The neurobiology of social cognition
Ralph Adolphs

Recent studies have begun to elucidate the roles played in social cognition by specific neural structures, genes, and neurotransmitter systems. Cortical regions in the temporal lobe participate in perceiving socially relevant stimuli, whereas the amygdala, right somatosensory cortices, orbitofrontal cortices, and cingulate cortices all participate in linking perception of such stimuli to motivation, emotion, and cognition. Open questions remain about the domain-specificity of social cognition, about its overlap with emotion and with communication, and about the methods best suited for its investigation.

Introduction
The ability to recognize, manipulate, and behave with respect to socially relevant information requires neural systems that process perception of social signals and that connect such perception to motivation, emotion, and adaptive behavior (Figure 1). Social cognition guides both automatic and volitional behavior by participating in a variety of processes that modulate behavioral response: memory, decision-making, attention, motivation and emotion are all prominently recruited when socially relevant stimuli elicit behavior.

Although social cognition has been investigated for some time within developmental, comparative and social psychology, recent findings from neurobiology shed light on its neural underpinnings, and several studies are beginning to integrate neurobiological and psychological approaches [1,2•,3•]. This review will focus on work in mammals, especially primates, on the visual system and on those aspects of social cognition closely related to emotion; as such, the review will omit aspects of social communication such as language.

Evolution and development of social cognition
Many species live in societies of multiple individuals, giving rise to opposing factors that shape the evolution of their social behavior: on one hand, groups can offer better prospects for survival; on the other hand, groups can generate within-group competition between individuals. A reconciliation of these factors is found in two distinct evolutionary solutions: rigid, eusocial behavior, typically seen in insects such as bees (but also found in the rare case of the mammalian naked mole rat), or the highly complex, flexible social behavior exemplified by primates. The latter solution requires social cognition: the ability to construct representations of the relations between oneself and others, and to use those representations flexibly to guide social behavior.

Are the information-processing demands made by social cognition different from those made by non-social cognition? In general, brains provide an advantage to survival in environments in which many factors change rapidly over time, by permitting organisms to extract complex patterns that aid prediction. Compared to the physical environment in general, the social environment is more complex, less predictable, and, critically, more responsive to one’s own behavior (this applies already to the broadest and most primitive social relation — that between predator and prey). These factors, and especially the reciprocity inherent in the last one, are thought by some to have driven the evolution of our cognitive abilities. To the extent that social cognition has been shaped by evolution, it is important to keep in mind that the environment in which such evolution took place differed from our present environment [4]: early human social groups were smaller, humans were hunter–gatherers, and, of course, humans did not have available all of the modern technology that dramatically widens the scope of our social abilities. Our understanding of the evolution of social behavior, especially of phenomena such as cooperativity and altruism, has also benefited from mathematical modeling, which has demonstrated the emergence of behaviors that are stable in a population even though they may not be rational from the point of view of the individual (see e.g. [5•]). A final phenomenon worth considering is the evolution of social relationships between different species: for instance, it has been speculated that a complete account of the evolution of human social cognition could include the reciprocal social behaviors that evolved to tie humans and wolves into cooperative dual-species societies [6•].

No less important to consider is the development of social cognition, influenced by two heritable components: genes and culture. Current models of personality acknowledge the importance of innate, biological factors, but emphasize that their developmental trajectory depends on the particular cultural and social context within which a person matures [7]. The idea that the influence of genes on behavior is very context-sensitive is corroborated by studies in mice, which have documented profound differences in behavioral traits despite genetic identity [8•]. The development of social cognitive abilities is tied closely to the development of emotion and of its communication between infant and mother, a topic that has seen enormous research from developmental social psychology. Recent neurobiological studies have demonstrated that maternal behavior directly influences social development. In rats, susceptibility to stress and anxiety in the infant is influenced by the mother’s
behavior (grooming and nursing); moreover, these personality traits in the infant remain stable across the lifespan and are transmissible to future offspring [9,10]. Specific neural structures that are involved in social cognition have been shown to subserve somewhat different functions during different stages of development: damage to the frontal lobes early during development results in a more severe impairment of moral knowledge than similar damage during adulthood [11•], and the amygdala’s role in aversive conditioning only switches on some time after birth, permitting early attachment regardless of parental behavior [12]. At the genetic level, there is evidence that specific sets of genes contribute to the development of aspects of social cognition with a particular timecourse, as found in genetic diseases such as Williams syndrome [13•].

Developmental and evolutionary approaches to understanding social cognition are now being fused in some studies that combine experiments in human infants with experiments in nonhuman primates. These studies have emphasized that humans quickly develop cognitive capacities (around three to four years of age) that no other primate shares: notably, only humans appear able to adopt the point of view of another individual [14••,15], a capacity whose rudiments may already be evident in the ability of newborns to mimic some facial gestures, and that may be the catalyst for the generation of culture.

Social perception: faces and the superior temporal sulcus
How are socially relevant stimuli and signals perceived? Most mammals use olfaction and touch as key sensory channels for social communication: rat mothers identify their pups by smell, and maternal and sexual behaviors are mediated by a specialized olfactory organ, the vomeronasal organ. Auditory communication is based on often complex signals that are adapted to a species’ particular environment: whale songs that can travel enormous distances underwater, ultrasonic separation cries of small mammals that are inaudible to many predators, and highly complex songs of birds that permit distinctions to be made among many cohabiting species. Arguably, species-specific communication signals are the most common source of stimuli for social perception, a large topic that has been extensively reviewed [16,17].

Not surprisingly, the perception of social stimuli in primates has been studied most in the sensory system we understand best: vision. Single-unit studies in monkeys have demonstrated neuronal responses in temporal visual cortices that appear to encode information about highly specific social aspects of stimuli. A proportion of cells in monkey inferotemporal cortex show visual responses that are relatively selective for faces [18], for direction of gaze, for body orientation, or for intended action [19]. These findings have now been complemented by studies in humans. Electrophysiological studies in epileptic patients have found regions of the temporal cortex that respond to socially salient parts of faces, such as eyes and moving mouth parts. A collection of regions in the superior temporal sulcus is activated in response to biologically and socially salient visual motion stimuli (for a review, see [20]). Functional imaging studies have also found responses to static faces specifically in the fusiform gyrus, which have sparked a debate: are there systems in the human brain for processing specific social stimuli, such as faces [21], or are there only systems that carry out more domain-general processing, on which social and face processing may draw on?
It seems likely that both aspects have some truth to them: face-responsive regions in fusiform gyrus and superior temporal sulcus evolved as part of a distributed neural system for processing faces; however, this system does not care whether the stimuli are in fact faces, or are non-face stimuli that make similar computational demands (notably, that require expert subordinate-level categorization amongst visually similar stimuli). A recent integration of findings proposes that perception of invariant features of faces, such as identity, relies heavily on the fusiform gyrus, whereas perception of changeable aspects of faces, such as gaze and expression, relies more on regions in superior temporal sulcus [23]. A wealth of socially relevant cues can be gleaned from faces, and there is a rich literature speculating on the possible evolutionary factors that could have resulted in mechanisms to signal and to detect such cues [24].

Neural structures involved in social cognition

Perception feeds into cognition, and cognition guides both automatic and planned behavior at multiple levels of organization (Figure 1). A number of structures are now being explored with lesion studies as well as with functional imaging studies, a sampling of which is given in Table 1.

<table>
<thead>
<tr>
<th>Task</th>
<th>Activation</th>
<th>Study</th>
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<tbody>
<tr>
<td>Theory of mind (both verbal and nonverbal)</td>
<td>Medial prefrontal/cingulate</td>
<td>[75]</td>
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<td>Theory of mind (autism versus normal)</td>
<td>L medial prefrontal (in normals only)</td>
<td>[76]</td>
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<td>Theory of mind (verbal)</td>
<td>L medial prefrontal</td>
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<td>Theory of mind (visual motion of simple shapes)</td>
<td>L medial prefrontal, STS, amygdala</td>
<td>[78]</td>
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<tr>
<td>Intentions (nonverbal)</td>
<td>R medial prefrontal</td>
<td>[79]</td>
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<tr>
<td>Viewing others' hand actions</td>
<td>L frontal; R superior parietal lobule</td>
<td>[80]</td>
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<tr>
<td>Viewing others' actions</td>
<td>Motor cortex; superior parietal lobule</td>
<td>[81]</td>
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<td>Gaze and mouth movements in faces</td>
<td>STS</td>
<td>[82]</td>
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<tr>
<td>Gaze discrimination</td>
<td>L amygdala for gaze, R for eye contact</td>
<td>[31]</td>
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<td>Biological motion (pointlight displays)</td>
<td>Ventral bank of R occipital STS</td>
<td>[34]</td>
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<td>Biological motion (pointlight displays)</td>
<td>R STS, R amygdala, L parietal</td>
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<td>Facial expressions focusing on the eyes</td>
<td>Amygdala in normals, but not in autistics</td>
<td>[84]</td>
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<td>Viewing faces of different race</td>
<td>Amygdala</td>
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<td>Viewing faces of different race</td>
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<td>Faces in social phobics</td>
<td>Amygdala</td>
<td>[30]</td>
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<td>Correlation with autism symptoms</td>
<td>Medial prefrontal/cingulate, insula, STG</td>
<td>[85]</td>
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L, left; R, right; STG, superior temporal gyrus; STS, superior temporal sulcus.

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Building on a large literature that implicates the amygdala in the regulation of social behavior, recent studies have used focal excitotoxic lesions to investigate this issue in more detail. Ibogenic acid lesions that are restricted to the amygdala produce surprisingly subtle alterations in the social behavior of monkeys: the animals are somewhat more placid and less timid, and they approach novel stimuli more readily than do normal monkeys [27]. Most striking is the finding that abnormal social interactions arise mostly from unusually affiliative social behavior initiated by normal monkeys towards monkeys with amygdala damage, rather than vice versa. Apparently, normal monkeys pick up subtle cues from monkeys with amygdala damage that lead them to perceive the amygdala-lesioned animals as less threatening and more approachable [27].

In humans, the evidence is clearest for a role in response to stimuli that signal danger or threat. Lesion studies have demonstrated the amygdala’s involvement in the recognition of emotions from facial expressions, especially certain negatively valenced emotions such as fear. Subjects with bilateral damage to the amygdala judge people to look more trustworthy and more approachable than normal subjects do [28], a positive bias that extends also to non-social stimuli. The amygdala is activated in functional imaging studies when normal subjects view facial expressions of fear [29], or when socially phobic subjects view neutral faces of other people [30]; it also appears to play a role in processing the direction of gaze of others [31]. The amygdala appears most critical for recognition, rather than for expression: a study of a patient with bilateral amygdala and perirhinal cortex, also contribute to social cognition, probably by acting in concert with the amygdala (see the review by Murray, pp 188–193, this issue).
damage found that the patient was able to express emotions on her own face normally, despite a severe inability to recognize emotions from other people’s faces [32]. A new set of studies is beginning to explore the neural mechanisms whereby we perceive animacy, personality, and other social information from visual motion. Simple visual motion stimuli can produce strong percepts of animacy and intent in normal subjects [33], and viewing biological motion activates the amygdala in functional imaging [34]. Subjects with bilateral amygdala damage perceive such stimuli in abnormally geometric terms, and fail to assign normal social attributes [3•,35].

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**Right somatosensory cortices and simulation**

A large literature, primarily from human lesion studies, has implicated the right hemisphere in the processing of emotional and social information. Damage to right hemisphere neocortex impairs performance on theory-of-mind tasks, in which subjects are required to reason about the mental states and beliefs of other individuals [42,43]. A lesion study found that damage within right somatosensory-related cortices (including SI, SII, insula, and anterior supramarginal gyrus) impaired the judgment of other people’s emotional states from viewing their faces [44••]; similar findings have been obtained in regard to judging emotion from people’s tone of voice (R Adolphs et al., unpublished data). A specific impairment in both the recognition of facial expressions of disgust and the experience of disgust was found in a patient with focal damage to the left insula and putamen [45]. These findings are consistent with the hypothesis that the reconstruction of knowledge about other people’s social and emotional states might rely on a simulation of how the emotion would feel in the perceiver. It has been proposed that humans may possess specialized (and perhaps phylogenetically unique; cf. [14••]) abilities that permit perspective-taking and empathy, but there is debate concerning the precise cognitive processes that might permit such an ability: do we rely on a theory of mind, or do we draw upon simulation in order to judge how others feel? Furthermore, are there neural systems specialized for obtaining knowledge about other minds, or does this ability draw upon structures whose function is more domain-general? These issues have been reviewed in detail elsewhere [3•,46,47].

**Prefrontal cortices and somatic markers**

Prefrontal and anterior cingulate cortices have been implicated in social cognition for some time: the former was first highlighted by the famous case of Phineas Gage [48]; the latter has been the topic of a large number of functional imaging studies that have assigned emotional, attentional, and ‘executive’ functions to this area. Both regions appear to participate in response selection, decision making, and volitional control of behavior — a collection of processes that figure prominently in social behavior (see the review by Tanji, pp 164–170, this issue).

Damage to the frontal lobes, particularly to orbitofrontal cortex, results in impaired social behavior in primates. In humans, the impairment 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 [48]. Recent functional imaging studies, as well as studies in nonhuman primates, have confirmed a role for the ventral prefrontal cortices in linking interoceptive and exteroceptive information [49•], a function in which they participate together with a network of other structures, notably the amygdala and ventral striatum. The medial prefrontal cortex has been linked to theory-of-mind abilities in a number of imaging studies (see Table 1), as well as to executive functions such as self-control — two sets of abilities that appear to emerge in a concerted fashion at around four years of age. Evidence for a specific role in reasoning about the mental states of other people also comes from lesion studies: subjects with damage to orbitofrontal cortex were unable to recognize a faux pas in a story [50]. A study in a rare neurosurgical patient found single-unit responses in the orbitofrontal cortex that were selective for socially and emotionally aversive visual stimuli (pictures of mutilations and war scenes) [51], findings complementing those obtained from recordings in the orbitofrontal cortex of animals [52].

Studies using a gambling task have shown that subjects with damage to the ventromedial frontal cortex are unable to represent choice bias in the form of an emotional hunch [53], findings consistent with prior reports that subjects with such damage are unable to trigger normal emotional responses to socially relevant stimuli. Such an emotional hunch, or ‘gut feeling’, will influence behavior, and may be experienced as a feeling that one would prefer to choose one action over another, but typically is prior to overt knowledge regarding exactly why one wants to make that particular choice. These data have corroborated the somatic marker hypothesis [48,54], which proposes that the prefrontal cortex participates in implementing a particular
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Molecular and genetic factors

The molecular and genetic underpinnings of social cognition are an underexplored domain that is seeing rapid progress. Several neurotransmitters appear to play a disproportionate role in social behaviors. The hypothalamic peptides oxytocin and vasopressin mediate affiliative and sexual behaviors in several mammalian species. Voles show different mate affiliation (monogamous versus polygamous) as a result of different oxytocin systems in their brains [63], and oxytocin-knockout mice show abnormalities in their social behavior, including a social memory impairment that is specific for the odors of conspecifics [64]. It has been speculated that abnormalities in oxytocin neurotransmission may contribute to the social pathology of autism [65]. Serotonin is another neurotransmitter linked to social behavior, especially social status and dominance in primates [66]. In fact, selective reuptake inhibitors for serotonin influence social behavior in humans [67], an issue that has implications for the prescription of drugs such as Prozac. Serotonin has also received recent interest specifically in relation to its role in modulating aggressive social behavior [55], a role supported by the finding that genetic diseases affecting serotonin metabolism can result in severely altered aggression [68]. Another class of neuropeptides that figures prominently in social behavior is the endogenous opiates, which modulate circuits involved in social bonding, separation anxiety, and play. An overview of the various neurotransmitter systems involved in social behavior is given in [69].

Genetic contributions to social cognition are being explored as well. There is evidence to suggest that at least some of the differences in social cognition between males and females are genetic, as borne out by studies of individuals with Turner's syndrome [70]. There is good evidence from genetic diseases that certain sets of genes can contribute disproportionately to social cognition, rather than to other aspects of cognition. Autism (which is partly heritable) and Williams syndrome [13•] (which is entirely genetic in etiology) both feature disproportionate changes in social cognition relative to general cognition (impaired social cognition and spared social cognition, respectively). The findings are consonant with reports that over 50% of the variability in performance on theory-of-mind tasks is heritable [71], a figure similar to that for the heritability of personality (e.g. as derived from studies of monozygotic twins separated since birth; cf. [7]). As described in the Introduction, genetic and environmental factors interact in a complex fashion that often makes it impossible to trace an aspect of social cognition only to one or the other.

A systems-level view of social cognition

The processing of social information is centrally distributed in both space and time. As Figure 1 indicates, the sequence of events leading from perception of a socially relevant stimulus to the elicitation of a social behavior is complex and involves multiple interacting structures. At least three general possibilities exist for how structures
such as those shown in Figure 2 interact with other brain regions: first, they may directly modulate cognition by virtue of their extensive connectivity with high-level neocortex; second, they may modulate emotional state, which in turn can be used indirectly to modulate cognition; and third, they may directly modulate perceptual processing via feedback. The latter possibility may be a major component of aspects of social cognition such as the recognition of facial expressions, and deserves some more discussion. Initially, perceptual processing of a socially relevant stimulus (e.g. a conspecific's facial expression) in visual cortices would feed into structures such as amygdala and prefrontal cortex. The early information that these higher structures receive may be sufficient only to distinguish a few categories (for instance, threatening versus not threatening in the case of the amygdala); moreover, initial, rapid processing may be entirely outside the scope of conscious awareness (as supported by the finding that the amygdala can be activated by subliminally presented facial expressions [72]). The response in prefrontal cortex could be modulated by the amygdala’s input regarding vigilance, threat, and ambiguity concerning the stimulus (as borne out by single-unit studies in animals); and the amygdala’s response may, in turn, be modulated by the contextual and habituating input from the prefrontal cortex (see [73] for such a scheme). However, the prefrontal cortex–amygdala network does not classify the social significance of the stimulus in isolation. Rather, it feeds back onto visual cortices and contributes to the temporal evolution of a fine-grained perceptual representation there. Single-unit studies in monkeys have shown directly that neurons in inferotemporal cortex signal information about different aspects of a stimulus at different times, such that social information about a face is encoded at a later point in time than coarser information that simply distinguishes a face from a non-face stimulus [74•]. It is thus plausible that the unfolding representation of the stimulus in visual and association cortices in temporal lobe relies in part on top-down influences from structures such as amygdala and prefrontal cortex that provide information regarding the social relevance of the stimulus.

**Conclusions and future directions**

Social cognition is a domain with fuzzy boundaries and vaguely specified components. Its processes overlap substantially with those that fall under the rubrics of ‘motivation’, ‘emotion’, and ‘communication’. Structures involved in social cognition include: sensory and association neocortex for social perceptual processing (e.g. superior temporal sulcus and fusiform gyrus in the case of vision); a network consisting of amygdala, prefrontal cortex, cingulate cortex, and right somatosensory-related cortices for mediating between perception and various cognitive processing components; and hypothalamus, brainstem nuclei, basal ganglia, and motor cortices in order to effect the social behavior (Figure 1).

Questions for the future are both conceptual and methodological. To what extent does social cognition differ from non-social cognition? Are there neural systems that evolved to guide social behavior, and that are specialized to process socially relevant stimuli? What is unique about human cognition—is it to be found in our social cognitive abilities? Answers to these questions will require inputs
from multiple disciplines, and will require the integration of data from human and nonhuman animals. Our understanding will also require a better operationalization of what is to count as ‘social’, and better ways of measuring social behavior; these are issues that ethologists have confronted for some time. As we come to better understand the mechanisms and causes behind social cognition and behavior, it also becomes important to consider their impact on social policy issues — to what extent can they inform guidelines for raising children, for prescribing what is permissible, and for therapeutic intervention when the regulation of social behavior breaks down in pathological cases?

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References and recommended reading
Papers of particular interest, published within the annual period of review, have been highlighted as:
• of special interest
•• of outstanding interest
A diverse collection of recent important papers relevant to social cognition, encompassing psychological as well as neurobiological approaches. The collection focuses on papers published within the last three to four years, and covers genetic, physiological, and psychological studies in both humans and animals. The emphasis, however, is on systems-level cognitive neuroscience in humans.
A review that provides a more in-depth treatment of many of the same issues raised in the present article, with an emphasis on questions of interest to cognitive scientists. The focus is almost exclusively on humans, and much of the review deals with the amygdala, orbitofrontal cortex, and right somatosensory cortices.
A recent installment in a series of papers by Sigmund, Nowak and co-workers that describe game-theoretic models of the evolution of social behaviors such as altruism. This paper discusses the Ultimatum Game, in which two players are offered a chance to win a certain sum of money by agreeing on how to divide it. The proposer suggests how to split the sum, and the responder can accept or reject the deal. If the deal is rejected, neither player gets anything. The rational solution is for the proposer to offer the smallest possible share and for the responder to accept it. If humans play the game, however, the most frequent outcome is a fair share. An evolutionary model of this situation shows that fairness will evolve if the proposer can obtain some information on what deals the responder has accepted in the past. Hence, the evolution of fairness, similarly to the evolution of cooperation, is linked to reputation.
Beautifully illustrated and very readable, this book ranges broadly to cover the evolution of brains, but its emphasis is on primate evolution including the contribution of data from human and nonhuman animals. Our understanding of social behavior will require the integration of data from human and nonhuman animals. Our understanding will also require a better operationalization of what is to count as ‘social’, and better ways of measuring social behavior; these are issues that ethologists have confronted for some time. As we come to better understand the mechanisms and causes behind social cognition and behavior, it also becomes important to consider their impact on social policy issues — to what extent can they inform guidelines for raising children, for prescribing what is permissible, and for therapeutic intervention when the regulation of social behavior breaks down in pathological cases?

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An important study that showed that genetically identical mice could show quite different behavioral traits when tested in different laboratories, even when the task was made as similar as possible. Apparently, such behavioral traits can be extremely sensitive to even small differences in environment that cannot be controlled for, an issue that is especially applicable to the development of social behaviors.
Two patients with developmental prefrontal damage showed impaired knowledge of morality on tasks, as well as impaired social behavior in real life. These impairments were more severe than when similar damage is sustained in adulthood — further evidence that structures involved in social cognition are critical for the normal development of social and emotional behaviors.
An up-to-date and comprehensive review of Williams syndrome, including its relation to social behavior. The disease results from deletion of a somewhat-variable small set of genes on chromosome 7. The phenotype features a peculiar facial morphology, heart abnormalities, and an intriguing cognitive profile. Although the subjects are generally mentally retarded and are severely impaired in visuospatial processing, they appear remarkably capable socially. They are able to perform many social tasks, including linguistic ones, normally; in fact, they appear hypersocial in terms of their real-life interaction with adults. The disease provides something close to the converse of autism, in which sub-jects can have high general intelligence despite poor social functioning.
A controversial thesis proposing that humans possess cognitive abilities different from those of any other primate, including the ability to adopt another person’s point of view. It is proposed that this ability was initially enabled by only one or a few genetic changes, but consequently laid the foundations for the possibility of cultural evolution. The book reviews psychological and anthropological data, but not neurobiological data.
A recent review of face processing in humans. The authors propose that processing the invariant features of faces may underlie our ability to recognize the identity of people, whereas processing changeable features of faces, such as eye gaze and expression, plays a larger role in social communication. They review
evidence that the former set of processes depends critically on the fusiform gyrus, whereas the latter set of processes depends more on superior temporal sulcus. Both these regions are components of a ‘core’ system that mediates the visual analysis of faces; a more extended system includes additional cortical regions that are recruited to extract meaning from the perceptual representation.


This is the strongest evidence yet for a contribution of amygdala dysfunction to autism. The study found both neuroanatomical abnormalities using magnetic resonance imaging (MRI) and impaired processing of faces on various behavioral tasks in subjects with autism, compared to controls. The behavioral findings are supported also by [41], and show some similarity to the performances of subjects with bilateral amygdala damage.


This is the best evidence that body-related information is required to obtain knowledge of other people’s emotional states, plausibly via a process of internal simulation of what it would feel like to be the other person. The study investigated recognition of facial expressions of emotion in a large sample of 108 subjects with focal cortical damage, using a novel statistical approach. Damage that included somatosensory (S)-related cortices (SII, SII insula, or anterior super marginal gyrus) resulted in impaired recognition of emotion from visually presented facial expressions. It will be important to see these findings replicated using functional imaging methods in normal subjects.


A comprehensive collection of review articles on the orbitofrontal cortex, including anatomical and physiological studies in both animals and humans. Lesion and functional imaging studies in humans provide good evidence for the role of this region in processing emotional and social information.


69. Panksepp J: Contributions of anterior cingulate cortex to unique aspects of primate, and especially human, cognitive abilities.

71. Hughes C, Cutting AL: Selective alteration of personality and the prefrontal cortex.


An important study that found that information was carried by neuronal responses about different aspects of a stimulus at different points in time. Coarse information sufficient for discriminating a face from a non-face was encoded relatively early, whereas more fine-grained information regarding the emotion shown in the face was encoded later. The finding is consistent with the idea that visual representations can include socially relevant information, perhaps via top-down inputs from structures such as the amygdala and the prefrontal cortex.


