Review

Social cognition and the human brain

Ralph Adolphs

Humans are exceedingly social animals, but the neural underpinnings of social cognition and behavior are not well understood. Studies in humans and other primates have pointed to several structures that play a key role in guiding social behaviors: the amygdala, ventromedial frontal cortices, and right somatosensory-related cortex, among others. These structures appear to mediate between perceptual representations of socially relevant stimuli, such as the sight of conspecifics, and retrieval of knowledge (or elicitation of behaviors) that such stimuli can trigger. Current debates concern the extent to which social cognition draws upon processing specialized for social information, and the relative contributions made to social cognition by innate and acquired knowledge.

Social cognition refers to the processes that subserve behavior in response to conspecifics (other individuals of the same species), and, in particular, to those higher cognitive processes subserving the extremely diverse and flexible social behaviors that are seen in primates. Its evolution arose out of a complex and dynamic interplay between two opposing factors: on the one hand, groups can provide better security from predators, better mate choice, and more reliable food; on the other hand, mates and food are available also to competitors from within the group. An evolutionary approach to social cognition therefore predicts mechanisms for cooperativity, altruism, and other aspects of prosocial behavior, as well as mechanisms for coercion, deception and manipulation of conspecifics. The former are exemplified in the smallest groups, in the bond between mother and infant; the latter in the largest groups by the creation of complex dominance hierarchies.

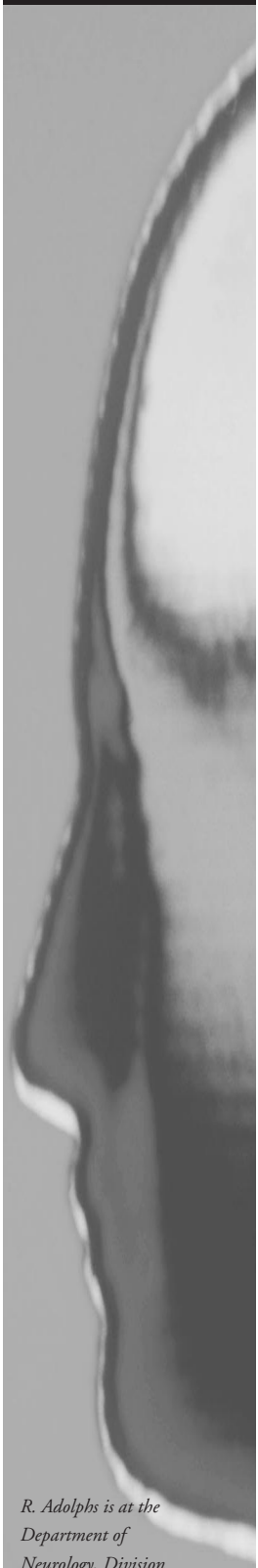
It is clear that primates are exceedingly adept at negotiating the social environment. This ability is most striking in the most social primate, *Homo sapiens*, suggesting the hypothesis that our exceptional cognitive skills may be traced back to evolution in an environment in which there was a premium on social skills. In support of this idea, there is a correlation between mean group size among various primate species and their neocortex volume (specifically, the ratio of neocortex volume to the rest of the brain[1]). Such a correlation has been found also for several other mammals that all feature a complex social structure (e.g. bats, carnivores and toothed whales) – the larger the social groups, the larger the brains (relative to body size). Although it has been proposed that brain size correlates with a number of other factors, including dietary foraging strategy, tool use and longevity[2,3], it might be that large brain size is at least a partial consequence of the fact that primates have a complex ecological niche with respect to social structure (including its effect on food and mate availability). This hypothesis, variously dubbed the 'Machiavellian Intelligence Hypothesis'[4] or the 'Social Brain Hypothesis'[1], depending on what theorists take to be its most salient features, suggests that the complexity of primate social structure, together with certain of its unique features, such as cooperativity and deception, led to an advantage for larger brains.

Aside from sheer brain volume, one would of course like to know more about the specific neural systems that subserve various aspects of social cognition. A seminal review[5] argued for the importance of the following set of structures: amygdala, temporal cortex, anterior cingulate cortex, and orbitofrontal cortex[6]. The neurobiological underpinnings of social cognition in humans, the topic of this review, are being investigated using various methods, including lesion studies and functional imaging, and can be situated in the context of what we know about social cognition from anthropological, comparative and developmental studies.

An overview of the neurobiology of social cognition in primates

Non-human primates

Two sets of findings, one at a macroscopic level, the other at a microscopic level, first suggested that the primate brain might contain neural systems specialized for processing socially relevant information. In the 1930s, Kluver and Bucy made large bilateral lesions in monkey brains, encompassing amygdala, temporal neocortex, and surrounding structures[7]. The animals subsequently appeared able to perceive and respond to objects in their environment, but they behaved inappropriately with respect to the emotional significance that objects would normally signal. This included a compulsive examination of objects, especially with the mouth, hypersexual behavior, unusual tameness, and a complete lack of awareness of the emotional significance of stimuli ('psychic blindness'; e.g. handling of snakes). Selective neurotoxic lesions of the monkey amygdala result in more subtle impairments; however they do still appear to impair disproportionately



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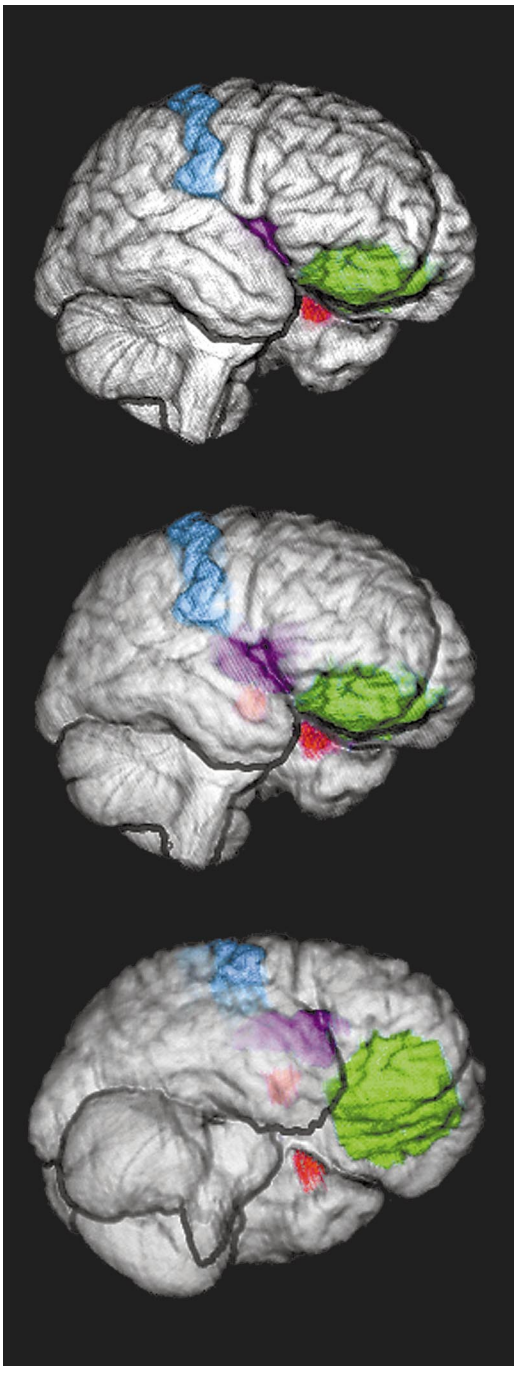

Fig. 1. Summary of neuroanatomical structures involved in social cognition. These renderings of a normal human brain, reconstructed from serial magnetic resonance (MR) images, show the neuroanatomical structures highlighted in this review. The same brain is shown in different views and with differing amounts of transparency, to permit visualization of interior structures. The images were generated by first tracing the structures on 2-D MR images, and then co-rendering these regions of interest in various colors together with the rest of the brain. Highlighted are ventromedial prefrontal cortex (green), amygdala (red), right somatosensory cortex (blue) and insula (purple), all of which play key roles in various aspects of social cognition discussed here. Additionally, other structures (not colored), such as the cingulate cortices, visual association cortices in temporal lobe, and structures in hypothalamus, thalamus, and brainstem contribute to social function. All of these structures also play varied roles in regulating emotion. (Figure kindly provided by Deema Fattal and Hanna Damasio, Human Neuroanatomy and Neuroimaging Laboratory, Department of Neurology, University of Iowa.)

those behaviors normally elicited by social cues[8–10]. Although the amygdala is a heterogeneous collection of nuclei that participate in several different functional systems[11], at least some of its components thus appear to contribute disproportionately to social behavior.

The other set of findings that first sparked interest in the neural basis of social cognition pertains to the level of single neurons. Neurophysiological studies in non-human primates have shown that single neurons in the monkey inferotemporal cortex respond relatively selectively to the sight of faces[12]. Moreover, specific neurons modulate their response preferentially with specific information about faces, such as their identity, social status or emotional expression[13–15]. There are also neurons whose responses are modulated by viewing complex scenes of social interaction[16,17], as well as by specific features of faces that can signal social information, such as gaze direction[18]. A neural code in which the responses of individual neurons are tuned relatively selectively to highly specific feature conjunctions may permit a neuronal ensemble to distinguish among complex, similar members of a large class of stimuli, such as the faces of conspecifics. Current information-theoretical approaches are providing more detail on how such socially relevant information might be encoded in a neuronal population[19].

Humans

Human social cognition has received extensive attention from cognitive, developmental and social psychologists. Some important current issues that might be informed by findings from cognitive neuroscience concern how social cognitive abilities develop in infants, and to what extent genetic factors might influence such abilities. Clearly, the emotional and social development of humans is extraordinarily complex, involving a multi-factorial interplay between genes, parental behavior, and the influence of culture.

There have been two major sets of studies that first argued for neural systems critical to social cognition in humans: social impairments following damage to the frontal lobe, and, more recently, social impairments in subjects with autism. The observation that the frontal lobes can contribute relatively specifically to behavior in the social domain was first made on the basis of a rather horrible accident: the injury of the railroad worker Phineas Gage[20]. Gage received a large bilateral lesion of his frontal lobe, including the ventromedial prefrontal cortex, from an accidental explosion that shot a metal rod through his head (see Fig. 1 for neuroanatomical structures highlighted in this review). Whereas Gage had been a diligent, reliable, polite and socially adept person before his accident, he subsequently became uncaring, profane, and socially inappropriate in his conduct. This change in his personality remained a mystery until it could be interpreted in the light of similar patients in modern times: like Gage, other subjects with bilateral damage to the ventromedial frontal lobes show a severely impaired ability to function in society, despite an entirely normal profile on standard neuropsychological measures, such as IQ, language, perception and memory. Recent theoretical explanations propose that the ventromedial frontal cortices play an important role in associating emotional experience with decision making in complex situations, especially perhaps situations in the social domain (see below).

Box 1. Social cognition, modularity and innateness

Focal brain damage can result in impaired processing that is limited to highly specific categories. For instance, patients have been reported who are specifically unable to recognize, or to name, tools, animals, people, or a variety of other selective categories. There is thus very strong evidence that categories are, in some sense, mapped in the brain (but in a way that differs from aspects of objects that are a direct consequence of topography at the sensory epithelium). While initially surprising, the finding is in fact predicted from the assumption of a few, very simple, local rules that specify how brains represent stimuli (Ref. a). In essence, local rules for organizing neural tissue as a function of activity suffice to generate topographic representations of abstract stimulus categories. The categories that are abstracted emerge naturally out of the covariances of our interactions with certain classes of stimuli in the environment. Thus, we typically interact with members of the class of animals in a similar way; that is, the similarity is greater among animals than it is to how we typically interact with members of the class of tools, or members of the class of people. Similarity in sensorimotor interaction can thus translate into functional and anatomical similarity in the brain (Refs b,c).

The above view suggests a strong component of experience and learning in such self-organized topographic maps. A different explanation comes from the view that there are innately specified modules in the brain for processing specific categories of knowledge. The evidence for this latter view is strongest from domains such as language, and it is the view that has historically been associated with the notion of 'modularity' (Ref. d).

As with many dichotomies, it is likely that both the above views are right, in the proper context, and recent interpretations suggest a softer version of 'modularity' that does not require a rigid set of criteria (Ref. e). It may well be that there are domain-specific modules for processing certain kinds of information that are ecologically highly relevant and that would benefit from a particular, idiosyncratic processing strategy that does not apply to other kinds of information. That is, one would expect the brain to provide problem-specific structures for processing information from those domains in which there is a premium on speed and survival. Within, and beyond, such a module there might also be topographic mapping of the same domain. It is likely that domain-specific processing draws upon innately specified modules, as well as upon self-organized maps that emerged as a consequence of experience with the world.

Is social cognition modular? And if so, is it innate, or is it a consequence of learning? It is likely that both are true, and that whether or not social cognition is modular will depend both on one's notion of modularity and on the aspects of social cognition under consideration. Some rather basic attributes of stimuli, such as self-directed motion, bilateral symmetry, presence of eyes, and so forth, might be processed similarly by different primate species, by mechanisms that are largely innately specified. But there also seems little doubt that the class of social stimuli needs to be explored during development in order to be able to make more fine-grained distinctions – a developmental process that is likely to include parental behavior and pretend play as critical aspects. The most plausible scenario, then, would view social cognition as relying on a neural architecture in which there is interaction between components that are innately specified and others whose operation emerges through experience in the context of a specific culture.

A similar answer would presumably obtain in regard to the broader question of cognition, not only with respect to the social world, but the animate world in general (see Ref. f for more extensive discussions of this topic). Future goals will be to provide a more detailed account of the relative contributions that innate and culturally acquired components make to social cognition, and to explore how such functional components might be subserved by specific neuroanatomical structures.

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A second line of evidence that has been used to argue for the functional modularity of social cognition (see Box 1) comes from a developmental disorder, childhood autism. Interest in the social cognitive abilities of subjects with autism was fueled by the argument that autism features a disproportionate impairment in one specific aspect of social cognition: the ability to attribute mental states, such as beliefs, to others[21,22]. While there is debate on the basic hypothesis, and while the link between autism and brain systems is also not well understood, the data point towards neural components that appear to have a high degree of domain-specific function. This idea is strengthened by comparison with another psychiatric disorder, entirely genetic in etiology: Williams syndrome. Subjects with Williams syndrome exhibit social behavior that comes close to being the opposite of that seen in autism – they are hypersocial, and their unusual social skills in the face of impairments in non-social domains have been taken as further evidence for the modularity of social cognition[23]. Of particular interest will be comparisons among subjects with Williams syndrome, autism, and focal brain lesions, some of which are now underway. For example, a recent study found that subjects with Williams syndrome showed selective sparing in their ability to recognize other people's mental states from photographs of their eyes[24], a task that high-functioning subjects with autism fail[25], and which in normal individuals, but not in subjects with autism, has been shown to activate the amygdala in functional imaging studies[26]. Subjects with Williams syndrome also show abnormally positive judgments of approachability when shown unfamiliar people's faces, one component of impaired social judgment that might share commonalities with impaired social judgment seen in patients with bilateral amygdala damage[27].

The amygdala: social judgment of faces

We glean considerable social information from faces, and there is evidence to suggest that faces are processed in a relatively domain-specific fashion by neocortical sectors of the temporal lobe. For instance, visual processing in regions of

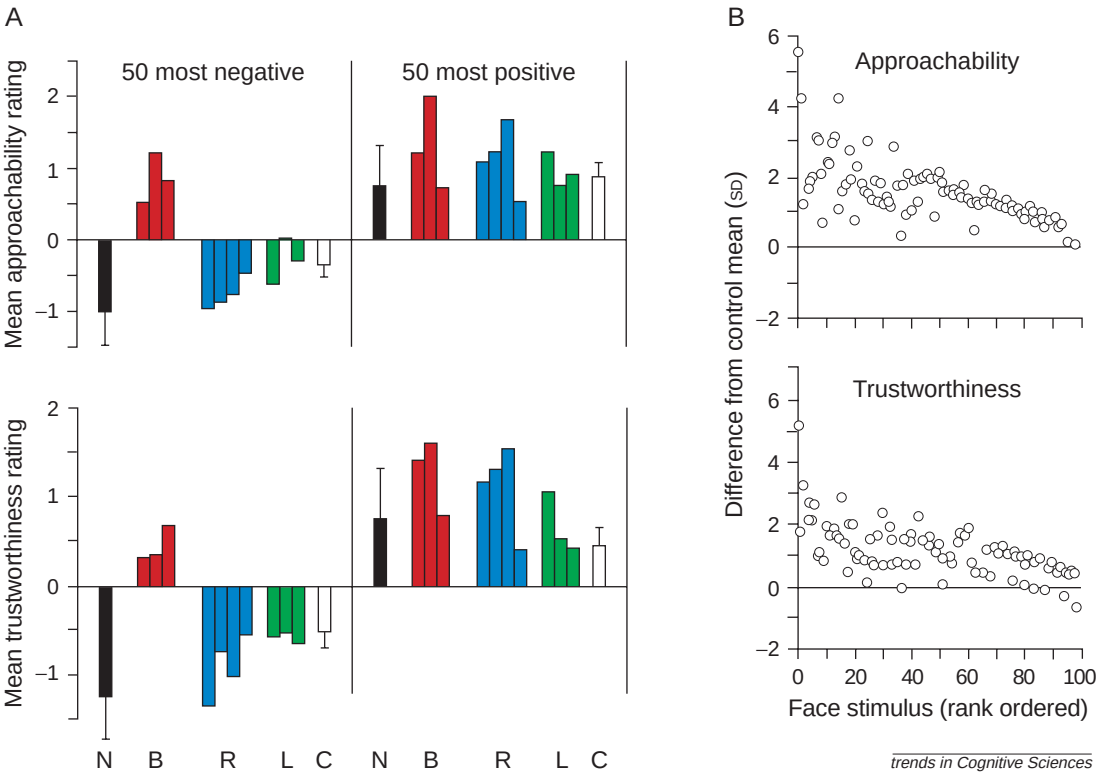

Fig. 2. Mean judgments of approachability and trustworthiness of 100 unfamiliar faces. (A) Means and SD for data from 46 normal controls are given at the left of each panel ('N'). Individual means are shown for each of three subjects with bilateral amygdala damage ('B'), four subjects with unilateral right ('R') and three with unilateral left ('L') amygdala damage. Group means and SEM are shown for seven brain-damaged controls with no damage to amygdala ('C'). The data, broken down into those obtained from the 50 faces that received the most negative mean ratings from normal controls (left) and the most positive (right) on each attribute, show that subjects with bilateral amygdala damage gave abnormally positive ratings to that half of the stimuli that normally receive the most negative ratings. **(B)** Standard deviations from the control mean are shown for one of the three subjects with bilateral amygdala damage (subject SM). Each face was judged on a scale of −3 (very unapproachable or untrustworthy) to +3 (very approachable or trustworthy). The data show that SM rated nearly all faces as positive. (Data redrawn from Ref. 43.)

the human fusiform gyrus appears to contribute disproportionately to the perception of faces[28], and viewing dynamic information from faces that convey socially relevant information (such as eye or mouth movements) activates regions in the superior temporal sulcus[29]. Recent data suggest that it is a particular property of how we interact with faces that leads to the specific neuroanatomical processing seen, namely, that we need to become expert at distinguishing many exemplars that are visually extremely similar and yet socially highly distinctive[30].

High-level visual cortices in the temporal lobe project to the amygdala[31], which has also received historical and recent interest in regard to its role in processing emotionally and socially salient information from faces. A small proportion of neurons within the amygdala show responses that are relatively selectively modulated by the sight of faces, compared with other visual stimuli[32,33]. Studies that have used functional neuroimaging in normal subjects[34,35], and studies that have examined patients with damage to the amygdala[36–38], have provided evidence that the amygdala is critical to recognize emotions from facial expressions, specifically certain negative emotions, such as fear. The findings have been broadly consonant with the amygdala's contribution to social behavior that was suggested by earlier lesion studies in animals, as well as with the large number of animal studies that have investigated the amygdala's role in fear conditioning[39,40]. While these threads of research have pointed to a disproportionately important role for the amygdala in processing stimuli related to danger and threat, there are also findings, primarily from studies in animals, that suggest a more general role for the amygdala in processing emotionally arousing stimuli that are either pleasant or aversive. One recent theoretical view suggests that the amygdala, in both humans and animals, might subserve a more general role in allocating processing resources to biologically salient stimuli that are ambiguous, and about which additional information needs to be acquired, regardless of the valence of those stimuli[41].

Given the above findings, one might expect that the amygdala would make important contributions also to higher-level social cognition, especially to those aspects of it that rely on recognizing social information from faces. This prediction is indeed borne out by recent studies. One important cue, direction of eye gaze in a face, has been shown to be processed by the amygdala in both some lesion[36] and functional imaging studies[42]. Other studies have examined the amygdala's role in more global social judgments. We investigated subjects' ability to judge how trustworthy or how approachable other people looked, from perceiving their faces. Such an ability would be expected to draw on aspects of social recognition, as well as on social decision making. In our study, we found that three subjects who had bilateral amygdala damage all shared the same pattern of impairment: they judged to be

Box 2. Attribution of social meaning from visual motion

Subjects were shown a short movie of simple geometric shapes in motion (a still from the movie is shown in Fig. I). After seeing the movie, they were asked to describe what they saw. While normal subjects immediately ascribe social meaning to what they see, a subject with developmental amygdala damage (subject SM) failed to do so, interpreting the stimulus in purely geometric terms. The findings suggest that the amygdala may be critical in order to acquire the social knowledge by which normal individuals automatically assign social meaning to stimuli.

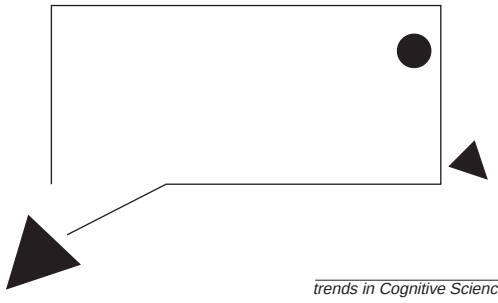

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Fig. I. A still from the movie of simple geometric shapes in motion that was shown to the subjects (see text for description). (Redrawn from Ref. a.)

Normal control subject

'I saw a box, like a room, that had an opening to it. There was a large triangle chasing around a smaller triangle, and a circle…got into the box, or the room, and hid. And then the big triangle chased the little triangle around. Finally he went in, got inside the box to go after the circle, and the circle was scared of him…but manoeuvred its way around and was able to get out the opening, and they shut it on him. And the little circle and the little triangle were happy that they got that, the big one, caught. And they went off on their way, and the big triangle got upset and started breaking the box open.'

Subject SM

'OK, so, a rectangle, two triangles, and a small circle. Let's see, the triangle and the circle went inside the rectangle, and then the other triangle went in, and then the triangle and the circle went out and took off, left one triangle there. And then the two parts of the rectangle made like an upside-down V, and that was it.'

Reference

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abnormally trustworthy and approachable the faces of those people who are normally judged to look the most untrustworthy or most unapproachable[43] (Fig. 2A). While the subjects with amygdala damage showed a general positive bias in judging all faces, they showed a disproportionate impairment when judging those faces normally given the most negative ratings (Fig. 2B). The amygdala's role in processing stimuli related to potential threat or danger thus appears to extend to the complex judgments on the basis of which we regulate our social behavior. Clearly, the cues that we normally use to make such judgments will be complex, and there will be multiple strategies available to utilize them, a topic deserving further study.

There are two issues of additional interest: the specificity of the above impairment to faces, and its consequences for social behavior in the real world. In regard to specificity, follow-up studies revealed that bilateral amygdala damage also impaired judgments for the preferences of non-social visual stimuli, such as color patterns or landscapes, although the effect was not as large. In one such study, subjects with amygdala damage liked pictures of non-social stimuli more than did controls[44]. Thus, the amygdala's contribution does not appear to be entirely restricted to processing stimuli in the social domain, but may encompass a more general function which is of disproportionate importance to social cognition. A further experiment assessed social judgments that were made about other people on the basis of written descriptions of them. Judgments about people from such lexical stimuli were not impaired by amygdala damage[43], perhaps because the stimuli provided sufficient explicit information such that normal task performance could result from reasoning strategies that did not necessarily require the amygdala. However, two other recent studies have suggested that the amygdala is involved in processing lexical stimuli, when such stimuli signal potential threat or danger[45,46].

The second question, concerning the social impairments following amygdala damage in real life, is more difficult to investigate. However, observation of patients with complete bilateral amygdala damage suggests a common aspect to their social behavior: they tend to be unusually friendly towards others, consistent with the idea that they lack the normal mechanisms for detecting individuals that should be avoided. Similar changes in behavior are seen in non-human primates with selective bilateral amygdala damage[8,9]. On the other hand, the human patients do not appear to be as severely impaired in their social behavior as do monkeys with similar brain damage. It may be that humans with amygdala damage, unlike other animals, possess additional mechanisms for social reasoning and decision making, and are able also to draw substantially on declarative knowledge encoded in language, resulting in partial compensation for their impairment[47].

One would also like to extend the above line of investigations to additional types of stimuli, and to additional types of social information that can be gleaned from such stimuli. We have begun such an investigation, using visual motion cues to provide information about biological and psychological categories. In one experiment, subjects were shown a short video that depicts three geometric shapes moving on a plain, white background[48]. Although visual motion is the only available cue in this experiment, normal subjects have no difficulty interpreting the motion of the shapes in terms of social categories: the shapes are attributed psychological states, such as goals, beliefs, desires and emotions, on the basis of their relative motion. By contrast, a subject with selective bilateral amygdala damage did not spontaneously make such attributions[49]. When shown the same stimulus, she described it in purely geometric terms, lacking the normal, automatic social interpretation (Box 2).

A final important consideration concerns the amygdala's role beyond recognition and judgment, to encompass such

processes as attention and memory. It is clear from studies in animals that the amygdala contributes importantly to these processes[50], and that its role extends well beyond a function restricted to recognizing potential threat or danger; but such a possible role in humans is just beginning to be explored. For instance, emotionally[51] or socially[52] salient stimuli are remembered better by normal individuals, an effect that correlates with activation of the amygdala in functional imaging studies[53,54] and one whose function is impaired in patients with amygdala lesions[55].

Taken together, all the above findings argue that the amygdala is one component of the neural systems by which stimuli trigger emotional reactions, broadly construed. Such emotional reactions would include autonomic, endocrine and somatomotor changes in the body, as well as neurophysiological and neuromodulatory changes in brain function. Such multi-dimensional emotional responses would serve to modulate and to bias cognition and behavior in important ways, as a function of the emotional and social significance of the stimulus that is perceived. This role for the amygdala may be of special importance for relatively fast, automatic evaluation of biologically important stimuli, and will no doubt function in parallel with other systems.

An active program of research has explored why it might be adaptive to make certain social judgments about faces with certain properties. For instance, average faces are perceived to be highly attractive[56], but very slight deviations from the average may be considered even more attractive[57]. A possible evolutionary explanation of this effect proposes that averageness, symmetry, or slight deviations from it, are correlated with fitness; consequently, one would predict that such features could have signal value, and one would predict the evolution of perceptual mechanisms for their detection. However, these interpretations are very contentious (see Ref. 58 for a review). Contrary to prediction, some recent data suggest that people with attractive faces are not more healthy[59], and that at least some aspects of attractiveness also do not correlate with social dominance[60], but may be a more complex function of weighing multiple short-term versus long-term benefits[61]. Notwithstanding the current debates, it will be essential in future studies to attempt to link specific physical features that plausibly index fitness, with specific neurobiological adaptations for their detection, hopefully subserved by neuroanatomically and neurofunctionally identifiable structures.

While the studies reviewed above strongly implicate the amygdala in several of the processes that are important for normal social cognition, they are problematic from an anatomical point of view: they are both too macroscopic and too microscopic. They are too macroscopic because it is clear that different nuclei within the amygdala subserve different functions[11], an issue that is addressed in animal studies by lesioning specific nuclei rather than the entire amygdala. Functional imaging studies using fMRI with high field strengths, as well as rare studies of human patients with chronically implanted depth electrodes for monitoring seizures[62,63], will provide some further neuroanatomical resolution in this regard.

Of equal importance, lesion studies of the amygdala are too microscopic in that it is important to consider the amygdala as one component of a distributed neural system for social cognition. In particular, amygdala and prefrontal cortex appear to function together in processing the rewarding contingencies of emotionally salient stimuli[64,65], and it is likely that they function as two components of a system also in social cognition, a topic I address next.

The Ventromedial (VM) prefrontal cortex: social reasoning and decision making

Decision making: the somatic marker hypothesis

The frontal lobes have a long history in social behavior, going back to the story of Phineas Gage discussed above. More recently, it has become clear that the frontal lobes, specifically their ventromedial sectors, are critical in linking perceptual representations of stimuli with representations of their emotional and social significance[66]. This function bears some resemblance to that of the amygdala outlined above, but with two important differences. First, it is clear that the ventromedial frontal cortices play an equally important role in processing stimuli with either rewarding or aversive contingencies; whereas the amygdala's role, at least in humans, is clearest for aversive contingencies. Second, reward-related representations in the ventromedial frontal cortex are less stimulus-driven than in the amygdala, and can be the substrate of more flexible computations, playing a general monitoring role in regard to both punishing and rewarding contingencies[67].

The impaired social behavior in humans with ventromedial frontal lobe injury is notable for an inability to organize and plan future activity, a diminished capacity to respond to punishment, stereotyped and sometimes inappropriate social manners, and an apparent lack of concern for other individuals, all in the face of otherwise normal intellectual functioning[66,68,69]. Particularly striking are the patients' often gross lack of concern for the wellbeing of others and remarkable lack of empathy. While the details of impaired emotional and social function following damage to the ventromedial frontal lobes can be complex, and can vary from case to case, the impairments share a core dysfunctional mechanism that no longer permits cognitive processes to incorporate certain types of emotional knowledge.

The role of the human ventromedial prefrontal cortex in decision making has been explored in a series of studies that used a task in which subjects had to gamble in order to win money. As with gambling in real life, the task involved probabilistic contingencies that required subjects to make choices based on incomplete information. Normal subjects learn to maximize their profits on the task by building a representation of the statistical contingencies gleaned from prior experiences: certain choices tend to pay off better than others, in the long run. The key ingredient that distinguishes this task from other tasks of probabilistic reasoning is that subjects discriminate choices by feeling; they develop hunches that certain choices are better than others, and these hunches can be measured both by asking subjects verbally, and by measuring autonomic correlates of emotional arousal, such as skin conductance response. Subjects with damage to the ventromedial frontal cortex fail this task[70], and they fail it precisely because they are unable to represent choice bias in the form of an emotional hunch[71]. Not only do subjects with VM

frontal damage make poor choices on the task, they also acquire neither any subjective feeling regarding their choices[71], nor any anticipatory autonomic changes[72].

These findings are consonant with prior reports that subjects with VM frontal lobe damage do not trigger a normal emotional response to stimuli, including socially relevant stimuli[73], and support a specific hypothesis that has been put forth to explain the data: the somatic marker hypothesis[66,74]. According to this hypothesis, the VM frontal cortex participates in implementing a particular mechanism by which we acquire, represent, and retrieve the values of our actions. This mechanism relies on generating somatic states, or representations of somatic states, that correspond to the anticipated future outcome of decisions. The function of these somatic states is to steer the decision making process toward those outcomes that are advantageous for the individual, based on the individual's past experience with similar situations. Such a mechanism may be of special importance in the social domain, where the enormous complexity of the decision space precludes an exhaustive analysis.

Reasoning: the Wason Selection Task

The ventromedial frontal cortex appears to play a key role in a second domain of high relevance to social cognition: social reasoning. Human reasoning strategies have been intensively investigated using the Wason selection task, the most popular experimental design for probing deductive reasoning[75]. The Wason selection task consists of a conditional statement ('if P then Q'), often presented in some context (e.g. 'If you are drinking beer, then you must be over the age of 18'), and subjects must use deductive reasoning in order to decide its truth. Typically, the proportion of logically correct choices made by normal subjects on this task is facilitated by conditionals about social rules, threats, and promises (see Ref. 76 for a review). Cosmides and her colleagues have argued that these data provide evidence for evolved mechanisms for reasoning about social exchange. Specifically, the findings from the Wason selection task support the hypothesis of an evolved skill to detect deception in the context of social contracts (cheating), because an ability to rapidly and reliably detect such deception would have been adaptive[77] (although there is considerable debate regarding the interpretation of the data, and alternative models have been proposed).

We investigated the role of the VM frontal cortex in such deductive reasoning, using three groups of subjects: patients with damage centered on the VM frontal cortex, patients with damage centered on the dorsolateral frontal cortex (specifically excluding the VM frontal cortex), and patients with damage outside the frontal cortex. Subjects with bilateral damage to the VM frontal cortex were disproportionately impaired in normal reasoning about social and familiar scenarios, whereas they showed no abnormality when reasoning about more abstract material[78] (Fig. 3). These findings are consonant with those presented above, and support a role for the VM frontal cortex in guiding reasoning and decision making by the elicitation of emotional states that serve to bias cognition. While the ventromedial frontal cortices, together with the amygdala, would participate in a more general function of linking stimuli to emotionally valued responses, they may be notably indispensable when reasoning and making decisions

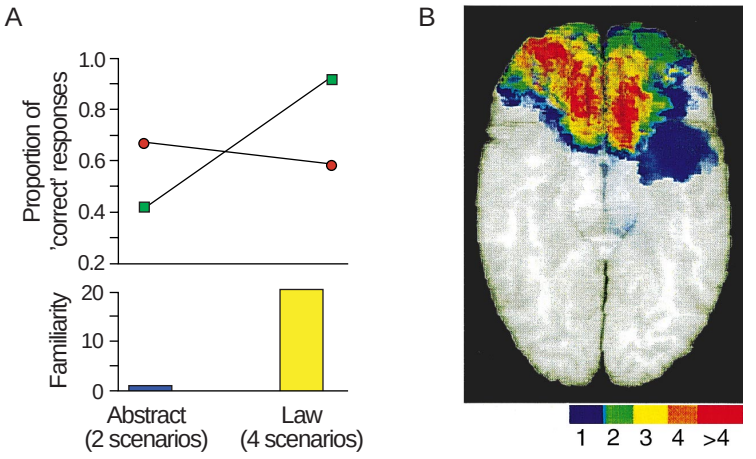

Fig. 3. Reasoning on the Wason Selection Task. (A) Subjects with ventromedial (VM) frontal lesions (circles) gave the logically correct answer more often than did control subjects (squares) when reasoning about scenarios where the subject matter was logical and abstract (e.g. 'If a student got an 'A' grade, then his card must be marked with the numeral '3'); however, they gave the logically correct answer less often than controls when the subject matter concerned familiar social situations, specifically social laws (e.g. 'If you are drinking beer, then you must over the age of 18') (result significant at $p=0.001$ level). These findings support the idea that, in normal subjects, the VM frontal cortices may be part of a system that facilitates correct reasoning about social matters. The results were especially striking, as the scenarios on which subjects with VM frontal damage failed are in fact the ones that are normally the easiest, and the most familiar, to reason about. When subjects were asked to indicate how familiar they found each of the scenarios, both VM frontal and normal control subjects judged the social laws to be much more familiar than the abstract scenarios (bottom, grouped data). **(B)** Volumetric overlap image of the lesions of all subjects with VM frontal lobe damage. Color encodes the number of subjects (indicated below) who have a lesion at a given anatomical location, rendered onto a ventral view of the human brain. The anatomical sites shared in common by all subjects in the group were in the ventromedial frontal cortex, bilaterally. (Data redrawn from Refs 78,92.)

about social matters. Additional future studies that attempt to dissect the broad collection of processes that comprise social cognition will help to shed light on the question of the specificity and modularity of ventromedial frontal cortex function. The evolutionary implications that can be drawn from such a disproportionate importance to social cognition remain a difficult and open question (Box 1).

The above findings from humans can be related to a large number of studies from non-human primates[79–81], which have shown abnormal social behavior, especially social isolation and avoidance[82] following damage to the orbital frontal cortices. The role of the orbitofrontal cortex in social affiliative behaviors is also of interest from a pharmacological point of view: the density of certain subtypes of serotonin receptors in the orbitofrontal cortex of monkeys correlates with the animal's social status. Pharmacological manipulation of serotonergic neurotransmission targeted at these receptors influences social affiliative behavior, and results in changes in social status[83]. These findings from monkeys may offer some explanation of the changes in social behaviors that can also be observed in humans following serotonergic manipulation [e.g. with drugs such as Prozac (fluoxetine) or ecstasy (MDMA)]. A further specification of neurotransmitter and neuromodulator systems will clearly be important in our understanding of the neural basis of social cognition; a more detailed discussion of this topic falls outside the scope of this review. Of special interest is the neurotransmitter serotonin (acting on specific subtypes of serotonin receptor), and the neuropeptide oxytocin, both of which appear to play a role

Box 3. How do we represent the minds of others?

Primates appear to be highly skilled at predicting other individuals' behavior, but there is vigorous debate about how to interpret such an ability. The mechanisms by which we represent and predict other people's behavior have been viewed from two different theoretical perspectives. The two main camps argue either for a 'theory of mind', or for a set of processes that permits 'simulation' of other minds. The 'theory'-theory has been floated for some time in philosophy of mind as a possible explanation of what is commonly called 'folk psychology': our commonsense understanding of other people's behavior in terms of intervening mental states, such as beliefs, desires and intentions, on the basis of which people act. The other camp, however, views our ability to recognize and reason about other people's states of mind as an example of experience projection; in essence, we know other minds by empathy, or by simulation. It is likely that both these views have some truth to them, depending on the circumstances (see Ref. a for examples of both sides of the debate). The theory-view might afford greater economy and generalizability of prediction, or might be particularly suited to information that can be lexically encoded; but simulation may be the only option in cases that are sufficiently idiosyncratic, or in cases where the information is not easily encoded into language. In the latter situation, it could be that the only way to predict what another person will do is to run in one's own brain the processes that the other person is running in theirs. If this possibility is taken seriously, it suggests a role for conscious experience in social cognition: to obtain information about another person's internal mental state, it may be necessary to imagine what it would be like to be the other person via direct simulation. Simulation might find its developmental origins in infants' ability to mimic facial expressions spontaneously (Ref. b), and it has found some recent neurophysiological support from the finding of so-called 'mirror neurons', which appear to participate in simulating the actions of other individuals (Ref. c).

Research into how we represent other minds began with a question about whether or not chimpanzees might possess a theory of mind (Ref. d), a question that is still unanswered (Ref. e). In humans, the theory-of-mind question was posed concretely in terms of the ability to attribute beliefs, specifically false beliefs, to other individuals. It has been shown that this ability begins to emerge around age four or possibly earlier (Refs f,g). The abilities that constitute a theory of mind have been fractionated into several distinct components, such as the ability to attribute desires, to recognize objects of shared attention, and to monitor others' direction of gaze. All these different components appear at distinct developmental stages in humans, and there is evidence that some of them may be selectively impaired in subjects with autism, a disorder that exhibits marked difficulties in social behavior (Ref. h).

Several lesion and functional imaging studies have investigated the neural structures by which subjects generate knowledge about other people's mental states. In addition to a large literature demonstrating the involvement of amygdala, orbitofrontal cortices, and right hemisphere cortices in more general processing of emotion, including recognition of emotion in others, some studies have explicitly investigated attribution of higher-order mental states, such as beliefs and intentions. A recent study by Stone *et al.* found that subjects with bilateral damage to the orbitofrontal cortex were specifically impaired in their ability to attribute higher-order mental states to other people from stories (Ref. i). In particular, they were unable to detect a *faux pas*, something that subjects with high-functioning autism (Asperger syndrome) also fail. A functional imaging study that compared brain activation during theory-of-mind tasks between normal and high-functioning autistic subjects found evidence that sectors of left medial prefrontal cortex were also important to reason about other people's mental states (Ref. j), a finding consistent with earlier studies that showed that processing words for mental states (Ref. k), or reasoning about the beliefs and intentions of others (Ref. l), normally activates regions in medial prefrontal cortex. In regard to the amygdala, an fMRI study demonstrated amygdala activation when normal subjects had to attribute mental states and intentions to other people from looking at pictures of their eyes (Ref. m). Interestingly, this is a task that high-functioning subjects with autism fail behaviorally (Ref. n), and also in which, unlike normal individuals, the amygdala does not appear to be activated (Ref. m). As far as right hemisphere somatosensory-related cortex is concerned, in addition to a large literature implicating this region in more general emotional processing, a recent lesion study showed that damage to this area can impair the ability to attribute mental states, such as false beliefs, to other individuals (Ref. o).

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in neurochemical systems relatively specialized for social behaviors (see Ref. 84 for a review).

Ultimately, one would like to see comparative investigations that examine frontal lobe structure and function in humans and other primates, but such studies are exceedingly difficult. While it is clear that primate species vary tremendously in terms of their social behaviors, it is not at all clear to what extent this variation might result from innate or from acquired factors[85], and it is also far from clear that there is any correlation between aspects of social behavior and comparative anatomy of the frontal lobes[86].

Somatosensory cortices: empathy and simulation

I have mentioned several examples of processes that all appear to operate in a relatively domain-specific fashion on socially relevant information. The examples range from specialized perceptual processing of eyes and faces to reasoning about social exchange. To qualify truly as high-level cognition, social cognition must rely on particular types of representations. Specifically, a social organism must be able to represent not only its own body states in response to conspecific stimuli, but must also possess mechanisms for constructing detailed representations of the conspecific stimuli themselves. Social

cognition should permit the construction of a mental model, a comprehensive representation, of other individuals, and of what it is about those individuals that is important to know about them as social agents who have the possibility of interacting with us.

In order to answer the question of how we represent other individuals, it is useful to consider how we represent ourselves. In fact, one line of thinking has argued that we represent the minds of others by attempting to simulate another person's state in our own brain (see Box 3). Our ability to judge other people's emotions, behavioral dispositions, beliefs and desires might draw substantially on our ability to empathize with them: that is, to create a model in our own minds of what the other person is feeling. It would seem that such an ability would be essential in order to adopt another person's point of view in a comprehensive manner, and that it would aid in the ability to predict other people's behavior.

This idea might help to explain why emotion and social cognition are closely related, not only in terms of shared processing strategies, but in fact in terms of their neural substrates: most structures important to social cognition are also important to normal emotional functioning. The common ingredient may be what we commonly call 'feeling': the representation of emotional body states, either in regard to one's own emotional reaction, or in regard to the empathy for, or simulation of, another person's internal state.

In addition to the amygdala and ventromedial frontal cortices, which can trigger emotional responses to socially relevant stimuli, there is evidence for a third important structure that contributes directly to our ability to construct representations of other individuals. In a study of subjects with focal brain lesions, we found that recognition of emotions from other people's facial expressions critically relied on the integrity of somatosensory-related cortices in the right hemisphere (including S-I, S-II, and insula[87,88]). In our study, somatosensory structures were particularly important in order to judge complex blends of multiple emotions in a single face. The finding may be explained as follows. When asked to judge the emotion shown in a face, there are at least two different strategies that could conceivably contribute to performance. Subjects might reason about the other person's emotion from knowledge regarding the facial configurations normally associated with certain emotions (e.g. reasoning that a smile signals happiness). A second strategy would be to generate somatosensory images that correspond to the way one would feel if one were making the facial expression shown in the stimulus; such a procedure might work best in cases where no prior factual knowledge is readily available (e.g. asking difficult questions concerning how much anger there is in a sad face, or an afraid face, as we did in our task). This second idea proposes that subjects judge another person's emotional state from the facial expression by reconstructing in their own brains a simulation of what the other person might be feeling. That is, subjects who are looking at pictures of facial expressions ask themselves how they would feel if they were making the facial expression shown in the stimulus (either overtly or covertly). The finding from our study is consistent with many other studies that have found social and emotional impairments following right hemisphere damage[89,90], including an impaired ability to attribute higher-order mental states to other people in theory-of-mind tasks[91] (Box 3).

Outstanding questions

- Most of the neural structures known to be important to social cognition are also important to emotion, and to associating stimuli with reward and punishment. What is the relation between social behavior, emotion, and reward/punishment? Can social cognition be thought of as an elaboration on reward mechanisms?
- What aspects of social cognition are truly unique to humans?
- What aspects of social behavior are innately specified, and what aspects are acquired through experience? Related to that, what aspects of social behavior are invariant across different cultures?
- How critical is language to social cognition? Is it possible that language evolved primarily to subserve social behavior?
- If the evolution of the human mind was driven in large part by the need for cognitive mechanisms that are socially adaptive, then it becomes interesting to consider the constraints that our social cognition might have on cognition in general. Are we limited in what we can think about, and in how we can think, by a design that has optimized human cognition for social behavior?

Conclusions

Social cognition draws upon a vast set of abilities. Some of these are quite specific to the social domain, and others may be more general in their application. Some classes of emotions, such as guilt, shame, embarrassment and jealousy, only make sense in a social context and may have evolved to subserve very specific roles in social communication. Other social signals, and other types of social judgments, draw upon systems that subserve emotional processing in general, systems that permit us to build models of other individuals through simulation, and a vast network of structures that contribute to reasoning, inference and language.

Three structures have been highlighted in this review: amygdala, ventromedial frontal cortex, and right somatosensory-related cortex. Normally, in a typical, complex, emotionally salient situation in real life, all three component structures will operate in parallel: the amygdala will provide a quick and automatic bias with respect to those aspects of the response that pertain to evaluating the potentially threatening nature of the situation, or with respect to allocating processing resources to those stimuli that are potentially important but ambiguous; ventromedial frontal cortex will associate elements of the situation with elements of previously encountered situations, and trigger a re-enactment of the corresponding emotional state; and right somatosensory-related cortices will be called upon to the extent that a detailed, comprehensive representation of the body state associated with emotional or social behavior needs to be made available. All of these components would be important to guide social behavior in a typical situation in real life, and all of them emphasize the close link between emotion and social cognition.

There is no doubt that humans differ from other animals in their social skills, in that they are able to form higher-order representations of the social environment, and to manipulate those representations in reasoning that can be quite flexible. On the other hand, there is also good evidence that our reasoning is biased in domain-specific ways, and that our judgment of other individuals, and our behavioral responses

towards them, are strongly influenced by mechanisms that we share in common with other animals. The challenge for the future will be to offer a more precise account of the interplay between all these different processes as a function of the detailed specification of the performance demands required by a given experimental task, or by a given situation in real life.

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