, is lacking. Adopting an agreed-upon lexicon will allow one to decide whether ToM, empathy and emotion recognition are distinct entities, and then to determine which is tested by the RMET and other commonly-used tasks.

A second, potentially important, distinction to be made when determining the structure of social cognition is between the *ability* to carry-out a social computation and the *propensity* to do so. The paradigmatic case for this distinction is ToM in ASD. When tested on explicit ToM tasks in a laboratory setting, intellectually-able adults with ASD can often perform at the same level as typical adults. In everyday life, however, ASD individuals typically exhibit problems interacting with others, difficulties with pragmatic language understanding, and other impairments thought to result from impaired ToM. Assuming laboratory-based tests are sensitive enough to detect a ToM impairment should it exist, then a potential explanation for this discrepancy is that these adults with ASD are able to use ToM, but have a reduced propensity to do so (see also Cage et al. 2013).

The ability/propensity distinction may interact with our third recommendation, that a distinction should be drawn between System 1 and System 2 social processes. If there really are two routes by which a particular social task can be accomplished, then care should be taken to determine *how* participants are addressing the task; are they using a fast, automatic, heuristic-based process, or are they instead using a slow, deliberative rational route? It may well be that the ability and propensity distinction interacts with the System 1 / System 2 distinction such that differences in propensity reflect the degree to which System 1 processes are automatically engaged during social interaction, whereas ability reflects the degree to which rational deliberative social reasoning can produce accurate results.

Finally, there is growing evidence of significant cultural learning in the development of various social abilities (Heyes 2012b, Heyes & Frith 2014). Exposure to literature (Kidd & Castano

2013) and playing videogames with a narrative storyline (Bormann & Greitemeyer 2015) causes better performance on the RMET, and reading fiction increases self-reported empathy (Bal & Veltkamp 2013). Also, the degree to which mothers use mental state language predicts the development of mental state and emotion understanding in infants from 15 to 33 months (Taumoepeau & Ruffman 2006, 2008). The implication of this research is that tests need to be sensitive to participants' cultural background and developmental history. While this fact has long been acknowledged within social perception research, where there is significant evidence of impaired facial identity recognition with other-race face stimuli for example (Barkowitz & Brigham 1982, Chance et al. 1975, Chiroro & Valentine 1995, Elliott et al. 2013), it is less often appreciated in other areas of sociocognitive research. For example, although my ToM system may function perfectly - in that it enables me to represent the propositional attitudes of others and how attitudes determine their behaviour - if my developmental environment consisted of a restricted range of individuals (with respect to political or religious affiliation, social class, education level, etc.) then I may frequently fail to infer accurately the mental states of others when in more mixed environments. Happé & Frith (1996), for example, suggested that conduct-disordered children from adverse family backgrounds might have developed a 'Theory of Nasty Minds'. In everyday life then, social abilities such as ToM, emotion recognition, and empathy may be determined by the *range* of minds of minds one has encountered previously and is therefore able to model, and the accuracy with which one can determine which model to apply to a particular individual.

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Uses dissociable deficits revealed by developmental disorders to outline a putative network of socio-cognitive abilities.

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Argues that social learning relies on the mechanisms that support individual / asocial learning.

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Training the inhibition of imitation improved perspective taking abilities arguably via training of self-other control.

FUTURE ISSUES

- An agreed-upon lexicon for sociocognitive processes would accelerate research.
- New methodologies are necessary to uncover the factor structure of social cognition (e.g. large-scale normative factor analysis; randomised modulator/intervention designs; 'fMRI adaptation').
- Conceptual and empirical distinctions are needed: e.g., ability versus propensity for specific social processing; dual-systems for social cognition (fast and automatic versus slow and effortful).

GLOSSARY

1. Social cognition – the processing of stimuli relevant to understanding agents and their interactions.
2. 'Theory of mind' (aka 'mentalising') – representing one's own and others' mental states.
3. Empathy – another's affective state mirrored in the self (with recognition that the other is the source of one's state, in some accounts).
4. Alexithymia - the inability to identify and describe one's own emotional state.
5. Neurotypical – an individual who does not display ASD or other neurologically atypical patterns of thought or behaviour.
6. Biological motion - the motion profile of other animate beings.
7. Autism Spectrum Disorder (ASD) - a neurodevelopmental disorder characterized by impaired communication and social interaction, and restricted and repetitive interests.
8. Domain-specific – psychological/neural mechanisms dedicated to the processing of specific content (e.g. social stimuli and information).
9. Developmental cascade – a sequential model in which the development (or absence) of one process is necessary for the development (or absence) of later processes.
10. Imitation – observation of an action causes the performance of a topographically similar action.

ABBREVIATIONS / ACRONYMS

1. Theory of Mind (ToM)
2. Autism Spectrum Disorder (ASD)
3. Medial Prefrontal Cortex (mPFC),
4. Temporoparietal Junction (TPJ)
5. Transcranial Magnetic Stimulation (TMS)
6. Transcranial Direct Current Stimulation (TDCS)
7. Reading the Mind in the Eyes Test (RMET)

Figure captions

Fig. 1

Necessary sub-components: Schematic illustration of two ways in which sociocognitive abilities may be related. 1) Some processes may constitute necessary sub-components of others (e.g. emotion recognition is a necessary sub-component of empathy). 2) Seemingly distinct socio-cognitive functions (e.g. empathy, false belief understanding and control of imitation) may recruit common sub-components (e.g. self/other control and social perception). Here ovals illustrate common processes and rectangles represent distinct processes relating to empathy (pink route), false belief understanding (blue route) and control of imitation (green route).

Fig. 2

A & B - Common factors: Some sociocognitive abilities are related due to a common factor which is important at some point in the development of each ability. For example, social attention may be important for the development of empathy (A) and Theory of Mind (B); individuals who can be characterized as "high social attenders" would develop these abilities more quickly than "low social attenders", resulting in a correlation between the two abilities. One should note that if an ability has a critical period (not shown) then an early deficit on one ability (e.g. social attention) may result in a life-long impact on another ability (e.g. empathy). Though these examples are hypothetical, they serve to illustrate the importance of accounting for development when assessing the factor structure of social cognition: in certain periods of development abilities that require common processes (e.g. empathy and theory of mind) may be correlated, but this correlation may vanish at different developmental stages.

C – '**Cascading' or 'Stepping Stone' Model**: Some sociocognitive abilities are related via what can be called a 'cascading' or 'stepping stone' effect whereby the development of one ability (e.g. face processing) acts as a stepping stone for the further development of other abilities (e.g. emotion recognition and empathy).