COGNITIVE NEUROSCIENCE OF HUMAN SOCIAL BEHAVIOUR

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We are an intensely social species — it has been argued that our social nature defines what makes us human, what makes us conscious or what gave us our large brains. As a new field, the social brain sciences are probing the neural underpinnings of social behaviour and have produced a banquet of data that are both tantalizing and deeply puzzling. We are finding new links between emotion and reason, between action and perception, and between representations of other people and ourselves. No less important are the links that are also being established across disciplines to understand social behaviour, as neuroscientists, social psychologists, anthropologists, ethologists and philosophers forge new collaborations.

COGNITIVE NEUROSCIENCE

COGNITIVELY IMPENETRABLE
Processes that are not influenced strategically by cognition. They cannot be influenced at will, and their engagement is beyond our control.

SELF-REGULATION
The ability to control one's behaviour effortfully and often in opposition to emotional drive (for example, controlling an anger outburst). Most prominent in adult humans, self-regulation depends on regions in the prefrontal cortex.

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A new field has emerged to investigate the cognitive neuroscience of social behaviour, the popularity of which is attested by recent conferences, special issues of journals[1,2] and by books[3,4]. But the theoretical underpinnings of this new field derive from an uneasy marriage of two different approaches to social behaviour: sociobiology and evolutionary psychology on the one hand, and social psychology on the other. The first approach treats the study of social behaviour as a topic in ethology, continuous with studies of motivated behaviour in other animals. The second approach has often emphasized the uniqueness of human behaviour, and the uniqueness of the individual person, their environment and their social surroundings.

These two different emphases do not need to conflict with one another. In fact, neuroscience might offer a reconciliation between biological and psychological approaches to social behaviour in the realization that its neural regulation reflects both innate, automatic and COGNITIVELY IMPENETRABLE mechanisms, as well as acquired, contextual and volitional aspects that include SELF-REGULATION. We share the first category of features with other species, and we might be distinguished from them partly by elaborations on the second category of features. In a way, an acknowledgement of such an architecture simply provides detail to the way in which social cognition is complex — it is complex because it is not monolithic, but rather it consists of several tracks of information processing that can be variously recruited depending on the circumstances. Specifying those tracks, the conditions under which they are engaged, how they interact, and how they must ultimately be coordinated to regulate social behaviour in an adaptive fashion, is the task faced by a neuroscientific approach to social cognition.

Social cognition and emotion

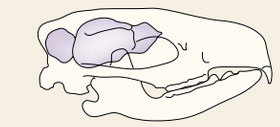
What is social cognition? If the social is ubiquitous, we face the problem of including all aspects of cognition as social. If it is special, we have to explain why and how (BOX 1). As a matter of practice, social brain science has indeed carved out a restricted domain of cognition. The bulk of studies emphasize motivational and emotional factors. Whereas other aspects of cognition — such as language, for example — contribute substantially to the regulation of social behaviour, the intuition has been that emotion stands in a privileged position. This intuition has its basis in our observations of other species and of human infants, whose social behaviour seems to be tightly coupled to emotion — a coupling that is heavily regulated in adults. But the intuition also has a functional explanation. Emotions can be thought of as states that coordinate homeostasis in a complex, dynamic environment; in so far as one aspect of the

Box 1 | Are our brains specialized for social cognition?

Brains and social behaviours vary across different mammalian species. Primitive insectivores (for example, hedgehogs) already show tightly regulated maternal behaviours that allow extended development of their offspring; non-human primates (for example, chimpanzees) live in extended societies of a few dozen subjects; and modern humans have created societies that encompass millions of interacting people.

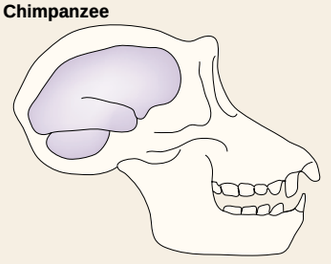
There is no question that humans are exceedingly skilled at large-scale social interaction, but it remains a puzzle how best to account for such abilities. Under one hypothesis[149], the competition for social skills led to the evolution of cognitive mechanisms for outsmarting others[150], and fuelled the expansion of the human brain and perhaps the elaboration of certain neural systems[151]. In support of this idea, there is a correlation across primate species between the size of their social group and the relative volume of neocortex[149].

Hedgehog

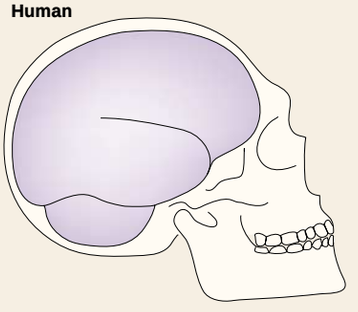


Courtesy of Laura Roberts

Chimpanzee



Human



environment is social, emotions will participate in regulating social behaviour. In fact, one class of emotions — the so-called social or MORAL EMOTIONS — serve specifically in this capacity and probably guide altruistic helping[5] and punishment[6].

Most structures that have been shown to be important in processing emotions have therefore also turned out to be important for social behaviour. These include: first, specific regions in higher-order sensory cortices; second, the amygdala, the ventral striatum and orbitofrontal cortex; and third, additional cortical regions such as the left prefrontal, right parietal, and anterior and posterior cingulate cortices. It is possible to relate these three sets of regions to three different sets of processes. Higher-order sensory cortices are involved in the perceptual representation of stimuli and their constituent features. The amygdala, striatum and orbitofrontal cortex mediate an association of this perceptual representation with emotional response, cognitive processing and behavioural motivation. Higher cortical regions are then involved in the construction of an internal model of the social environment, involving representation of other people, their social relationships with oneself, and the value of one's actions in the context of a social group. To some extent, these three sets of processes build on one another, although their interactions are complex (FIG. 1). For organizational purposes, the sections that follow consider these neural structures roughly in the same order as above.

Perception of social signals

A large variety of stimuli are available for investigating social cognition (FIG. 2). Many recent studies on this topic have started, so to speak, at the input end — by showing pictures of social relevance to subjects (often under passive viewing conditions), and associating differences in the social content of stimuli with differences in the neural structures that are engaged in their processing. This work — primarily functional imaging studies — has found covariances between stimulus dimensions and brain structures. However, it is important to keep in mind that lesion studies are also needed to further elucidate a causal role for a given structure in a neural system (that is, to confirm that its role is essential). These lesion data are often lacking at this stage. It is also important to note that several of the structures that appear in this section will reappear later, reflecting their roles in implementing several social processes.

Investigations have focused on the visual modality in primates, although a few studies have examined other sensory modalities as well (BOX 2). Social visual signals include information about the face (such as its expression and the direction of gaze), as well as about body posture and movement. Although prototypical facial expressions reliably signal the so-called basic emotions such as fear or happiness, human viewers are also surprisingly adept at making reliable judgements about social information from impoverished stimuli, such as faint changes in facial expression[7], or a few seconds of

MORAL EMOTIONS
Guilt, shame, embarrassment, jealousy, pride and other states that depend on a social context. They arise later in development and evolution than the basic emotions (happiness, fear, anger, disgust, sadness) and require an extended representation of oneself as situated within a society. They function to regulate social behaviours, often in the long-term interests of a social group rather than the short-term interests of the individual person.

MODULES
Functional and/or anatomical components that are relatively specialized to process only certain kinds of information. Modules were originally thought of as cognitively impenetrable and informationally encapsulated (having restricted access to only certain information). Although most people do not view modules in such strict terms, there is evidence of domain-specific processing that is specialized for specific ecological categories (such as faces and social contract violations), although there is debate on this issue.

EVENT-RELATED POTENTIALS
(ERPs). Electrical potentials that are generated in the brain as a consequence of the synchronized activation of neuronal networks by external stimuli. These evoked potentials are recorded at the scalp and consist of precisely timed sequences of waves or 'components'.

MAGNETOENCEPHALOGRAPHY
(MEG). A non-invasive technique that allows the detection of the changing magnetic fields that are associated with brain activity, similar to the detection of changing electric fields measured by ERPs.

CATEGORIZATION
Stimulus categories function to group together stimuli to which a similar behavioural response should be mounted. Coarse, generic categorization (for example, a dog as an animal) is superordinate; subordinate categorization includes basic-level (a dog as a dog) and unique categories (a dog as your own pet).

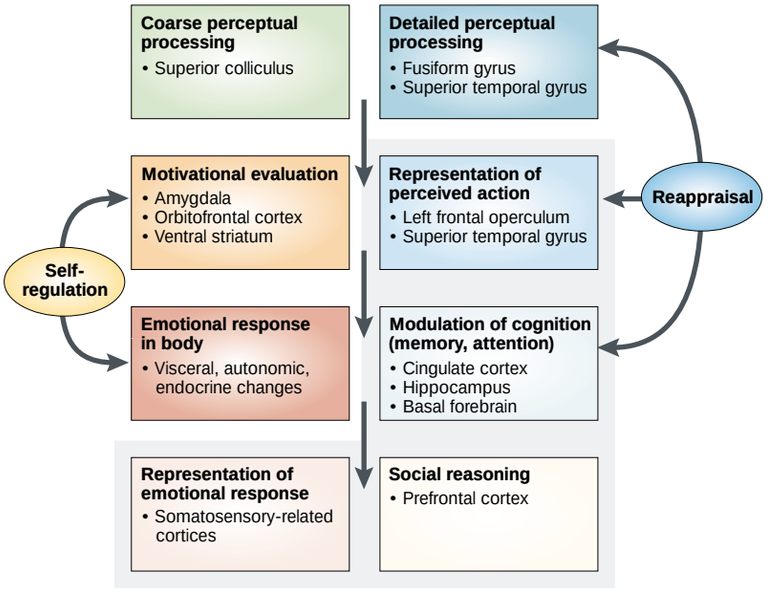


Figure 1 | **Processes and brain structures that are involved in social cognition.** It is possible to assign sets of neural structures to various stages of information processing, as I argue in this review. However, the flow of social information defies any simple scheme for at least two reasons: it is multidirectional and it is recursive. A single process is implemented by a flexible set of structures, and a single structure participates in several processes, often during distinct windows of time. Processing routes differ in terms of their automaticity, cognitive penetrability, detail of the representations they involve and processing speed. The structures outlined in this figure share some core features of a social information processing system: selectivity (they make distinctions between different kinds of information), categorization and generalization, and the incorporation of past experience. Several of the components of social cognition (inside the grey shaded region) contribute to social knowledge. Reappraisal and self-regulation are particular modes of feedback modulation whereby evaluation and emotional response to social stimuli can be volitionally influenced.

full-body interpersonal interactions[8]. Not only are we exceedingly sensitive to the social signals themselves, but we are also sensitive to the details of the context in which they occur.

Regions of non-primary sensory cortices might be relatively specialized to process certain socially relevant stimulus attributes. The best evidence comes from the study of faces, for which higher-order visual cortices can be regarded as an assembly of MODULES that process distinct attributes, as borne out by various lesion studies, scalp and intracranial recordings, and functional imaging data. The results point to a role for the fusiform gyrus in processing the structural, static properties of faces, which are reliable indicators of personal identity. By contrast, regions more anterior and dorsal in the temporal lobe (such as the superior temporal gyrus and sulcus) are involved in processing information about the changeable configurations of faces, such as facial expressions, and eye and mouth movements[9,10] (FIG. 3). Activation along the superior temporal sulcus and gyrus has been found when subjects view stimuli depicting biological motion, such as gaze shifts[11,12] and mouth movements[13]. Processing in this region might draw on both dorsal and ventral visual streams in integrating shape and motion information[14], and it might reflect a comparison of the observed action with the viewer's simulation of it[15]. The fusiform gyrus, the superior temporal gyrus and other less well specified regions of occipitotemporal cortex could therefore be thought of as an interconnected system of regions that construct a spatially distributed perceptual representation of different aspects of faces. There is good evidence that activation in all of these regions can be modulated by attention[16] and by the context in which the visual social signal appears[17,18].

The above anatomical investigations are complemented by data on the timing of face processing. Studies using EVENT-RELATED POTENTIALS (ERPs) and MAGNETOENCEPHALOGRAPHY (MEG) show that some coarse CATEGORIZATION of face features, such as gender and emotion, can occur at latencies as short as 100 ms (REFS 19–22). Peak activity that is related to face-specific processing near the fusiform gyrus is seen around 170–200 ms (REF. 23). Whereas the construction of a detailed structural representation of the face therefore seems to require about 170 ms, some rapid, coarse categorization can occur with substantially shorter latencies, presumably indicating coarse perceptual routes that are parallel to a full structural encoding of the stimulus. A recent study[24] investigated these different levels of categorization in detail and corroborated the idea of a fast, superordinate categorization of faces at a relatively short latency (around 100 ms). This categorization was followed by a subordinate categorization with a longer latency (around 170 ms), which was sufficient to discriminate individual identity. Similar evidence for the extraction of information at subordinate levels with increasing processing time has been provided by single-unit studies of face-selective cells in the monkey inferotemporal cortex[25]. At least three non-exclusive mechanisms could implement such computations: initial feed-forward processing followed by top–down modulation from higher regions, progressive processing within a region, or iterative cycles of processing between a region and others (either 'lower' or 'higher' in a processing hierarchy).

A growing body of work has used visual stimuli that signal biological motion (FIG. 2) to study social cognition. Social psychologists first showed our propensity to make social inferences from visual motion of abstract shapes in the 1940s (REFS 26,27), and recent studies indicate that specific movement cues might generate attributions of ANIMACY, intentionality and AGENCY[28,29]. Visual motion stimuli elicit attributions of intentionality and animacy in infants, and robustly elicit intentional, emotional and personality attributions in adults, even when only static depictions of their trajectories are shown. More specific information about the movements of a human body are offered by POINT-LIGHT DISPLAYS[30], which generate exceptionally robust shape-from-motion cues that allow the recognition of identity[31], gender[32], emotions[33] and personality traits[34]. In line with the role of the superior temporal cortex in processing dynamic aspects of faces, this region is also activated by viewing biological motion in whole bodies[35] or their point-light displays[36,37], and by more abstract movements of geometric shapes[38,39].

ANIMACY
The subjective impression that a stimulus is alive.

AGENCY
The subjective impression of a willful, goal-directed action.

POINT-LIGHT DISPLAYS
Visual motion stimuli created by attaching small lights to an actor's body joints and filming the person moving in an otherwise dark room. Although they seem random when static, the biological motion of the lights immediately generates the compelling perception of a person moving about the room.

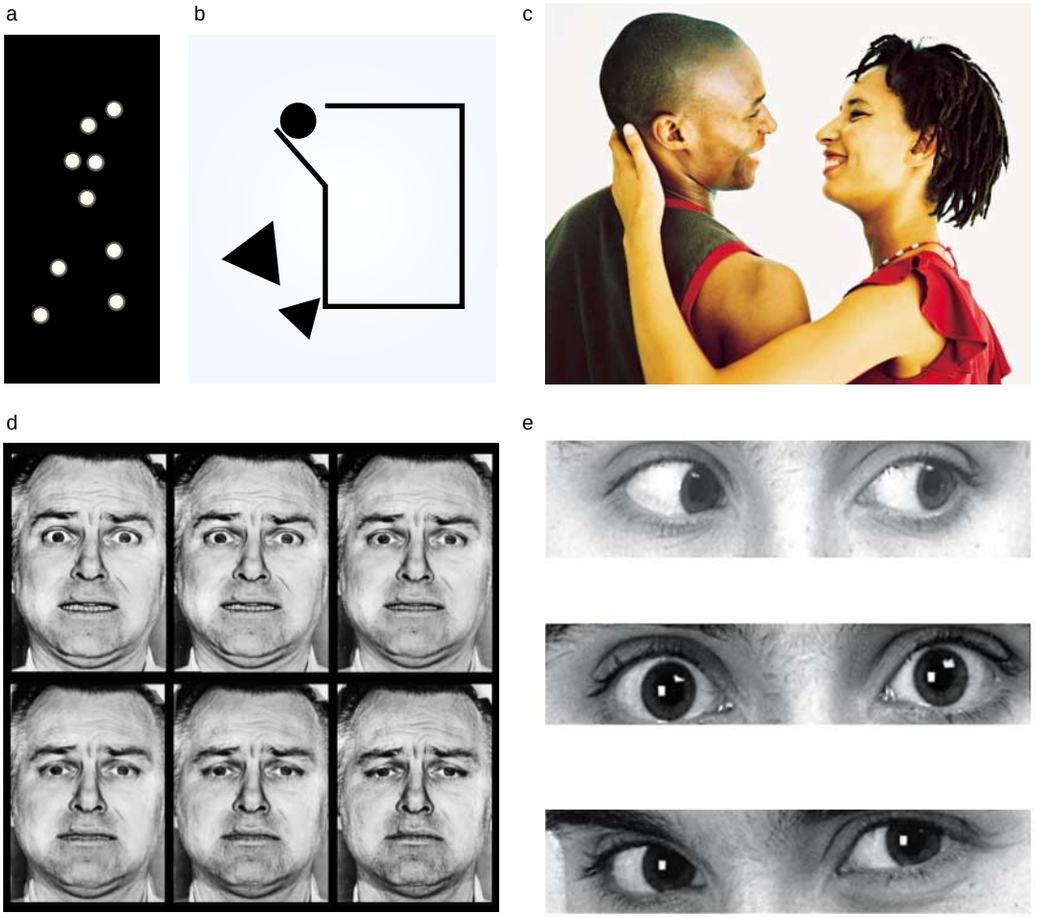


Figure 2 | **Visual stimuli for investigating social cognition.** These range from **a** | point-light walkers and **b** | dynamic geometric figures, to **c** | actual human social interactions. Facial expressions have been one of the most commonly used stimuli. **d** | They can be morphed from, say, fear to sadness and **e** | their eye region can signal specific social information, such as guilt, fear or flirtatiousness. Stimuli are from sets developed by: F. Heider (**b**), P. Ekman (**d**) and S. Baron-Cohen (**e**).

Box 2 | Other sensory modalities used to study social cognition

Most studies on social cognition have used visual stimuli, but it is clear that real-life social interactions draw on additional modalities. Whereas touch is an important social communication channel in other mammals, in modern humans it is relatively restricted to those with whom we have the most intimate relationships. A recently described distinct neural pathway of slow-conducting, C-afferent fibres that convey information about pleasant, light touch to the insula could underlie processing of social somatosensory signals, such as a caress[152].

The sense of smell provides powerful social signals in other mammals but, again, it seems to be less important in humans. Laboratory studies have found influences of odorants on human physiology, but the effects of odours on social behaviour are less clear. Whereas the orbitofrontal cortex and the amygdala are activated by the emotional quality of odours in humans[153,154], and pheromones differentially activate the human hypothalamus[155], the links of these findings to actual social behaviour remain unclear.

Audition provides important social signals in addition to language. The intonation of speech — prosody — can signal various emotions, and is recognized using some of the same structures that we use for recognizing facial expressions[156]. Music is an especially intriguing stimulus, as it might serve a social function that is not found in other animals, and it has been shown to elicit intense emotional responses that activate the orbitofrontal cortex, the insula and the amygdala[157].

These activations probably reflect the role of the superior temporal cortex in processing information about biological motion, on the basis of which we make social attributions.

From perception to judgement

Several brain regions are activated not only as a function of properties that are inherent to the stimuli, but also as a function of the psychological judgements that we make about them. In a sense, the influence of such judgements reflects a progressive decoupling from responses that are dictated by the stimulus itself to information that is generated by the brain through associations and inferences. The amygdala is one structure that is anatomically positioned to participate in such post-perceptual processing, as it receives highly processed visual information from the anterior temporal cortices, and stores codes for subsequent processing of such perceptual information in other brain regions. In this way, it can influence memory, attention, decision making and other cognitive functions on the basis of the social significance of the stimuli that are being processed.

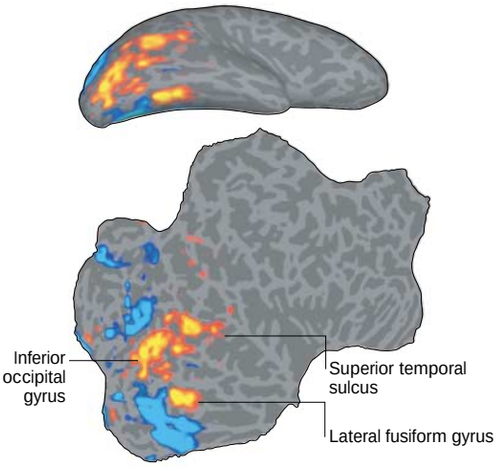


Figure 3 | **Activation in visual cortices to viewing faces.** Changeable, dynamic aspects of faces, such as expression and gaze, activate the superior temporal sulcus, whereas static aspects activate the fusiform gyrus. The top panel shows these activations on a human brain smoothed to reveal both sulci (darker grey) and gyri. The bottom panel shows a flattened representation of the same data. Data were generated by contrasting the activations to viewing faces with those to viewing houses (orange, greater activation to faces; blue, greater activation to houses). Modified, with permission, from REF. 10 © (2000) Elsevier Science.

The bulk of research on the human amygdala has used emotional facial expressions as stimuli and has pointed most consistently to this region being involved in the processing of fear and related emotions[40–42], although recent evidence indicates that its role is probably much broader[43,44]. Functional imaging studies show processing at several stages: a rapid, automatic evaluation and tagging of stimuli for further processing[16], feedback modulation of attentional processing in visual cortices[45], and modes of processing that are subject to self-regulation and volitional guidance[46,47]. The first and last of these stages show complementary roles for the amygdala, probably operating at complementary timescales. On the one hand, some amygdala activation is seen early[48], regardless of the conscious perception of the stimulus (for example, in response to subliminal stimuli[49,50] or in patients with BLINDSIGHT[51] or hemispatial NEGLECT[52]), and regardless of attention allocation in some tasks[16]. On the other hand, effortful self-regulation of the emotions induced by stimuli[47], REAPPRAISAL of their emotional importance[46] and difficult attentional tasks[53], all modulate amygdala activation. These findings urge caution in the rigid assignment of cognitive processes to neural structures, because it is probable that a given structure participates in several processes, depending on the time at which its activity is sampled and on the details of the task and context. It is conceivable that the amygdala participates both in the initial, rapid evaluation of the emotional significance of stimuli, and in a later assessment within a given context and goal.

BLINDSIGHT
The ability of a person with a lesion in the primary visual cortex to reach towards or guess at the orientation of objects projected on the part of the visual field that corresponds to this lesion, even though they report that they can see nothing in that part of their visual field.

NEGLECT
A neurological syndrome (often involving damage to the right parietal cortex) in which patients show a marked difficulty in detecting or responding to information in the contralesional field.

REAPPRAISAL
Reinterpretation of a situation to assign it a different value. Whereas reappraisal changes emotional response by changing one's perception of the stimulus, other strategies of self-regulation directly modulate emotional response despite one's original perception.

POLYMORPHISM
The simultaneous existence in the same population of two or more genotypes in frequencies that cannot be explained by recurrent mutations.

Judging race, trustworthiness and attractiveness

Beyond its role in recognition of basic emotions, the amygdala is involved in more complex social judgements. For example, it shows differential habituation of activation to faces of people of another race[54], and its activation has been found to correlate with race stereotypes of which the viewer might be unaware[55]. However, the role of the amygdala in processing information about race is still unclear. Other brain regions in the extrastriate visual cortex are also differentially activated as a function of race[56], and lesions of the amygdala do not seem to impair race judgements[57].

Other kinds of social judgement also seem to involve the amygdala. In one study, patients with bilateral amygdala damage were found to be impaired in judging how much to trust another person from viewing their face. They all judged other people to look more trustworthy and more approachable than did normal viewers[58], a pattern of impairment that is also consistent with the often indiscriminately friendly behaviour of such patients in real life (FIG. 4a). The role of the amygdala in processing stimuli related to potential threat or danger therefore extends to the complex judgements on the basis of which we approach or trust other people.

These lesion studies have been complemented by functional imaging studies on the role of the amygdala in judging trustworthiness (FIG. 4b). When normal subjects view faces of people that look untrustworthy, activation is found in the superior temporal sulcus, the amygdala, the orbitofrontal cortex and the insular cortex[59], perhaps outlining a sequence of processes that encompass perception, judgement and aspects of emotional response. Interestingly, some activation of the amygdala by untrustworthy-looking faces is independent of factors such as gender, gaze, race or emotional expression of the face[59]. Given that much of the variance in the physical dimensions of different faces can be eliminated yet still produce amygdala activation, it is possible to assume that this activation reflects the judgements and inferences that subjects make about the face, rather than its perceptual properties. An important future direction will be to examine the variance in viewers' personality traits in these social judgements, as has been done in two recent studies correlating amygdala activation to emotional expressions with viewers' extraversion[60] or anxious temperament due to a POLYMORPHISM in the serotonin transporter promoter[61]. To the extent that the amygdala activation covaries with differences in the personality of the viewer, rather than the physical composition of the stimulus, we can conclude that we are tapping processes more distal to perception and closer to judgement, decision making, and the interpersonal behaviours that are based on them.

Another class of social judgement that we make from faces is attractiveness, which can be manipulated by specific properties of faces. For instance, faces are perceived to look more attractive the more average or symmetrical they are, or with greater exaggeration of robusticity and NEOTENY features, all of which have been proposed to signal differential fitness. Moreover, such preferences by women can vary across different phases

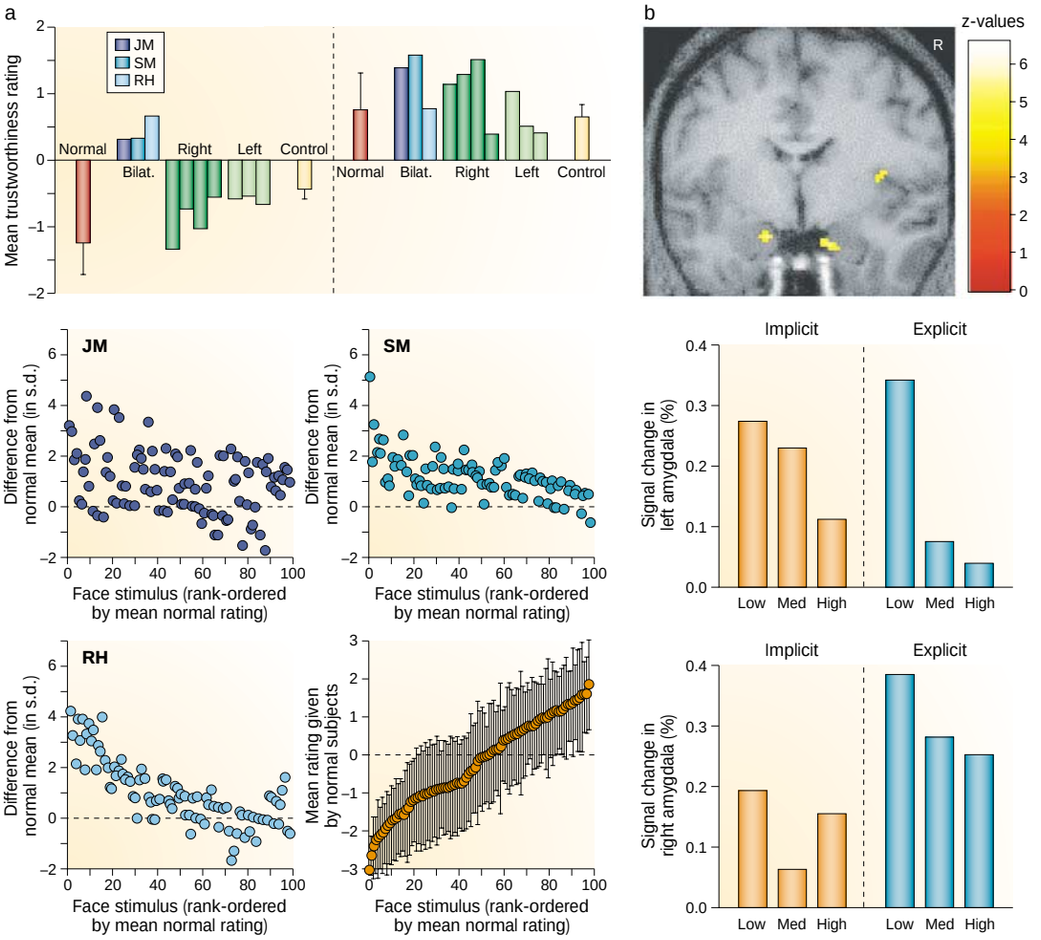


Figure 4 | **Investigating social judgement with two different methods.** Investigations of the neural basis of judging trustworthiness have yielded convergent results from functional imaging studies in normal people, and from lesion studies in neurological patients. **a** | Evidence from lesion studies. Bilateral damage to the amygdala selectively impairs the ability to judge untrustworthiness of faces. These data, from three patients (JM, SM and RH), show that the amygdala is not only involved when we normally make social judgements, but that amygdala dysfunction precludes normal social judgement. This does not mean that the amygdala is sufficient for judging trustworthiness, but that it is necessary. Bilat., bilateral amygdala damage (n = 3); Control, brain-damaged controls with no damage to the amygdala (n = 10, standard error shown); Left, unilateral left amygdala damage (n = 3); Normal, neurologically normal controls (n = 46), standard deviation shown; Right, unilateral right amygdala damage (n = 4). The lower panels show individual scores from the three patients and the mean rating given by normal subjects. Modified, with permission, from *Nature* REF. 58 © (1998) Macmillan Magazines Ltd. **b** | Evidence from functional imaging. The top image shows the activation in the amygdala observed when viewing untrustworthy faces is contrasted with viewing trustworthy faces. The z-values (colour scale) observed in the amygdala correspond to $p < 0.025$. The bar graphs below show the activation in the left and right amygdala for those faces that received the lowest (Low), medium (Med) or highest (High) ratings of trustworthiness. These activations were measured under two task conditions: an implicit task in which viewers were asked to judge the gender of the face, and an explicit task in which viewers were asked to judge the trustworthiness of the face. R, right hemisphere. Reproduced, with permission, from *Nature Neuroscience* REF. 59 © (2002) Macmillan Magazines Ltd.

of the menstrual cycle[62], as do other aspects of their categorization of men[63], possibly providing a link between mate preference and probability of conception. Judgements of attractiveness can reflect both aesthetic judgements (for example, males can judge faces of both males and females to look beautiful), as well as motivational aspects (for example, heterosexual males prefer to look at beautiful female faces rather than at beautiful male faces). These two aspects have been dissociated in functional imaging studies[64]. The motivational aspects of facial attractiveness activate the ventral striatum[64,65] and the orbitofrontal cortex[66]. These structures probably have a broad role in processing the motivational properties of stimuli. For example, they are also activated when males find pictures of sports cars more rewarding than pictures of limousines or small cars[67].The ventral striatum and the orbitofrontal cortex are reciprocally connected with the amygdala; all three structures can be thought of as components of a neural system that links sensory representations of stimuli with the social judgements we make about them on the basis of their motivational value. Given that the same

NEOTENY
The retention of juvenile characteristics in the adults of a species.

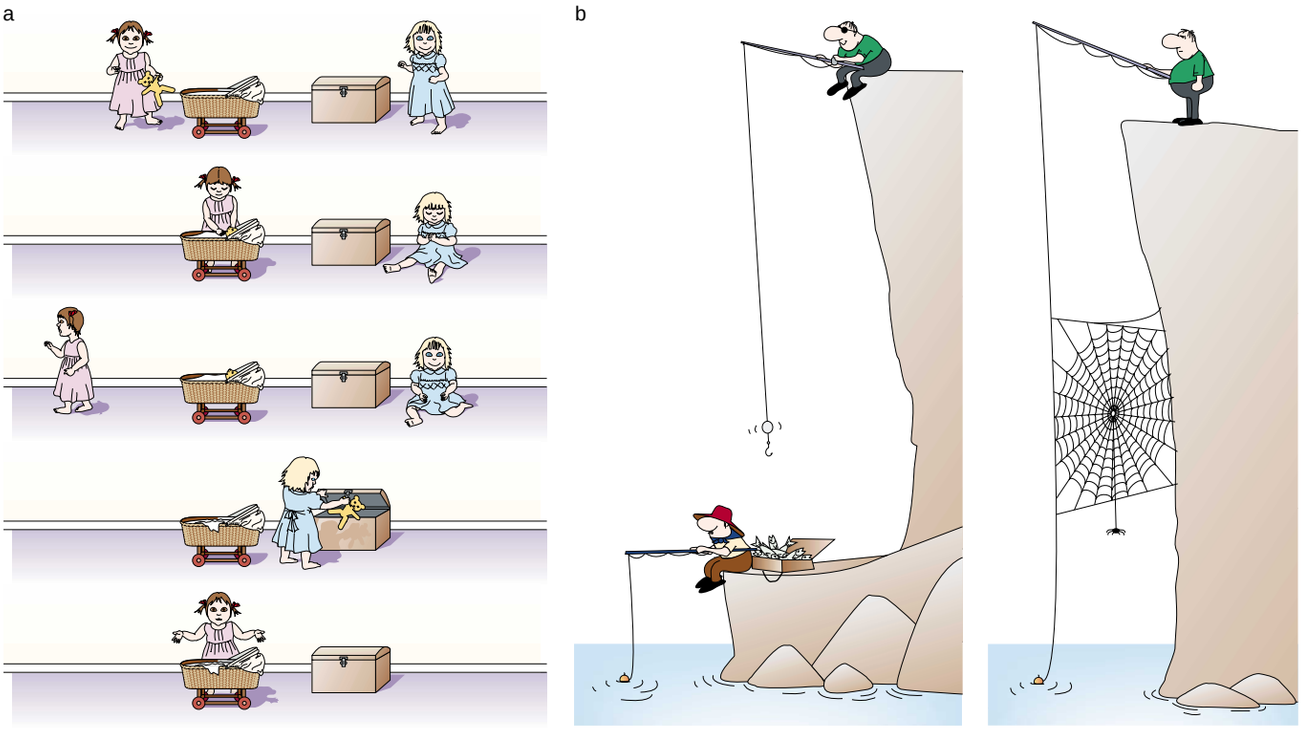


Figure 5 | **Investigating theory of mind. a** | Sally-Ann task. Schematic of the scenario that is shown to infants and children to assess theory-of-mind abilities, specifically the capacity to attribute false beliefs. Sally has a pram and Ann has a box. Sally puts a toy into her pram, and then she goes out for a walk. While she is outside, Ann takes the toy from the pram and puts it into her own box. When Sally comes back, where will she look for the toy? Normal children of four years of age and older answer that Sally will look inside her pram, because that is where she (falsely) believes the toy is. **b** | Theory-of-mind from cartoons. Assessing theory of mind in adults without ceiling effects is more difficult and various tasks have been used. This figure shows examples of cartoon stimuli in which understanding the joke depends on the ability to attribute mental states to others (left) or does not (right). Contrasting these two kinds of cartoons results in brain activation that reflects engagement of theory-of-mind processes, specifically an activation in medial prefrontal cortex (**c**). Panel **c** reproduced, with permission from REF. 71 © (2000) Elsevier Science.

structures that mediate social judgements also mediate basic reward processing, we are led to question whether the former might be reducible to the latter, an issue that I take up in the concluding section of this review.

Thinking about other people

Several of the processes discussed in the preceding section involve more than perception; they bring in additional information beyond what is conveyed by the stimulus to guide our social decisions and judgements about it. Primates, and especially humans, stand out in their ability to take into account what others are thinking — an ability that requires representing what might be going on in other people's minds. A varied collection of processes comprise such higher-level manipulation of social information.

Theory of mind. Abilities that have been dubbed 'theory of mind'[68] allow us to attribute mental states to other people[69]. Attributions of beliefs, specifically false beliefs, to other subjects have been particularly studied (FIG. 5a). Such abilities, which emerge at about four years of age, might be unique to humans, and might be assembled out of a collection of more basic skills by which we assign animacy, actions, goals and intentions to stimuli, an issue that has seen intense recent investigation using visual motion stimuli[70]. In addition to the reliable activation of superior temporal gyrus that I mentioned earlier, several functional imaging studies have shown activation of the medial frontal lobe and inferior parietal lobule when people view visual motion[38,71,72] or gaze stimuli[73] that signal such directed mental states.

Although there is convergent evidence that theory-of-mind abilities emerge in a coordinated fashion during development, so far there is only preliminary evidence to indicate that they are a neuroanatomical package. The evidence for a role for the amygdala in theory-of-mind abilities comes from a small number of patients with amygdala lesions[74,75]. A single functional imaging study has argued for amygdala activation in a theory-of-mind task requiring judgements about facial expressions[76], and another study has found impairments after amygdala damage using the same stimuli[77]. The evidence is stronger for the medial prefrontal cortex, as several functional imaging studies have found that it is activated when subjects perform theory-of-mind tasks[71,78,79] (FIG. 5c). In addition, a few studies have found that patients with damage to the frontal lobes are impaired on theory-of-mind tasks[69,80]. Furthermore, there is some evidence that the role of the medial prefrontal cortex in theory-of-mind tasks can be dissociated from its broader role in behavioural control and executive function that is also engaged by most of the tasks that are commonly used[81,82].

Rather than attempting to assign the whole set of theory-of-mind abilities to a particular neural structure or system, it might be more promising to explore the dependency of specific components of this ability on specific neural structures. In one study, it was found that damage to orbitofrontal cortex impaired the ability to detect a *faux pas*[83], perhaps indicating that this brain region contributes to our understanding of other people in part by engaging the emotions and feelings that accompany social interaction. In support of this idea, it was found that appreciation of humour[84], social-norm transgression resulting in embarrassment[85], viewing of erotic stimuli[86] and elicitation of other moral emotions[87], all activate the medial prefrontal cortex. The role for the medial orbital and anterior cingulate regions in monitoring and regulating social emotions is consistent with their activation during interactions between attention, awareness and emotion[88–90].

The data can be interpreted along two different directions: the specialization of prefrontal cortices for aspects of social cognition, or the reliance of social cognition on more general resources that are provided by this region of the brain. In line with the second interpretation, sectors of prefrontal cortex seem to be crucial for integrating the allocation of cognitive resources on the basis of automatic emotional evaluation and volitional, effortful direction[90,91]. These mechanisms might therefore reflect aspects of a more general function in regulating the fit between goals and behaviour. There could then be specific examples of such domain-general processing on which social behaviour draws: contextual inhibition by prefrontal cortex of emotional responses triggered by the amygdala[92], or response inhibition and reversal in the face of a changing social context[82].

In line with the alternative interpretation that sectors of the prefrontal cortex are specialized for processing social information, medial and orbital prefrontal cortices have been linked to the regulation of interpersonal relationships, social cooperativity, moral behaviour, and social aggression[93–96]. In this case, its role has been stressed particularly in the context of social development and its pathologies (see the section on neuropsychiatry below). It could be that the integration of information about other people and oneself[97], and the social relationship between the two, are the hallmarks of medial prefrontal processing.

Many of the same stimuli that engage the superior temporal gyrus, and lead viewers to attribute actions, intentions and goals, also activate regions of the neocortex that are involved in representing actions[70]. These regions include premotor- and somatosensory-related cortices — the efferent and afferent sides of actions, respectively. A series of recent studies have investigated the role of the right somatosensory-related cortices and the left premotor cortex in making emotional and personality attributions from point-light displays and movements of geometric shapes. Damage in both regions impairs the ability to make such attributions[98].

Simulation. There is a rapidly growing literature supporting the idea that we understand other people's behaviour, in part, by simulation[99]. Observing another person's actions results in desynchronization of motor cortex activity measured with MEG[100]. Imitating another subject's actions through observation activates the premotor cortex in functional imaging studies[101]; such activation is somatotopic with respect to the body part that is observed to perform the action, even in the absence of any overt action on the part of the observing subject[102]. In fact, in both humans[103] and monkeys[104], so-called 'mirror neurons' have been discovered. These neurons respond both when the subject is doing something specific, and when he or she observes another person doing the same thing. Damage restricted to somatosensory cortex impairs the ability to recognize complex blends of emotions in facial expressions[105] (FIG. 6), and there is an association between the impaired somatic sensation of one's own body and the impaired ability to judge other people's emotions[105]. Functional imaging studies also support a role for right somatosensory-related cortices in representing the actions that we observe other people performing, as being distinct from those that we perform ourselves[106].

So, there is good evidence that we can figure out how others are feeling, what they intend and how they are likely to act, in part by putting ourselves in their shoes, so to speak. This process could be entirely automatic and covert, but it seems likely that there are considerable differences in how skilled different people are at employing it. These differences would be expected to correlate with differences in empathy, emotional awareness or their dysfunction (as seen in sociopathy and ALEXITHYMIA, for example). There are also some unanswered questions about the extent of simulation that is necessary to construct social knowledge. For example, does the recognition of emotions from facial expressions rely on the internal generation of a motor or somatosensory representation of the face alone? Or does it rely on the generation of a more comprehensive

ALEXITHYMIA
Cognitive disturbance that is characterized by the difficulty in describing one's own emotions.

WASON SELECTION TASK
The most popular experimental design for probing deductive reasoning. It consists of a conditional statement, the truth of which the subject must decide. Typically, conditionals about social rules, threats and promises all show a facilitation in the proportion of logically correct choices, and it has been argued that humans evolved a specialized skill to detect deception in the context of social contracts (for example, cheating).

SOMATIC MARKERS
Emotional states that are triggered during the consideration of potential future outcomes of choices.

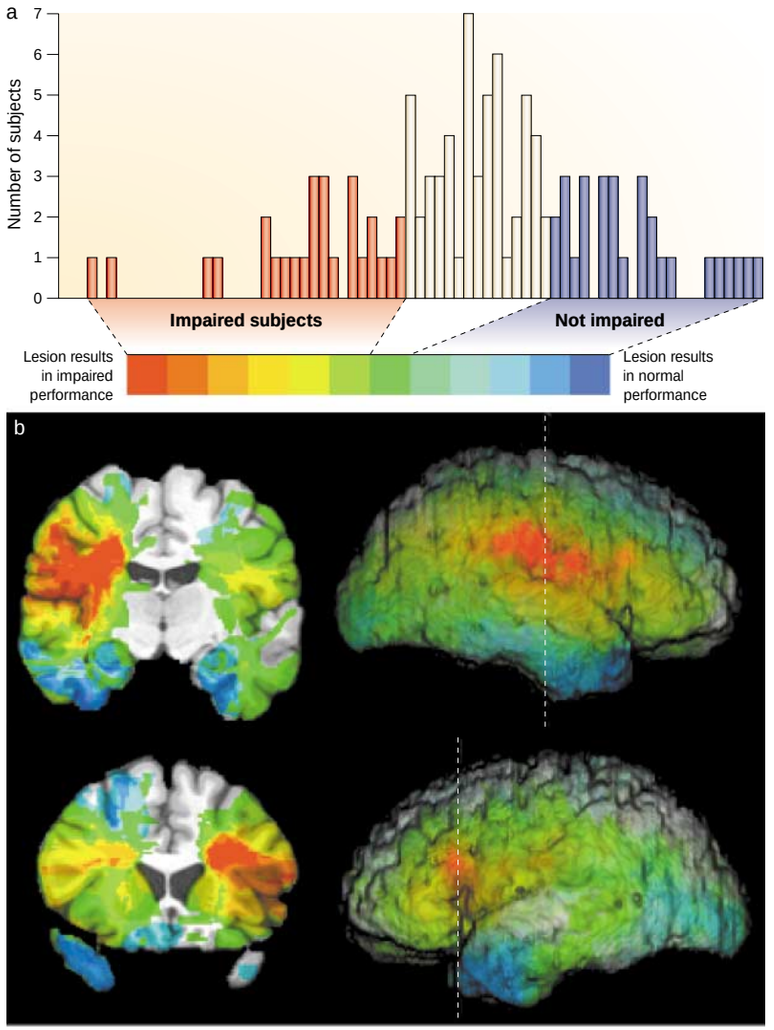


Figure 6 | **Brain regions that might support simulation.** This figure shows the association between lesions in particular regions and the impaired ability to judge other people's emotional state. **a** | Performance scores of 108 subjects with focal cerebral lesions. The subjects were divided into two groups according to their performance score: impaired and normal. **b** | To investigate the possibility that some lesions were systematically associated with either a high or a low performance, the overlaps of lesions from subjects within each of these two groups was computed. Different colours encode the difference in the density of lesions between subjects with the lowest and the highest scores. So, red regions correspond to locations at which lesions resulted in impairment more often than not, and blue regions correspond to locations at which lesions resulted in normal performance more often than not. Lesions in the right somatosensory cortex, the right insula, the anterior supramarginal gyrus and the left frontal operculum were systematically associated with impaired recognition of emotion from facial expressions. White dashed line indicates the plane of the section shown on the left. Modified, with permission from REF. 105 © (2000) Elsevier Science.

representation that simulates the entire state of the body that is associated with the emotion shown by the face? Perhaps the depth of detail to which the body state associated with an action, emotion or social process needs to be simulated depends on the demands of the task at hand, ranging from partial and coarse representations in association cortices to fine-grained topographic representations in the primary somatosensory cortex.

Social reasoning, decision making and dilemmas

The orbitofrontal cortex has also been implicated in social reasoning. Damage to this region impairs the ability to figure out that other people are being deceptive[80], and results in an impaired performance in reasoning about social exchange using the WASON SELECTION TASK[107,108]. These findings could reflect the previously mentioned role for the orbitofrontal cortex in guiding social cognition by the elicitation of emotional states that serve to bias cognition, a role that is further supported by investigations of decision making and moral reasoning.

It has long been known that damage to the ventromedial prefrontal cortices impairs the ability to decide advantageously in real life[109], an ability that relies on SOMATIC MARKERS[95]. Investigations using a gambling task have shown that somatic markers appear in anticipation of making a risky choice, even prior to its execution[110], and that they can appear in the absence of overt knowledge about the risks of the choice (or about the somatic marker, for that matter)[111]. There is further evidence that the reward and punishment that come into play during such gambling tasks activate the medial prefrontal cortex[112], and might engage distinct sectors of the orbitofrontal cortex[113]. The amygdala might also be activated during decision making, specifically when waiting for a potentially negative outcome after a risky decision has been made[114]. Whereas damage to the orbitofrontal cortex in adults impairs somatic markers for decision making, it spares abstract knowledge regarding decision making; such patients can usually describe what to do in an abstract choice, but become impaired when faced with actually having to choose themselves[115]. By contrast, damage to the orbitofrontal cortex incurred early in childhood impairs not only actual decision making, but also abstract knowledge regarding advantageous choices and specifically about right and wrong — that is, moral knowledge[116].

The role of specific brain structures in moral behaviour has been investigated using social and moral dilemmas (BOX 3) in which the choice options are structured so that they conflict[96]. Such conflict could arise from short-term versus long-term goals, or from goals that are advantageous to oneself versus those that are advantageous for others or for society as a whole. It is therefore closely related to altruistic behaviour, to social cooperativity and to the cognitive processes that guide behaviour in fields as diverse as politics and economics. A subset of moral dilemmas involve one's own agency and trigger strong emotions in their consideration; these have been found to engage structures that are involved in emotion processing, such as the superior temporal sulcus, and the cingulate and medial prefrontal cortices[117]. Social cooperation in the Prisoner's dilemma (BOX 3) engages a similar set of structures, including the orbitofrontal and anterior cingulate cortices and the ventral striatum[118]. All of the above data on the medial and orbital prefrontal cortex are consistent with a role for this region in guiding the strategic adoption of someone else's point of view — perhaps by triggering emotional states, by engaging simulation routines or by more cognitive strategies.

Box 3 | Moral dilemmas

Moral dilemmas are choices for which all outcomes are morally undesirable. In the 'trolley dilemma', for example, a trolley is heading down bifurcating tracks. One set of the tracks goes towards a group of people, the other towards a single person. The default path of the runaway trolley is towards the group of people. If you do nothing, they will all be crushed to death. But you have the option of switching the tracks so that the trolley instead kills only the single person. In a variant of this dilemma, there is only a single track leading to the group of people, and you have the option of pushing a single person in front of the trolley to stop it. The options are similar in the two versions, but most people choose the action leading to the single death only in the former one, a decision influenced by emotions and sense of responsibility[96].

In the 'prisoner's dilemma' — a formal two-person game that is used to investigate social cooperativity — players can choose to give or keep money, which determines how much they are paid in turn. If only you keep the money and the other player gives it away, you make the most money and the other player loses the most. If both of you give it away, you both make a moderate amount of money. If both of you keep money, you both lose a moderate amount of money. So, there is a conflict between the selfish strategy of keeping money (you could win a lot of money, or both of you could lose money) and the cooperative strategy of giving it away (you could lose a lot of money, or both of you could win money). If playing multiple rounds, various kinds of patterns in social behaviour can emerge, including reciprocity and mutual cooperation. Cooperation in a single-round game might depend on the prior evolution of social emotions[5].

How does the brain process social information?

The roughly serial processing architecture around which this review is organized (FIG. 1) belies the complexity of social information processing. This complexity arises in at least three ways. First, there are parallel processing routes. For instance, pathways involving the amygdala and subcortical structures can trigger rapid emotional responses, whereas slower emotional behaviour relies on prefrontal and parietal cortical processing that involves self-regulatory components. Second, there is extensive feedback between different processing levels, such that it becomes difficult to assign levels to any particular hierarchy. Third, stimuli are processed in the context of a background, baseline mode of brain operation that might already introduce substantial biases. For example, semantic judgements about people from words that describe them, compared to judgements about other objects, activates many of the regions mentioned earlier, such as the medial prefrontal, superior temporal and fusiform cortices[119]. However, these activations arise from a high baseline activation in those regions at rest, compared to their deactivation when processing non-people stimuli. This indicates that the brain's baseline activity might reflect a mode of operation that is already tuned to interpreting and categorizing the world as social[119].

Although the data that I have reviewed earlier in this article converge on several key brain structures that mediate social cognition, it cannot be overemphasized that the causal role of these structures remains unclear. The most commonly used technique — functional brain imaging — is statistically complicated and limited by degeneracy in the function of brain structures: a structure might be activated but not result in impairment when lesioned, perhaps reflecting redundancy within the systems in which it participates[120]. It is striking that patients with lesions in brain regions that are normally activated during certain processing often have impairments that are very subtle, and that only emerge under the constraints of a specific task. This probably reflects the considerable redundancy and plasticity of the brain. It also indicates that caution should be taken in attempts to predict people's behaviour from knowledge about their brains.

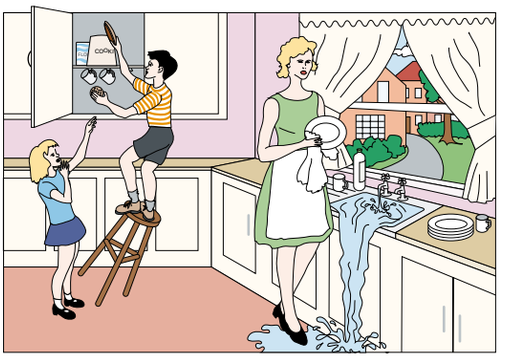
Neuropsychiatric aspects of social cognition

The marked differences in social cognitive abilities in the normal population are continuous with those seen in neuropsychiatric disorders. Many of these disorders are developmental in nature and emphasize the important role that social environment[121,122] and neural systems[123] have in early socioemotional development. The neural basis of abnormal social cognition has been investigated in disorders such as autism, WILLIAMS SYNDROME, psychopathy and social phobia.

Interest in the social cognitive abilities of subjects with autism was fuelled by the argument that autism features a disproportionate impairment in a specific aspect of social cognition — the ability to attribute mental states to others (that is, to have a theory of mind)[124–126]. This impairment might explain why some people with autism have difficulty in their social behaviour, even though they can function with high efficacy in other respects. Something like the inverse pattern of impairment is seen in Williams syndrome[127], a genetic disease that features hypersociability (FIG. 7). Our knowledge about the neural underpinnings of these disorders is limited, although the amygdala has been implicated in both of them. Given that autism and Williams syndrome are developmental disorders, it is intriguing to note that the amygdala and adjacent structures show an increase in volume that seems to persist into adulthood[128,129], although the functional significance of this observation is unknown.

Intense interest and debate has also focused on the cognitive neuroscience of human violence and aggression[130], the emergence of which depends on complex interactions between genetic predispositions and the environment[131]. The orbitofrontal cortex[116,132] and the amygdala[82] have been implicated in violent behaviour, especially if their activity is compromised early in life. For example, criminal psychopaths show structural abnormalities (reduced grey-to-white matter ratio) in the prefrontal cortex[132], and abnormal activation of the orbitofrontal cortex and the amygdala in functional imaging studies[133], together with reduced autonomic emotional responsivity. As we might predict from these data, psychopaths also show impaired performance on gambling tasks that have been used to assess the role of somatic markers in decision making[134]. The predominant interpretation of these findings has been that they reflect the broader role of these structures in emotional regulation, a role that translates into aspects of violent and aggressive behaviour when situated within a specific context[93].

WILLIAMS SYNDROME
A genetic condition caused by a deletion on chromosome 7 that is characterized by an unusually social personality, limited spatial skills and motor control, and mental retardation. Patients with the disease also have heart problems, hypercalcaemia, kidney abnormalities, sensitive hearing and musculoskeletal problems.



Examiner: I want you to look at the picture and tell me everything that is going on.

WMS age 10: (*Laughs*). Oh no. The mommy left the tap on (*pointing to the water*). And the boy is trying to get a cookie but the chair is tipping over. (*In a high voice, as if addressed to the mother*) Mom, won't you save the boy? (*Returning to normal tone*) Gosh. She better quickly save her boy. Her son and her daughter. Oh, there's going to be a flood on her floor. The boy's in the cookies. Maybe it's after supper. Maybe. Oh, the mommy is drying the towel. Poor boy, he could get hurt and break his arm. Poor boy, oh poor thing.

DNS age 10: Mom wash dishes. A bowl fell. Boy slips, boy pushed. Boy helps mom with dishes. Mom big mess in water. Pushing. (Examiner: Can you tell me anything else about the picture?) (*Shakes head*.)

Figure 7 | **Hypersocial function in subjects with Williams syndrome.** Williams syndrome results from the deletion of a small set of genes on one copy of chromosome 7. Although the patients are mentally retarded and severely impaired in visuospatial function, they show an exaggerated interest in other people and remarkable expressiveness and social communicative abilities. When shown a picture, such as the 'cookie theft' picture that is commonly used in neuropsychology, people with Williams syndrome tell stories (top) that are not only longer and more complex than those told by same-age subjects with Down syndrome (bottom), but their narratives are also infused with expressive details and social attention devices. DNS, Down syndome subject; WMS, Williams syndrome subject. Modified, with permission, from REF. 127 © (2000) MIT Press.

Social phobia is a debilitating fear of others, public places or public social interaction that might arise from a fear of losing a fundamental aspect of social behaviour — the need to belong to a social group[135]. Increased amygdala activation has been found in subjects with social phobia when viewing neutral[136] or angry [137] faces, or when preparing to give a public speech[138]. People with psychopathy or social phobia show inverse extremes of emotional responsiveness to social stimuli, and there is evidence that they feature, respectively, exaggerated and reduced activation in the amygdala and the orbitofrontal cortex[139].

The neuropsychiatric aspects of social cognition that I have discussed so far focus on disabilities. Much less work has been done on exceptional social abilities, although research into Williams syndrome[127] and TURNER SYNDROME[140] tackle an angle of this issue. But it would be interesting to study what happens in the brains of people who are especially kind, empathic, altruistic or socially skilled. There is a growing interest in enhancement of normal social cognitive skills through interventions ranging from educational strategies during development to pharmacological manipulation in adults, but the paucity of data makes their application to real life premature.

Conclusions

Progress in our understanding of the neural basis of social behaviour has been rapid. We have identified collections of processes and neural structures that participate in our perceptions and judgements of social stimuli, the ways in which we reason about and decide among them, and the ways in which individual and collective behaviour is guided. Further progress crucially depends on advances on two fronts: the development of methods and of theory.

In the near future, we will see more studies that use virtual-reality stimuli[141], and new techniques such as simultaneous scanning of multiple subjects engaged in mutual social interactions[142]. Both approaches will give us a better approximation to real life, an issue that is vital for investigating social behaviour. In addition to the examination of the genetic contributions to specific cognitive abilities (for example, in rare human diseases, transgenic mice or different dog breeds), there are also genetic techniques on the horizon that might offer new ways of reversibly lesioning specific brain structures[143].

Even once future technology shows us more clearly how neural events co-vary with stimuli and behaviour, how to interpret such data will remain a deep theoretical challenge[144]. One view is that there are specialized processes for social cognition; another is that social cognition arises out of more basic components that are not so specialized themselves (TABLE 1). This raises the question of whether social cognition is reducible to emotional or motivational processing. For example, when we find a face attractive or trustworthy, do we engage the same mechanisms as when our behaviour (and that of other animals) is reinforced by food? Or does the way in which social stimuli are processed differ fundamentally from reward and punishment for nonsocial stimuli? There is some indication that the orbitofrontal cortex might be more specialized for social and moral judgements, whereas the amygdala might subserve a broader role in emotional processing that includes basic emotions[145,146].

On the one hand, all behaviour can be dichotomized as some form of approach or withdrawal, and some theoretical schemes have attempted to map all emotions onto a two-dimensional space of reward and punishment[147]. On the other hand, all the different basic and social emotions, and all the different words that we have for describing patterns in social behaviour, differentiate such behaviour at a considerably finer grain. So the question is: what is the appropriate level of grain at which we should describe social behaviour and the central states that regulate it? Although it is implausible that we will find a distinct neural substrate for every different emotion and personality factor, it also seems that there must be systems more differentiated than just

TURNER SYNDROME
A genetic disease in which females carry only one healthy X chromosome. It is characterized by an inhibition of sexual development and is accompanied by infertility. There is some evidence from patients with Turner syndrome for the existence of an imprinted X-linked locus that affects social cognition.

Table 1 | Different ways of classifying behaviour

Category of behaviour	Example
Social disposition	Personality traits (extraversion, neuroticism)
Strategic	Deception, reconciliation
Ecological	Attachment, aggression
Moral	Social emotions (guilt, embarrassment, pride, jealousy)
Emotional response	Basic emotions (happiness, fear, anger, disgust, sadness)
Reinforcement	Motivational state (reward, punishment)

reward and punishment[148]. We might need to invent a new set of terms that can translate between the different ways of describing social behaviour, and that correspond more closely to the neural processes that underlie them.

It might be that certain social cognitive skills — notably the ability to represent other people's minds — distinguish humans and perhaps apes from all other animals. If we understand other people in part by simulating processes within ourselves, the converse is also true: we understand ourselves in part by observing other people and their reactions to us. Our ability to think about other people might be an aspect of our ability to re-describe events from several points of view — an ability that might fuel the emergence of science, art and culture in general. Just as we can think about other people, we can step outside ourselves and think about ourselves, have conversations with ourselves, and imagine things happening to ourselves in the future.

The data that are available at present raise as many questions as they provide answers. How can the diverse findings that we accumulate be situated under a single functional framework? Specifically, how can causal networks explain the many correlations between brain and behaviour that we are discovering? What are the relative contributions of innate and acquired factors, culture and individual differences to social cognition? To what extent do these factors contribute to psychopathology? Can large-scale social behaviour, as studied by political science and economics, be understood by studying social cognition in individual subjects? Finally, what power will insights from cognitive neuroscience give us to influence our social behaviour, and hence society? And to what extent would such pursuit be morally defensible? How we approach these questions will largely shape social brain science in the coming decades.

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