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CHAPTER 21

Towards a unifying neural theory of social cognition

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Abstract: Humans can effortlessly understand a lot of what is going on in other peoples' minds. Understanding the neural basis of this capacity has proven quite difficult. Since the discovery of mirror neurons, a number of successful experiments have approached the question of how we understand the actions of others from the perspective of sharing their actions. Recently we have demonstrated that a similar logic may apply to understanding the emotions and sensations of others. Here, we therefore review evidence that a single mechanism (shared circuits) applies to actions, sensations and emotions: witnessing the actions, sensations and emotions of other individuals activates brain areas normally involved in performing the same actions and feeling the same sensations and emotions. We propose that these circuits, shared between the first (I do, I feel) and third person perspective (seeing her do, seeing her feel) translate the vision and sound of what other people do and feel into the language of the observers own actions and feelings. This translation could help understand the actions and feelings of others by providing intuitive insights into their inner life. We propose a mechanism for the development of shared circuits on the basis of Hebbian learning, and underline that shared circuits could integrate with more cognitive functions during social cognitions.

Keywords: mirror system; social cognition; emotions; actions; sensations; empathy; theory of mind

Humans are exquisitely social animals. The progress of our species and technology is based on our capacity for social learning. Social learning and skilled social interactions rest upon our capacity to gain insights into the mind of others. Not surprisingly, humans are indeed excellent at understanding the inner life of others. This is exemplified in our inner experience of watching a Hollywood feature film: we relax while effortlessly attributing a vast range of emotions and motivations to the main character simply by witnessing the actions of the character, and the events that occur to him. Not only do we feel that we need very little explicit thoughts to understand the actors, we actually *share* their emotions and motivations: our hands sweat and our heart beats faster while we see actors slip off the roof, we shiver if we see an actor cut himself, we grimace in disgust as the character has to eat disgusting food. This sharing experience begs two related questions: How do we manage to slip into the skin of other people so effortlessly? Why do we *share* the experiences we observe instead of simply understanding them?

The goal of this chapter will be to propose that a single principle — shared circuits — could provide a unifying perspective on both of these questions. To foreshadow the main message of our proposal, we claim that a circuit composed of the temporal lobe (area STS (superior temporal sulcus) in monkeys or MTG (middle temporal gyrus) in humans), the rostral inferior parietal lobule (PF/IPL) and the ventral premotor cortex (F5/BA44 + 6) is involved both in our own actions and those of others, thereby forming a shared circuit for performing and observing actions. We will show

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that the somatosensory cortices are involved both in experiencing touch on our own body and in viewing other human beings or objects being touched; that the anterior cingulate and insular cortices are involved in the experience of pain, and the perception of other people's pain; and finally that the anterior insula is also involved both in the experience of disgust and in the observation of disgust in others (for the case of actions, this model is similar to those put forward by other authors: Gallese et al. (2004), Rizzolatti and Craighero (2004) and Hurley S.L. [http://www.warwick.ac.uk/staff/S.L.Hurley]).

Common to all these cases is that some of the brain areas involved in the first person perspective (I do or I feel) are also involved in the third person perspective (she does or she feels). We will argue that this sharing transforms what we see other people do or feel into something very well known to us: what we do and feel ourselves. By doing so it provides an intuitive grasp of the inner life of others.

We will review separately key evidence for shared circuits for actions, sensations and emotions. We will then show that these systems appear to generalize beyond the animate world. We will conclude by suggesting how Hebbian learning could account for the emergence of these shared circuits.

Shared circuits for actions

The first evidence that certain brain areas might be involved both in the processing of first and third person perspectives comes from the study of actions in monkeys. Understanding the actions of others is a pragmatic need of social life. Surprisingly, some areas involved in the monkey's own actions are activated by the sight of someone else's actions (Dipellegrino et al., 1992; Gallese et al., 1996). Today, we start to understand more about the circuitry that might be responsible for the emergence of this phenomenon (Keysers and Perrett, in press). Imaging studies suggest that a similar system exists in humans (see Rizzolatti and Craighero, 2004 for a review).

Primates

Three brain areas have been shown to contain neurons that are selectively activated by the sight of the actions of other individuals: the STS (Bruce et al., 1981; Perrett et al., 1985, 1989; Oram and Perrett, 1994, 1996), the anterior inferior parietal lobule (an area sometimes called 7b and sometimes PF, but the two names refer to the same area, and we will use PF in this manuscript; Gallese et al., 2002) and the ventral premotor cortex (area F5; Dipellegrino et al., 1992; Gallese et al., 1996; Rizzolatti et al., 1996; Keysers et al., 2003) (Fig. 1). These three brain areas are anatomically intercon-

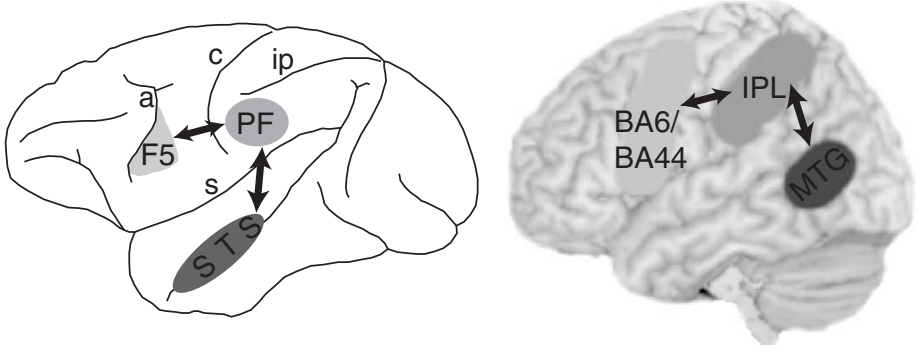

Fig. 1. (a) Lateral view of the macaque brain with the location of F5, PF and STS together with their anatomical connections (arrows). The following sulci are shown: a = arcuate, c = central, ip = intraparietal, s = sylvian sulcus. (b) Corresponding view of the human brain.

nected: STS has reciprocal connections with PF (Seltzer and Pandya, 1978; Selemon and Goldmanrakic, 1988; Harries and Perrett, 1991; Seltzer and Pandya, 1994; Rizzolatti and Matelli 2003) and PF is reciprocally connected with F5 (Matelli et al., 1986; Luppino et al., 1999; Rizzolatti and Luppino, 2001; Tanne-Gariepy et al., 2002), while there are no direct connections between F5 and the STS (see Keysers and Perrett, in press, for a recent review). All three areas contain neurons that appear to selectively respond to the sight of hand–object interactions, with particular neurons responding to the sight of particular actions, such as grasping, tearing or manipulating (Perrett et al., 1989; Dipellegrino et al., 1992; Gallese et al., 1996, 2002; Keysers et al., 2003). There is however a fundamental difference among the three areas. Virtually all neurons in F5 that respond when the monkey observes another individual perform a particular action also respond when the monkey performs the same action whether he is able to see his own actions or not (Gallese et al., 1996). These neurons called mirror neurons therefore constitute a link between what the monkey sees other people do and what the monkey does himself. A substantial number of neurons in PF shows a similar behaviour (Gallese et al., 2002). While in F5 and PF, motor information has an excitatory effect on activity, the situation in the STS is quite different. None of the neurons in the STS responding to the sight of a particular action have been shown to robustly respond when the monkey performs the same action with his eyes closed (Keysers and Perrett, in press). While some neurons in the STS respond similarly when the monkey sees himself perform an action and when it sees someone else perform the same action (Perrett et al., 1989, 1990), many actually cease to respond to the sight of their preferred movement if the monkey himself is causing this movement (Hietanen and Perrett, 1993). For these latter neurons, the motor/proprioceptive signal therefore assumes an inhibitory function, in contrast to the excitatory function observed in F5 and PF. As a result, half of the cells in the STS appear to treat self and other in similar ways, the other half of the STS sharply distinguishes other- from self-caused actions.

Considering the STS-PF-F5 circuit as a whole, we therefore have a system that responds to the actions of others. Two of its components (PF and F5) link the actions of others to our own motor programs, and may therefore give us an intuitive insight into the actions of others because they transform the sight of these actions into something very well known to ourselves: our own actions (Gallese et al., 2004; Keysers and Perrett, in press).

An essential property of mirror neurons is their congruent selectivity, namely, the fact that if they respond more to a particular action (e.g. precision grip) during execution, they also respond more to that same action during observation, compared to other actions (Gallese et al., 1996). Importantly, not all mirror neurons show the same selectivity: some are very precisely tuned for a particular action (e.g. they respond strongly to a precision grip, but not to a whole-hand prehension), while others are much more broadly tuned (responding to all kinds of grasps, but not to other actions not related to grasping). This combination of precisely and broadly tuned neurons is very important: the precisely tuned neurons can give very detailed insights into the actions of others, but require that these actions are within the motor vocabulary of the observing monkey. The more broadly tuned neurons on the other hand will also respond to the sight of novel actions that are not within the motor vocabulary of the monkey, but resemble actions that are within the monkey's vocabulary. Examples of the latter are the neurons responding to tool use, which have now been found in F5 (Ferrari et al., 2005): the monkeys used in this experiment have never used tools (e.g. a pincer) and yet the sight of someone using a tool activated some F5 neurons that responded when the monkey performed similar but different actions (grasping with its hands).

The STS-PF-F5 circuit also responds in cases where we recognize the actions of others but are unable to fully see these actions. In the STS, some neurons respond strongly to the invisible presence of a human hiding behind an occluding screen in a particular location. The same human hiding in a different location often caused no response (Baker et al., 2001; Fig. 2a). Although this capacity has been demonstrated for hidden humans, similar re-

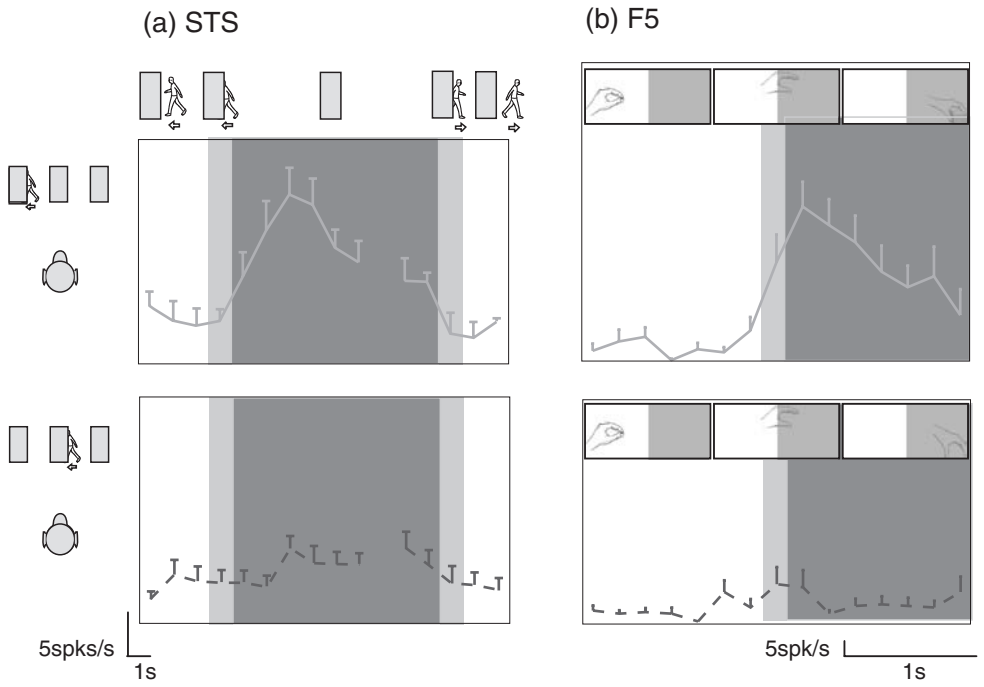

Fig. 2. (a) Response of a neuron in STS while the monkey observes a human walk towards, hide behind and then reappear from an occluding screen. The top and bottom histograms show its activity when hiding behind the left and centre occluder, respectively (see cartoon on the left). The different experimental phases are shown on top, and coded as a white background when the subject is fully visible, light grey when partially and dark grey when fully occluded by the screen. The discharge is stronger in the top compared to the bottom occluded phase although in both cases, there were only three occluders to be seen without any visible individual (Baker et al., 2001). (b) An F5 neuron while a human demonstrator grasps behind an occluding screen. In the top but not the bottom case, the monkey previously saw an object being placed on a tray before the occluder was sled in front of the tray. The discharge starting as the hand begins to be occluded (light and dark grey background) is much stronger in the top case, yet at that moment both visual stimuli (top and bottom) are equal (Umilta et al., 2001). The scales are different in (a) and (b).

sponses may exist for hidden objects. In F5, about half of the mirror neurons responding when the monkey himself grasps an object also respond to the sight of a human reaching behind an occluder but only when the monkey previously saw an object being placed behind the occluder (Umilta et al., 2001; Fig. 2b). This observation begs the question of where the information necessary for this type of F5 responses originates. As shown above, the STS could provide a representation of the reaching and a representation of the hidden object. The STS-PF-F5 circuit may then extrapolate from these two pieces of information towards a complete representation of the action, causing F5 grasping neurons to fire. The circuit is particularly well suited for such extrapolations because it is an inherent function of the premotor cortex to code movement sequences unfolding in time. The same hardware could be used to extrapolate the visible beginning of a grasping into the full action. Many important actions around us are not fully visible: a leopard may be approaching a monkey, intermittently disappearing behind trees. In such cases, understanding the leopards action, although it is not fully visible, will make the difference between life and death for the observing monkey.

Both STS and F5 also contain neurons that respond to the sound of actions. Neurons were found in the STS that respond to the sound and/or the vision of walking, with much smaller responses to other actions such as clapping hands (Fig. 3a). Similar neurons have been found in F5, but responding to seeing and/or hearing a peanut being broken (Fig. 3b; Kohler et al. 2002; Keysers et al.,

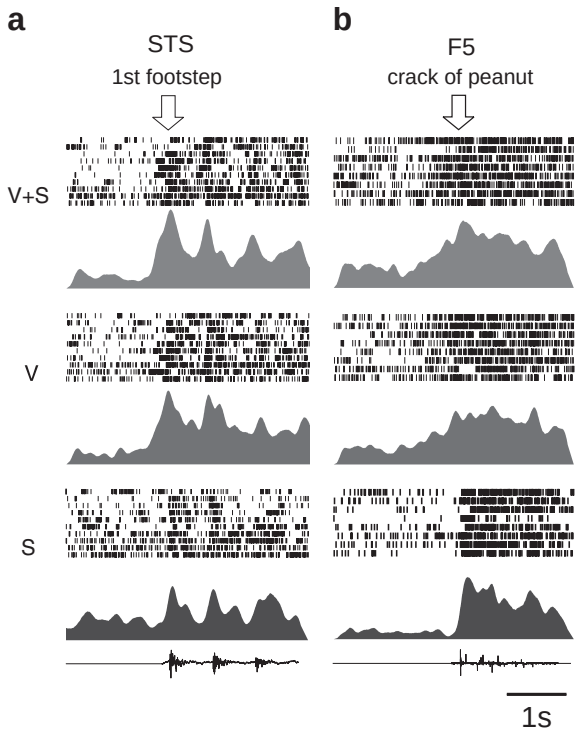

Fig. 3. (a) response of an STS neuron while the monkey heard (S = sound), saw (V = vision) or saw and heard (V + S) an experimenter walk. Note the strong response in all three cases. (b) response of an F5 neuron in the same three conditions but for the action of breaking a peanut. This neuron also responded while the monkey broke a rubber peanut out of sight. The curves at the bottom are sonographs (figure adapted from Keysers and Perrett, in press).

2003). The latter neurons in F5 also respond when the monkey breaks a rubber peanut out of sight (i.e. without sound or vision of his own action). It therefore appears as though the entire STS-PF-F5 circuit is multimodal: some of its neurons respond in similar ways to an action independently of whether it is seen or heard. Given its connections to both the auditory and visual cortices, STS appears to be a likely site for this audiovisual integration (see Ethofer and Wildgruber, this volume). In the F5-PF-STS circuit, this audiovisual action representation then appears to be integrated with the motor program of the matching action. With such a multimodal system, the mere sound of someone knocking on the door would activate a multimodal, audio-visuo motor representation of the action, leading to a deep understanding and sharing of the heard action. Indeed, mirror neurons with audiovisual properties are able to discriminate which of two actions was performed by an actor with >90% accuracy based either on the sound or the vision of the action alone (Keysers et al., 2003).

Humans

A mirror system similar to that found in the monkey has now been described in humans. Regarding the observation of actions, a number of imaging studies, including fMRI, PET and MEG experiments, have reported the three following areas being particularly involved in the observation of actions: the caudal inferior frontal gyrus and adjacent premotor cortex (Broadman areas [BAs] 44 and 6) corresponding to the monkey's area F5, the rostral inferior parietal lobule (IPL) corresponding to the monkey's area PF, and caudal sectors of the temporal lobe, in particular the posterior superior temporal sulcus (pSTS) and the adjacent MTG corresponding to the monkey's STS (see Fig. 1; Grafton et al., 1996; Rizzolatti et al., 1996; Decety et al., 1997; Grezes et al., 1998; Iacoboni et al., 1999; Nishitani and Hari, 2000; Buccino et al., 2001; Grezes et al., 2001; Iacoboni et al., 2001; Perani et al., 2001; Decety et al., 2002; Nishitani and Hari, 2002; Grezes et al., 2003; Manthey et al., 2003; Buccino et al., 2004b; Wheaton et al. 2004). Two of these three areas, the IPL and BA44/6 are known to play an important role in motor control. A smaller number of studies have also measured brain activity during the execution of actions in the same individuals in order to check if certain parts of the brain are involved both during motor execution and the observation of similar actions (Grafton et al., 1996; Iacoboni et al., 1999; Buccino et al., 2004b). These studies found sectors of the IPL and BA44/6 to be involved both in the observation and execution of actions, representing a human equivalent of the monkey's mirror neurons found in PF and F5.

The situation in the pSTS/MTG is less clear: Iacoboni et al. (2001) find the STS to be active both during motor execution and observation,

while Grafton et al. (1996) and Buccino et al. (2004b) fail to find robust STS activation during motor execution. Two explanations have been offered for this STS/MTG activation during the execution of actions. The first holds that a reference copy of executed actions is sent to congruent visual neurons in the STS/MTG to create a forward model of what the action should look like (Iacoboni et al., 2001). The second, based on the fact that in monkeys the execution of actions reduces the spiking activity of STS neurons, holds that a reference copy is sent in order to cancel the visual consequences of our own actions (Keysers and Perrett, in press). Why, though, should a reduction in spiking show up as an increase in blood oxygen level dependent (BOLD) signal? Logothetis (2003) has suggested that the BOLD effect is dominated by synaptic activity, not spiking activity; the metabolic demands of inhibitory synaptic input could thus outweigh a reduction of spiking activity and thus be measured as an overall increase in BOLD signal (but see Waldvogel et al., 2000). Either way, the STS/MTG is an important element of the 'mirror circuitry' involved both in the observation and execution of actions (Keysers and Perrett, in press).

A key property of the mirror system in monkeys is its congruent selectivity: mirror neurons responding for instance to a precision grip more than to a whole-hand prehension during motor execution also respond more to the observation of a precision grip compared to a whole-hand prehension (Gallese et al., 1996). Can the same be demonstrated for the human mirror system? A promising alley for providing proof of such selectivity stems from studies looking at somatotopy in the premotor activations. Buccino et al. (2001) and Wheaton et al. (2004) showed participants' foot, hand and mouth actions, and observed that these actions activated partially distinct cortical sites. They interpret these activations as reflecting the mapping of the observation of hand actions onto the execution of hand actions, and so on for foot and mouth. Unfortunately, neither of these studies contained motor execution tasks, and both therefore fail to establish the congruence of the somatotopical organization during observation and execution. Leslie et al. (2004) asked participants to imitate facial and manual actions, and observed the existence of patches of premotor cortex involved in either manual or facial imitation. Unfortunately, they did not separate the vision of faces/hands from the motor execution, and therefore congruent somatotopy cannot be proven by their study either. It is noteworthy, that during motor execution in other studies (e.g. Rijntjes et al., 1999; Hauk et al., 2004), a somatotopy for action execution was observed, which apparently resembled the visual one found in the above-cited studies.

Corroborating evidence for the existence of selective mirror neurons in humans stems from a number of transcranial magnetic stimulation (TMS) studies (Fadiga et al., 1995; Gangitano et al., 2001; see Fadiga et al., 2005 for a review), which suggests that observing particular hand/arm movements selectively facilitates the motor execution of the specific muscles involved in the observation.

Evidence that BA44 is essential for recognizing the actions of others comes from studies that show that patients with premotor lesions show deficits in pantomime recognition that cannot be accounted for by verbal problems alone (Bell, 1994; Halsband et al., 2001). Also, repetitive TMS induced virtual lesions of BA44 impair the capacity to imitate actions, even though they do not impair the capacity to perform the same actions when cued through spatial stimuli instead of a demonstrator's actions (Heiser et al., 2003).

The mirror system in monkeys was shown to also respond to the sound of actions (Kohler et al., 2002; Keysers et al., 2003). In a recent study, we could demonstrate that a similar system also exists in humans (Gazzola et al., 2005). In this study, the same participants were scanned during execution of hand and mouth actions and when they listened to the sound of similar actions. The entire circuit composed of MTG-IPL-BA44/6 responded both during the execution and the sound of hand and mouth actions. Most importantly, the voxels in the premotor cortex that responded more during the sound of hand actions compared to mouth actions also responded more during the execution of hand actions compared to mouth actions, and vice versa for the mouth actions, demonstrating for the first

time a somatotopical organization of the mirror system in humans, albeit for sounds.

If the observation of other individuals' actions are mapped onto our own motor programs, one may wonder how the perception of actions change when we acquire new skills. Seung et al. (2005) and Bangert et al. (2005) show that pianists demonstrate stronger activations of BA6/44, IPL and MTG while listening to piano pieces compared with nonpianists, suggesting that the acquisition of the novel motor skill of piano playing enhanced also the auditory mirror representation of these actions while listening — an observation that might relate to the fact that pianists often find it harder to keep their fingers still while listening to piano pieces. Calvo-Merino et al. (2005) showed male and female dancers' dance movements that were specific for one of the genders. They found that female dancers activated their premotor cortex more to female dance moves, and male dancers more to male dance moves. This finding is particularly important, as both male and female dancers rehearse together and have therefore similar degrees of visual expertise with both types of movements, but have motor expertise only of their own gender-specific movements. The premotor differences observed therefore truly relate to motor expertise. It is interesting, that although in both examples, responses were stronger for experts compared to nonexperts, mirror activity was not absent in people devoid of firsthand motor expertise of the precise actions they were witnessing. These weaker activations thus probably reflect the activity of more broadly tuned mirror neurons (Gallese et al., 1996) that may discharge maximally to other, similar actions (e.g. walking, jumping), but also respond slightly to these different actions (e.g. a specific dance move involving steps and jumps). With these more widely tuned neurons, we can gain insights into actions that are novel to us, by drawing on analogies with similar actions already within our motor vocabulary.

Conclusions

Both monkeys and humans appear to activate a circuit composed of temporal, parietal and frontal neurons while observing the actions of others. The frontal and parietal nodes of this circuit are active both when the subjects perform an action and when they perceive someone else perform a similar action. These nodes are therefore shared between the observation and execution of actions, and will be termed 'shared-circuits for actions'. The implications of having shared circuits for actions are widespread. By transforming the sight of someone's actions into our motor representation of these actions, we achieve a very simple and yet very powerful understanding of the actions of others (Gallese et al., 1996; Keysers, 2003; Gallese et al., 2004). In addition to providing insights into the actions of others, activating motor programs similar to the ones we have observed/heard is of obvious utility for imitating the actions of others, and shared circuits for actions have indeed been reported to be particularly active during the imitation of actions (Iacoboni et al., 1999; Buccino et al., 2004b). Finally, as will be discussed below in more detail, by associating the execution and the sound of actions, mirror neurons might be essential for the acquisition of spoken language (Kohler et al., 2002; Keysers et al., 2003).

Sensations

Observation and experience of touch

If shared circuits may be essential to our understanding of the actions of others, how about the sensations of others? If we see a spider crawling on James Bond's chest in the movie *Dr. No*, we literally shiver, as if the spider crawled on our own skin. What brain mechanisms might be responsible for this automatic sharing of the sensations of others? May shared circuits exist for the sensation of touch?

To investigate this possibility, we showed subjects movies of other subjects being touched on their legs. In control movies, the same legs were approached by an object, but never touched. In separate runs finally, we touched the legs of the participant. We found that touching the subjects' own legs activated the primary and secondary somatosensory cortex of the subjects. Most interest-

ingly, we found that large extents of the secondary somatosensory cortex also respond to the sight of someone else's legs being touched. The control movies produced much smaller activations (Fig. 4; Keysers et al., 2004a,b).

Intrigued by the observation of a patient C, who reported that when she sees someone else being touched on the face she literally feels the touch on her own skin (Blakemore et al., 2005), we scanned both C and a group of normal controls while touching them on their faces and necks. In a following session they showed video clips of someone else being touched on the same locations. As in our study, the experience of touch activated primary and secondary somatosensory cortices. During observation, they found SI and SII activation. In C, these activations were significantly stronger, potentially explaining why she literally felt the touch that happened to others.

It therefore appears as seeing someone else being touched activated a somatosensory representation of touch in the observers, as if they had been touched themselves. This finding is particularly important as it demonstrates that the concept of shared circuits put forward for actions appears to be applicable to a very different system: that of touch.

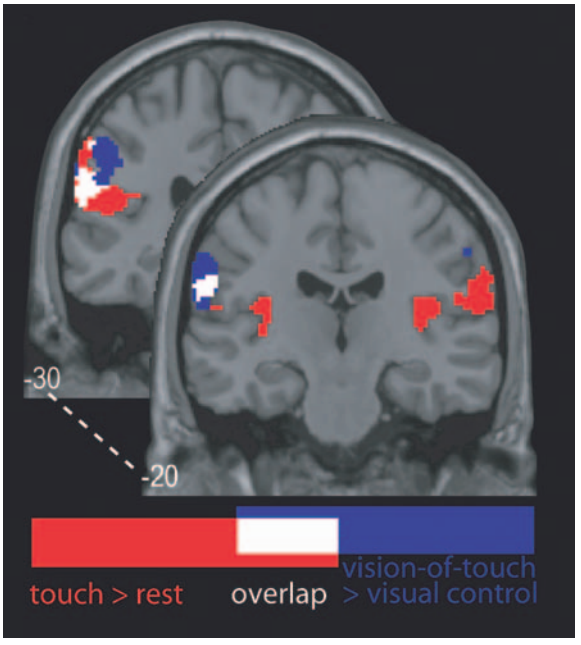

Fig. 4. Brain activity when a human is touched on his leg in the scanner (red), and when he sees another individual being touched on his leg (blue). The white voxels represent voxels active in both cases. (Adapted from Keysers et al., 2004). The right hemisphere is shown on the right of the figure (neurological conventions)

From touch to pain

Painful stimulation of the skin and the observation of a similar stimulation applied to others also appear to share a common circuitry including the anterior cingulate cortex (ACC) and the anterior insula. First, a neuron was recorded in the ACC responding both to pinpricking off the patients hand and to the sight of the surgeon pinpricking himself (Hutchison et al., 1999). Later, this anecdotic finding was corroborated by an elegant fMRI investigation, where on some trials the participant received a small electroshock on her hand; on other trials she saw a signal on a screen signifying that her partner was receiving a similar electroshock. Some voxels in the ACC and the anterior insula were activated in both cases (Singer et al., 2004), and the amount of that activation correlated with how empathic the subjects were according to two paper-and-pencil empathy scales that measure specifically how much an observer shares the emotional distress of others. The presence of activations in the anterior cingulate and anterior insula during the observation of pain occurring to others was corroborated by Jackson et al. (2005). In a TMS study, Avenanti et al. (2005) observed that observing someone else being pinpricked on his hand selectively facilitated TMS induced movements of the hand, suggesting that the sharing of pain influences the motor behaviour of the observer. This observation supports the existence of cross-talks between different shared circuits.

Emotions

The insula and disgust

Do shared circuits exist also for emotions? A series of elegant imaging studies by Phillips and collab-

orators (Phillips et al., 1997, 1998) suggested that the anterior insula is implicated in the perception of the disgusted facial expressions of others. The same area has been implicated in the experience of disgust (Small et al., 2003). In addition, both Calder et al. (2000) and Adolphs et al. (2003) reported patients with insular lesions that lost both the capacity to experience disgust and to recognize disgust in the faces of others. It therefore appears as though the insula may provide a shared circuit for the experience and the perception of disgust.

Using fMRI we measured brain activity while subjects viewed short movie clips of actors sniffing the content of a glass and reacting with a pleased, neutral or disgusted facial expression. Thereafter, we exposed the subjects to pleasant or disgusting odorants through an anaesthesia mask. The latter manipulation induced the experience of disgust in the subjects. We found that the anterior insula were activated both by the experience of disgust and the observation of the disgusted facial expressions of others (Wicker et al., 2003) (Fig. 5, yellow circles). These voxels were not significantly activated by the pleasant odorants or the vision of the pleased facial expressions of others. We then superimposed the location of the voxels involved in the experience of disgust and in the observation of disgust onto an MRI image of a patient with insular damage reporting a reduced experience of disgust and a deficient capacity to recognize disgust in others (Fig. 5, blue zone; Calder et al., 2000). The lesion encompassed our activations.

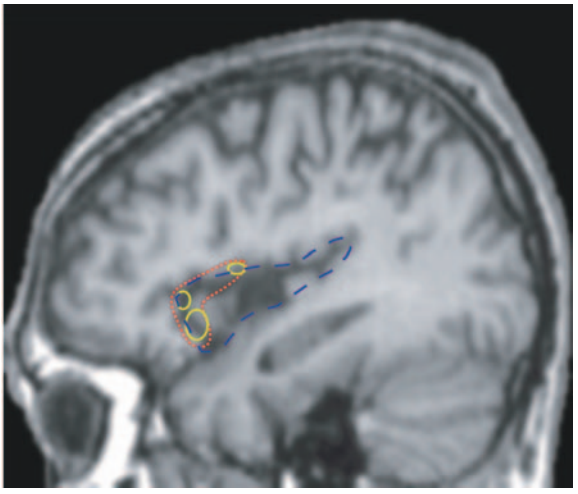

Fig. 5. agittal T1-weighted anatomical MRI of patient NK (Calder et al., 2003) normalized to MNI space. The blue outline marks the zone of the left insular infarction. The red outline shows the zone we found to be activated during the experience of disgust; the yellow outline indicates those zones found to be common to this experience and the observation of someone else's facial expression of disgust (Wicker et al., 2003). Adapted from Gallese et al. (2004).

Penfield and Faulk (1955) demonstrated that electrical stimulation of the anterior insula can cause sensations of nausea supporting the idea that the observation of the disgusted facial expressions of others actually triggered an internal representation of nausea in the participant.

It therefore appears that the anterior insula indeed forms a shared circuit for the first and third person perspective of disgust, a conclusion corroborated by electrophysiological studies (Krolak-Salmon et al., 2003). The lesion data support the idea that this circuit is indeed necessary for our understanding of disgust in others. Interestingly, just as we showed for the shared circuits for actions, the insula also appears to receive auditory information about the disgusted emotion state of others. Adolphs et al. (2003) showed that their patient B with extensive insular lesions was unable to recognize disgust, even if it was acted out with distinctive sounds of disgust, such as retching and vocal prosody. Imaging studies still fail to find insular activation to vocal expressions of disgust (Phillips et al., 1998).

The amygdala and fear

A similar logic has been put forward for the relationship between fear and the amygdala, suggesting that the amygdala responds to the sight of fearful facial expressions and during the experience of fear. According to this logic, without amygdala, both the capacity to perceive fear in the face of others and that to experience fear would be greatly affected. The state of that literature is undergoing a recent re-evaluation (Spezio and Adolphs, this volume). Below we will describe the arguments first in favour, then against the role of the amygdala as a central site both for the experience and recognition of fear.

For: Anatomically, the amygdala is linked both to face processing and to bodily states. The amygdala is a complex anatomical structure that receives highly processed sensory information from higher sensory cortices (Amaral and Price, 1984), including the temporal lobe where single neurons respond to the sight of faces and facial expressions (Perrett et al., 1984; Hasselmo et al., 1989). These connections would enable the amygdala to process facial expressions. It sends fibres back to subcortical structures such as the hypothalamus, enabling it to induce the kind of changes in the state of the body that are so typical of fear. It also sends fibres back to the cortex, including the STS, which could enable it to influence the way faces are processed.

In humans, bilateral amygdala damage does affect the capacity of subjects to recognize fear in the face of other individuals, but only in about half the subjects. A review of the literature reveals reports of 24 subjects with bilateral amygdala damage (see Table 1). When asked to rate how afraid, angry, sad, happy, disgusted or surprised the emotional facial photographs of (Ekman and Friesen, 1976) appeared, 12 of 24 subjects rated facial expressions of fear as less afraid than did control subjects without bilateral amygdala lesions (see

Table 1. The amygdala and the emotion of fear

	Damage		Ethiology	Perceptual deficits		References
Subject	Left	Right		Fear	Other	
SM	+ + +	+ + +	UW	Yes	Surprised	a,b,c,g
JM	+ + +	+ + +	E	Yes	Sad, disgusted, angry	c,g
RH	+ + +	+ + +	E	No	Angry	c,g
SE	+ + +	+ + +	E	Yes	Surprised	d,g
DR	+ +	+	S	Yes	Sad, disgusted, angry, surprised	e,g
GT	+ + +	+ + +	E	No		f,g
EP	+ + +	+ + +	E	No	Angry	f,g
SP	+ +	+ + +	S	Yes	Sad, disgusted	g
DBB	+ + +	+ +	S	No	Sad, disgusted, angry	g
NM	+ +	+ + +	?	Yes	Sad	h
SZ	+ + +	+ +		No	Angry	k
JC	+ +	+ + +	E	Yes	Angry	i
YW	+ +	+ + +	E	Yes		i
RB	+ + +	–	E	Yes		i
JK	+ +	+ +	UW	No		j
MA	+ + +	+ + +	UW	No		j
FC	+ + +	+ + +	UW	No		j
AF	+ + +	+ + +	UW	Yes		j
AW	+ + +	+ + +	UW	No		j
EW	+ + +	+ + +	UW	No		j
WS	+ + +	+ + +	UW	No		j
AvdW	+ +	+ +	UW	Yes		j
RL	+ +	+ +	UW	No		j
BR	+ + +	+ + +	UW	Yes		j

Note: A number of neuropsychological studies have asked subjects with bilateral amygdala damage to rate how afraid six photographs of the Ekman series of emotional facial expression photographs looked. Here we show a table reviewing all these studies, reporting for each patient whether he rated these facial expressions as looking less afraid than do healthy control subjects. This information is taken from the referenced publications except for patients JK to BR. For these patients, the original publication (Ref. j) reported only group data. M. Siebert and H. Markowitsch gave us the single subject ratings of their patients and healthy subjects, and we considered deficient those patients that fell below 1.64 standard deviations of the healthy controls. In total, 12 of 24 subjects with bilateral amygdala damage rated scared facial expressions as less afraid than normal subjects do. *Abbreviations:* '–': no damage, or no deficit; '+': minimal damage; '+ +': partial damage; '+ + +': extensive or complete damage; UW: Urbach-Wiethe disease, a congenital disease that causes bilateral calcifications in the amygdala; E: encephalitis, usually affecting extensive regions of the brain; S: surgical removal, usually for treatment of epilepsy. *References*: a: Adolphs et al. (1994); b: Adolphs et al. (1995); c: Adolphs et al. (1998); d: Calder et al. (1996); e: Young et al. (1995); f: Hamann et al. (1996); g: Adolphs et al. (1999); h: Sprengelmeyer et al. (1999); i: Broks et al. (1998); j: Siebert et al. (2003). k: Adolphs and Tranel (2003).

Table 1). This 'fear-blindness' was not due to general facial recognition deficits (the patients never had problems recognizing happy faces as happy), nor was it due to the patients not understanding the concept of fear (all patients specifically tested could provide plausible scenarios of situations in which people are scared). Other negative emotions such as anger were often also affected.

Imaging studies using *static* facial expressions corroborate the idea that the amygdala is important for the perception of fear in others: in the majority of the cases, the amygdala was activated preferentially when subjects viewed fearful or angry facial expressions as compared to neutral facial expressions (Zald, 2003). Studies using movies provide a different message (see below).

Lesions of the amygdala also corroborate to some extent the idea of its involvement in generating fear. Monkeys with lesions in the amygdala appear to be disinhibited: unlike their unlesioned counterparts, they immediately engage in social contacts with total strangers and in play with normally scary objects such as rubber snakes — as if, without amygdala, the monkeys fail to be scared of other individuals and objects (Amaral et al., 2003). In addition, three of the amygdala patients of Table 1 (SM, NM and YW) were tested with regards to their own emotions of fear. SM appears to have reduced psychophysiological reactions to fear (Adolphs et al., 1996); NM only remembers having been scared once in his life and enjoyed activities that would be terrifying to most of us (e.g. bear hunting in Siberia, hanging from a helicopter, Sprengelmeyer et al., 1999, p. 2455); YW did not even experience fear while being mugged at night. This suggests that without amygdala, there is something different and reduced in the subjective experience of fear.

Electrical stimulations of the amygdala in humans lead to a variety of experiences, but whenever it evoked an emotion, it was that of fear (Halgren et al., 1978). Taken together with the neuroimaging data in humans and the lesion data in monkeys, the amygdala thus appears to be important for the normal associations of stimuli with our personal, first person perspective of fear.

The role of the amygdala in experiencing fear is corroborated by a number of imaging studies. Arachnophobic individuals, when viewing spiders, experience more fear and show stronger BOLD signals in their amygdala compared with control subjects (Dilger et al., 2003). Cholecystokinin-tetrapeptide (cck-4) injections induce panic attacks that are accompanied by intense feeling of fear and cause augmentation of regional cerebral blood flow (rCBF) in the amygdala (Benkelfat et al., 1995).

The above evidence therefore suggests a role for the amygdala both in the recognition and the experience of fear. The idea of shared circuits would require that parts of the neural representations of the experience of fear should be triggered by the observation of other peoples fear. This prediction receives support from a study by Williams et al. (2001). They showed subjects Ekman faces of fear, and simultaneously recorded brain activity and skin conductance. They found that the trials in which the fear-faces produced increases of skin conductance were accompanied by increased BOLD responses in the amygdala. It therefore appears as though the vision of a fearful facial expression activates the amygdala and induces a body state of fear/arousal in the observer, as indicated by augmented skin conductance. This link between amygdala and body state is also corroborated by Anders et al. (2004).

Against: While there is evidence both from lesion studies and imaging supporting the dual role of the amygdala in experiencing and recognizing fear, there is a number of recent studies that shed doubts on this interpretation.

First, half of the patients with bilateral amygdalar lesions show no impairments in rating fear in fearful faces. Authors have failed to find etiological or anatomical differences between the patients with and without fear-blindness (Adolphs et al., 1998).

Second, a recent study on SM, one of the subjects with bilateral amygdala damage, indicate that the patient's problem in identifying the expression of fear in others is not due to an inability to recognize fear per se, but an inappropriate exploration of the stimuli (Adolphs et al., 2005): unlike control individuals, she failed to look at the eye region of photographs. If she was encouraged to do so, her fear recognition became entirely normal.

In the context of the connections of the amygdala with the STS, the function of the amygdala may not be to recognize the facial expression of fear, but to render the eye region of facial expressions a salient stimulus, selectively biasing the stimulus processing in the STS towards the eye region (see also Spezio and Adolphs, this volume).

If the amygdala is indeed not responsible for the *recognition* of fear but only in orienting visual inspection towards the eye region, one would predict equal activation of the amygdala to all facial expressions. While this is often not the case when static images of facial expressions were used (Phan et al., 2004 for a review), using short movies of facial expressions we found that the amygdala was indeed activated similarly by all facial expressions, be they emotional or not (van der Gaag et al., 2005). We used movies of happiness, disgust, fear and a neutral expression that contained as much movement as the other facial expressions (blowing up the cheeks). This finding sheds doubt on the idea of the amygdala as showing direct fear selectivity, and supports the idea of the amygdala participating in the processing of all facial expressions (for instance by biasing visual processing to the eyes). The reason why we found neutral faces to cause as much activation as emotional and fearful expressions using movies while studies using static stimuli have often reported differences remains to be fully understood. Ecologically, facial expressions are dynamic stimuli, not still photographs: the task of detecting emotions from photos is evolutionary rather new. We thus suggest that the lack of amygdalar selectivity found using movies, although needing replication, may be a more valid picture of amygdalar function than the selectivity often observed using photographs.

Doubt must also be shed on the importance of the amygdala in feeling fear. Monkeys with very early lesions in the amygdala still demonstrate signs of panic, although they occur in contexts that are not normally inducing fear (Amaral et al., 2003). In addition, there is no good evidence that patient SM completely lacks fear as an emotion, although it may well be that she does not exhibit fear appropriately in context — this is a difficult issue to measure in humans, and still remains unresolved (Adolphs, personal communication). However, Anderson and Phelps (2002) have assessed this question in patients with amygdala damage, and also found no evidence that they lack fear as an emotion. Together, it might thus be speculated that the amygdala has a role both in the normal experience of fear and in the recognition of fear in others, but that this role may be indirect, through focusing gaze on the eye region and by linking normally fear-producing stimuli with other brain areas that, in turn, are responsible for fear. The amygdala may thus be part of a circuit that enables us to share the fear of other individuals, but its role in doing so may be indirect, by biasing attention towards informative sections of facial stimuli and by relaying information towards brain areas responsible for the experience of fear. The other nodes of this circuitry remain to be investigated.

Shared circuits for actions, sensations and emotions

Subsuming the above evidence, it appears that in three systems — actions, sensations and emotions — certain brain areas are involved both in first person experience (*I* do, *I* feel) and third person perspective (knowing what *he* does or *he* feels). These areas or circuits, that we call shared circuits, are the premotor cortex and inferior parietal lobule interconnected with the STS/MTG for actions, the insula for the emotion of disgust, the ACC and the anterior insula for pain, and somatosensory cortices for touch. Possibly, the amygdala may be part of a shared circuit for fear. In all these cases, observing what other people do or feel is therefore transformed into an inner representation of what we would do or feel in a similar situation — as if we would be in the skin of the person we observe. The idea of shared circuits, initially put forward for actions (Gallese and Goldman, 1998) therefore appears much broader.

In the light of this evidence, it appears as though social situations are processed by the STS to a high degree of sophistication, including multimodal audio-visual representations of complex actions. These representations privilege the third person perspective, with lesser responses if the origin of the stimulus is endogenous. Through the recruit-

ment of shared circuits, the brain then adds specific first person elements to this description. If an action is seen, the inferior parietal and premotor areas add an inner representation of actions to the sensory third person description. If touch is witnessed, the somatosensory cortices add an inner representation of touch. If pain is witnessed, the ACC and the anterior insula add a sense of pain. If disgust is witnessed, the insula adds a sense of disgust. What emerges from the resulting neural activity is a very rich neural description of what has been perceived, adding the richness of our subjective experience of actions, emotions and sensations to the objective visual and auditory description of what has been seen.

We are not normally confused about where the third person ends and the first starts, because although the shared areas react in similar ways to our own experience and the perception of others, many other areas clearly discriminate between these two cases. Our own actions include strong M1 activation and week STS activations, while those of others fail to normally activate M1 but strongly activate the STS. When we are touched, our SI is strongly active, while it is much less active while we witness touch occurring to others. Indeed, patient C who is literally confused about who is being touched shows reliable SI activity during the sight of touch (Blakemore et al., 2005). In this context, the distinction between self and other is quite simple, but remains essential for a social cognition based on shared circuits to work (Gallese and Goldman, 1998; Decety and Sommerville, 2003). Some authors now search for brain areas that explicitly differentiate self from other. Both the right inferior parietal lobule and the posterior cingulate gyrus have been implicated in this function (Decety and Sommerville, 2003 and Vogt, 2005 for reviews)

The account based on shared representation we propose differs from those of other authors in that it does not assume that a particular modality is critical. Damasio and coworkers (Damasio, 2003) emphasize the importance of somatosensory representation, stating that it is only once our brain reaches a somatosensory representation of the body state of the person we observe that we understand the emotion he/she are undergoing. We, on the other hand, believe that somatosensory representations are important for understanding the somatosensory sensations of others, but may not be central to our understanding of other individuals' emotions and actions. The current proposal represents an extension from our own previous proposals (e.g. Gallese et al., 2004), where we emphasized the motor aspect of understanding other people. We believe that motor representations are essential for understanding the actions of others, yet the activity in somatosensory cortices observed during the observation of someone else being touched is clearly nonmotor. Instead we think that each modality (actions, sensations and emotions) is understood and shared in our brain using its own specific circuitry. The neural representation of actions, emotions and sensations that results from the recruitment of shared representations are then the intuitive key to understanding the other person, without requiring that they have to pass necessarily a somatosensory or motor common code to be interpreted.

Of course many social situations are complex, and involve multiple modalities: witnessing someone hitting his finger with a hammer contains an action, an emotion and a sensation. In most social situations, the different shared circuits mentioned above thus work in concert.

Once shared circuits have transformed the actions, sensations and emotions of others into our own representations of actions, sensations and emotions, understanding other people's boils down to understanding ourselves — our own actions, sensations and emotions, an aspect that we will return to later in relation to theory of mind.

Demystifying shared circuits through a hebbian perspective

Neuroscientific evidence for the existence of shared circuits is rapidly accumulating. The importance of these circuits for social cognitions is evident. Yet, for many readers, the existence of single neurons responding to the sight, sound and execution of an action — to take a single example — remains a very odd observation. How can single neurons with such marvellous capacities emerge? The plau-

sibility of a neuroscientific account of social cognitions based on shared circuits stands and falls with our capacity to give a plausible explanation of how such neurons can emerge. As outlined in detail elsewhere (Keysers and Perrett, in press), we propose that shared circuits are a simple consequence of observing ourselves and others (please refer to Keysers and Perrett, in press, for citations supporting the claims put forward below).

When they are young, monkeys and humans spend a lot of time watching themselves. Each time, the child's hand wraps around an object, and brings it towards him, a particular set of neural activities overlaps in time. Neurons in the premotor cortex responsible for the execution of this action will be active at the same time as the audiovisual neurons in the STS responding to the sight and sound of grasping. Given that STS and F5 are connected through PF, ideal Hebbian learning conditions are met: what fires together wires together. As a result, the synapses going from STS grasping neurons to PF and then F5 will be strengthened as the grasping neurons at all three levels will be repeatedly coactive. After repeated self-observation, neurons in F5 receiving the enhanced input from STS will fire at the mere sight of grasping. Given that many neurons in the STS show reasonably viewpoint-invariant responses, responding in similar ways to views of a hand taken from different perspective, the sight of someone else grasping in similar ways then suffices to activate F5 mirror neurons. All that is required for the emergence of such mirror responses is the availability of connections between STS-PF-F5 that can show Hebbian learning, and there is evidence that Hebbian learning can occur in many places in the neocortex (Bi and Poo, 2001).

The same Hebbian argument can be applied to the case of sensations and emotions. While seeing ourselves being touched, somatosensory activations overlap in time with visual descriptions of an object moving towards and touching our body. After Hebbian association the sight of someone else being touched can trigger somatosensory activations (Keysers et al., 2004a,b; Blakemore et al., 2005).

Multimodal responses are particularly important for cases where we do not usually see our own actions. How, for example, associating the sight of someone's lip movements with our own lip movements is an important step in language acquisition. How can we link the sight of another individual's mouth producing a particular sound with our own motor programs given that we cannot usually see our own mouth movements? While seeing other individuals producing certain sounds with their mouth, the sound and sight of the action are correlated in time, and can lead to STS multimodal neurons. During our own attempts to produce sounds with our mouth, the sound and the motor program are correlated in time. As the sound will recruit multimodal neurons in the STS, the established link also ties the sight of other people producing similar sounds to our motor program. The visual information thereby rides on the wave of the auditory associations (Keysers and Perrett, in press).

The case of emotions might be similar, yet slightly more difficult. How can the sight of a disgusted facial expression trigger our own emotion of disgust, despite the fact that we do not usually see our own disgusted facial expression? First, disgust can often have a cause that will trigger simultaneous disgust in many individuals (e.g., a disgusting smell). In this case, one's own disgust then correlates directly with the disgusted facial expression of others. Second, in parent–child relationships, facial imitation is a prominent observation (e.g. Stern, 2000). In our Hebbian perspective, this imitation means that the parent acts as a mirror for the facial expression of the child, leading again to the required correlation between the child's own emotion and the facial expression of that emotion in others. As described above, the insula indeed receives the required visual input from the STS, where neurons have been shown to respond to facial expressions (Mufson and Mesulam, 1982). The insula also receives highly processed somatosensory information about our own facial expressions. These somatosensory data will be Hebbianly associated with both the sight of other individual's facial expressions and our own state of disgust. After that Hebbian training, seeing someone else's facial expressions may trigger a neuronal representation of the somatosensory components of our own matching facial expres-

sions. The debilitating effect of somatosensory lesions in understanding the emotions of others (Adolphs et al., 2000) may indicate that this triggering is indeed important in understanding the emotions of others.

To summarize, Hebbian association (a simple and molecularly well-understood process) can therefore predict the emergence of associations between the first and third person perspective of actions, sensations and emotions.

Shared circuits and the inanimate world

The world around us is not inhabited only by other human beings: we often witness events that occur to inanimate objects. Do the shared circuits we described above react to the sight of an inanimate object performing actions or being touched?

To investigate the first question, we showed subjects movies of an industrial robot interacting with everyday life objects (Gazzola et al., 2004, *Society for Cognitive Neuroscience Annual Meeting*). The robot for instance was grasping a wine glass or closing a salt box. These actions were contrasted against the sight of a human performing the same actions. Fig. 6a illustrates a frame from a typical stimulus, as well as the BOLD signal measured in BA 44 as defined by the probabilistic maps (Amunts et al., 1999). As seen in 1.2, this area has been shown to be activated both during the execution of actions and the observation of another human being performing a similar action. Here, we see that the same area was also activated during the sight of a robot performing an action involving everyday objects. This result is in contrast with previous reports in the literature that failed to find premotor activation to the sight of a robot performing actions (Tai et al., 2004). Our experiment differs in some important aspects from these studies: first, we used more complex actions

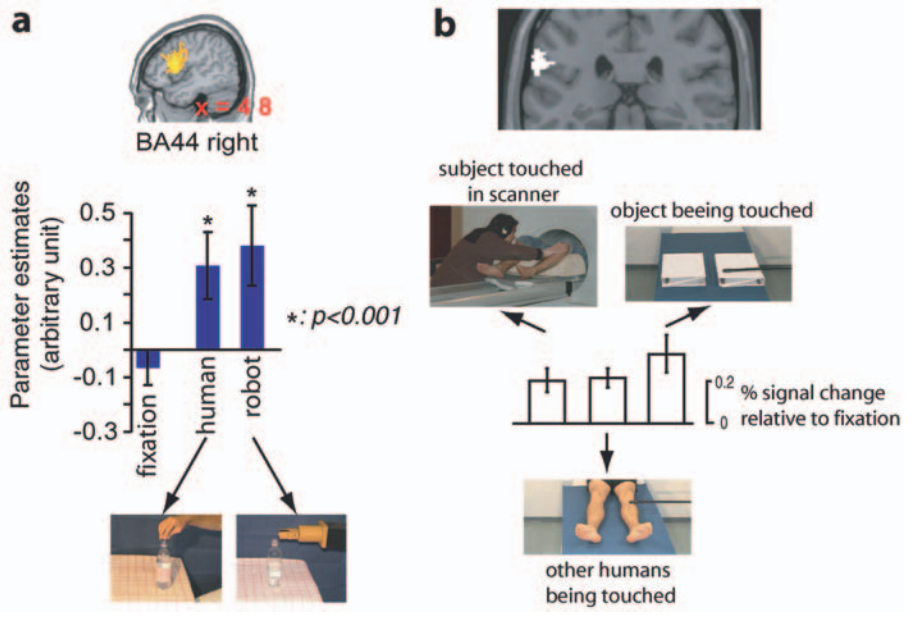

Fig. 6. (a) Top: location of the right BA44 according to Amunts et al. (1999), defined as the voxels where at least 1 of her 10 subjects satisfied the cytoarchitectonic criterions for BA44. Below: the brain activity in this right BA44 for 14 subjects, expressed in terms of parameter estimates in the GLM while subjects looked at a fixation cross or at a human or a robot opening a bottle. A star indicates significant differences from the fixation. (b) Location of the region of interest (white) defined in Keysers et al. (2004), and below, the mean BOLD signal of eight independent subjects while being touched on their legs, seeing another human being touched, and seeing objects being touched. All three cases differ significantly from fixation, but not from one another (adapted from Keysers et al., 2004). All error bars refer to standard error of the means.

instead of grasping of a ball; second our blocks contained a variety of actions and not the same action repeated over and over again. Both of these factors could account for the observed difference. In the light of our results, it thus appears as though the shared circuit for actions responds to complex meaningful actions regardless of whether they are performed by humans and robots. Half way along this human–robot continuum, the premotor cortex also responds to the sight of animals from another species performing actions that resemble ours, such as biting (Buccino et al., 2004a).

To test whether the secondary somatosensory cortex responds to the sight of objects being touched, we showed subjects movies of inanimate objects such as ring binders and rolls of paper towels being touched by a variety of rods. These conditions were compared against the activity when the subject himself was touched, and when he saw another human leg being touched in similar ways. Results, shown in Fig. 6b indicate that the SII/PV complex was at least as activated by the sight of objects being touched as by the sight of humans being touched. Blakemore et al. (2005) also showed movies of objects being touched, and found that compared to seeing a human face being touched, seeing a fan being touched induced smaller activations. Unfortunately, the authors did not indicate if the activation to seeing an object being touched was significant. Why Blakemore et al. found stronger activity to the sight of a human face being touched compared to an object, while we found similar activity to a human leg being touched compared to toilet paper rolls remains to be investigated.

Together, data appear to emerge suggesting that the sight of the actions and tactile 'experiences' of the inanimate world may be transformed into our own experience of these actions and sensations, but further investigations of this aspect are important considering the somewhat contradictory findings from different studies.

Shared circuits and communication

We show that the brain appears to automatically transform the visual and auditory descriptions of the actions, sensations and emotions of others into neural representations normally associated with our own execution of similar actions, and our own experience of similar sensations and emotions. Hebbian learning could explain how these automatic associations arise. Once these associations have been learned, they transform what other people do and feel into our own experience of these actions, sensations and emotions. This transformation represents a intuitive and powerful form of communication: it transmits the experience of doing and feeling from one brain to another. This simple form of communication has obvious adaptive value: being able to peek into someone else's mind, and to share his experiences renders constructive social interactions faster and more effective. For instance, sharing the disgust of a conspecific probing a potential source of food will prevent the observer from tasting potentially damaging items.

Most forms of communication have a fundamental problem: the sender transforms a content into a certain transmittable form according to a certain encoding procedure. The receiver then receives the encoded message, and has to transform it back into the original content. How does the receiver learn how to decode the message? When we learn a spoken language we spend years of our life guessing this encoding/decoding procedure. For the case of actions, the shared circuits we propose use the correlation in time in the STS-PF-F5 circuit during self-observation to determine the reciprocal relationship between the motor representation of actions and their audio-visual consequences. Similar procedures may apply to sensations and emotions. The acquired reciprocal relationships can then be used to decode the motor, somatosensory and emotional contents contained in the behaviour of other individuals and in the situation they are exposed to (see also Leiberg and Anders, this volume).

When dealing with other human beings this decoding procedure is generally very successful. Our brain appears to use the same procedure to understand members of other species and even inanimate objects and robots. In the case of members of other animal species, the decoded motivations, emotions and feelings are anthropocentric, and

imperfect: when monkeys for instance open their lips to show their teeth, interpreting this as a smile is a misinterpretation — it is actually a display of threat. Often such interpretations will enable us to predict the forthcoming behaviour of the animal better than if we make no interpretation at all. In the case of inanimate objects, the interpretations are very often wrong (e.g. the ring binders probably 'felt' nothing when being touched, and the robot was not thirsty when it grasped for the glass of whine). This overgeneralization may simply be a bug in our brain. Alternatively, overall, it might be better to apply the rule of the thumb: everything is probably a bit like myself, than to make no assumption at all. A clear implication of this tendency is that to make the human–machine communication as smooth as possible, robots should be made to behave as similarly to humans as possible.

The limits of simulation — a word of caution

The shared circuits we describe have received considerable interest. Often they now tend to be seen as a panacea to explain any issues of social cognition. It is important to note that while we believe shared circuits to be very important for our intuition of the inner life of others, they cannot explain everything.

We can for instance try to imagine what it feels like to fly like a bird, although we do not have the motor programs to do so. Such abstract imaginations are detached from our own bodily experience, and should thus not be attributed to shared circuits. We can of course imagine what it feels like to flap our hands, as kids do to pretend to fly, but that would still leave us with doubts about what real flying feels like.

These limitations are often cruelly clear to us during imitation: we have often seen our mothers knit, feeling that we can truly understand the movement, yet when we tried for the first time to knit something ourselves, we realise that our understanding had been quite superficial indeed, as we were lacking the true motor programs on which to mirror the actions we saw.

But even with the required motor skills, we do not understand all the inner life of other human beings through shared circuits. C. Keysers, E. Kohler and M. Goodale (unpublished observation) have for instance examined brain activity while watching the eye movements of other individuals in the hope to find evidence that brain areas such as the frontal eye field (FEF) normally responsible for our own eye movements are critical for our understanding of the eye movements of others. We found very little evidence for such a system: the sight of eye movements activated the FEF no more than the sight of random dots moving by the same amount. Despite the difficulty of interpreting negative results, this finding is not too surprising: if two people face each other, and one suddenly stares at the wall behind the other person, the other person will tend to look behind him. The motor programs involved are very different: a very small saccade for the first individual, and a turning of the head and torso for the second. There being so little in common in motor terms, it makes no sense to analyse the gaze direction of others through one's own motor programs. An external frame of reference, and an analysis of gaze in this external frame is needed to understand what the other person is looking at — a task that our motor system, working in egocentric coordinates, is very poorly equipped for. Shared circuits and mirror neurons therefore have little to contribute to this task. It will remain for future research to outline the limits of what shared circuits can explain.

Simulation and theory of mind — a hypothesis

Social cognitions are not restricted to the simulations that shared circuits provide. Explicit thoughts exist in humans and clearly supplement these automatic simulations. It is hard for instance to imagine how a gossip of the type: 'Did you know that Marry still believes that her husband is faithful while everyone else knows that he is having an affair with another women every week?' can be the result of simulation, yet thinking about the (false) beliefs of others is clearly an important part of our social intelligence.

The words theory of mind (ToM) and 'mentalizing' have often been used to describe the set of cognitive skills involved in thinking about the mind of others, in particular their beliefs (Frith and Frith, 2003). People are considered to have a ToM if they are able to deal with the fact that other people can have beliefs that differ from reality, a capacity that is tested with so called false belief tasks such as the famous 'Sally and Anne' test (Baron-Cohen et al., 1985). In that test, an observer sees Sally hide an object in a basket. Sally then goes away for a while, and unbeknown to her, Anne moves the object from the basket into a box. Sally then returns, and the observer is asked: 'where will Sally [first] look for her object?' If the observer answers 'in the basket, because she doesn't know that Anne moved it', the observer is thought to have a ToM. If the answer is 'in the box', the observer failed. Children from the age of 4 years pass this test, while autistic individuals often fail the test even in their teens (Baron-Cohen et al., 1985).

Comparing this ToM task with the tasks involved in the neuroimaging of shared circuits, it is quite clear that these two fields of research tap into phenomena that differ dramatically in the amount of explicit thoughts that are involved (see also Leiberg and Anders this volume). In research on shared circuits, subjects simply watch short video clips of actions, emotions and sensations, without being asked to reflect upon the meaning of these stimuli or the beliefs and thoughts of the actors. In ToM tasks, subjects are directly invited to reflect about states of minds of others. Strangely enough, a number of authors have introduced a dichotomy between simulation processes and more theory driven processes involved in understanding others (e.g. Gallese and Goldman, 1998; Saxe, 2005), suggesting that either ToM or simulation should explain all of social cognitions. Here we will attempt to provide a model that proposes that ToM might utilize simulations to reflect on the mind of others.

ToM-type tests have now been investigated a number of times using fMRI and PET (see Frith and Frith, 2003 for a comprehensive review), and all tasks have activated medial prefrontal cortex (mPFC) compared to conditions requiring less mentalizing. What is intriguing, is that similar sites of the mPFC are also involved in reflecting about ourselves and our own emotions (Gusnard et al., 2001; Frith and Frith, 2003), which lead Uta and Frith to speculate that thinking about other people's minds might be a process related to thinking about one's self. If seen in the context of shared circuits, this leads to a simple framework for associating shared circuits and ToM. The mPFC may originally interpret states of our own mind and body, as evidenced by experiments such as that of Gusnard et al. (2001). In this experiment, subjects saw emotional pictures, and were asked to judge if the image evoked pleasant or unpleasant emotions in themselves, or whether the images were taken indoors or outdoors. The mPFC was more activated in the former task, where subjects had to introspect their own emotions.

The mPFC receives indirect inputs about all aspects of our own body, including motor, somatosensory and visceral representations, which could allow it to create secondary representation of our bodily state (Frith and Frith, 2003). Considering our first person perspective, one could thus differentiate a first level representation, being our actions, emotions and sensations as they occur, and a second level representation in the mPFC of these states more related to our conscious understanding and interpretation of ourselves. To illustrate that difference, if you vomit, you will feel disgust, and activate your insula (primary representation). If asked what you feel, you may start reflecting upon what you are feeling in a more conscious way, one that you can formulate in words ('I felt like having a stone in my stomach, I guess those mussels weren't that fresh…') and you are likely to activate your mPFC in addition to the primary representations.

This is where simulation ties into the concepts of theory of mind. Through the shared circuits we have described, the actions, emotions and sensations of others are 'translated' into the neural language of our own actions, emotions and sensations. By doing so, they have been transformed into what we called primary representations of these states. This could generate an implicit sharing and hence understanding of the states of others. If asked explicitly what went on in

the mind of that other person, you would need to generate a secondary, more conscious and cognitive representation of his state. Given that his state has already been translated into the language of our own states, one may hypothesize, that this task would now be no different from reflecting about your own states, and therefore activate the same mPFC sites. Testing this hypothesis directly will be an exciting issue in future neuroimaging work.

In this concept, shared circuits act like a translator, converting the states of others into our own primary state representations. Often social processing can stop at that: we share some of the states of our partner, her/his sadness or happiness for instance, without thinking any further. These are the cases that simulation proponents have concentrated on. In some cases, we might reflect further upon her/his mind, just like we often reflect about our own states. Such reflections then provide much more elaborate, cognitive and differentiated understandings of other individuals. These latter are the processes that ToM investigators are excited about.

With those mentalizing processes on top of simulation, thinking about others can reach levels of sophistications that go far beyond using simulation alone. Using simulation, we inherently assume that we are all equal. This is not the case: actions that may make us happy may make other people sad, reflecting biological and cultural differences, and keeping in mind those differences may be a critical role for higher processes (see also Leiberg and Anders, this volume).

Uncited References

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