Impairments of emotion and social behaviour are often observed after damage to the ventromedial (VM) region of the prefrontal cortex [Brodmann areas (BA) 25, lower 24, 32, and medial aspect of 11, 12 and 10] (Fig. 1). Previously well-adapted individuals become unable to observe social conventions and decide advantageously on personal matters. Their ability to express emotion and to experience feelings in appropriate social situations becomes compromised. Studies aimed at understanding the nature of these deficits revealed that impaired ‘judgement and decision-making’ is at the heart of the problem (Bechara et al., 2002). Specifically, evidence suggests that the VM region serves as a link between (i) a certain category of event based on memory records in high order association cortices and (ii) effector structures that produce an emotional response, and also (iii) the substrates of feeling. During judgement/decision-making, category events are brought to ‘working memory’, which includes several processes (Baddeley, 1992). However, maintaining an active representation of memory over a delay period involves the dorsolateral sector of the prefrontal cortex (Fuster, 1997). Effector structures that mediate the emotional response are in the brainstem, whereas neural representations of feelings are thought to involve the insula, surrounding parietal cortices, and the cingulate (Damasio, 1994).

Thus judgement/decision-making depends on systems for memory, emotion and feeling. Damage to the systems that impact emotion, feeling and/or memory compromise the ability to make advantageous decisions. The VM region links these systems together, and therefore when damaged there are many manifestations, including alterations of emotional experience and social functioning. However, the cognitive processes that subserve the extremely diverse and flexible social behaviours are complex, and very little is known about their neural substrates. In this issue of *Brain*, Berthoz and colleagues (Berthoz et al., 2002) have taken the lead in tackling this important challenge. Using functional MRI methodology, they borrowed concepts from social cognition and sought to identify neural systems involved in the processing of transgressions of social norms.

In the experiment, subjects were instructed to imagine what they would feel if they were in the situation described in a story incorporating: (i) a normal social situation; (ii) an unintentional violation of social norms, e.g. choking on food during a social dinner; and (iii) an intentional violation of social norms, e.g. purposefully spitting food on a plate during a social dinner. Each story was delivered in: (i) a personal condition, where the events were described as they occurred to the subject; and (ii) an impersonal condition, where the events were described as they occurred to another person. Presumably, there were no differences between the personal and impersonal conditions, so the data from the two conditions were collapsed. The difference in brain activity was between intentional and unintentional violations of social norms on one hand versus normal social situations on the other. Except for slight differences (e.g. BA 21), the activation sites in the intentional and unintentional conditions were by and large very similar.

Surprisingly, increased activity was not observed in the VM region, but it was observed bilaterally in a more dorsal region of the medial prefrontal cortex (the medial aspects of BA 6, 8 and 9), and in the temporal poles (BA 38). Both regions overlap with areas engaged in Theory of Mind, i.e. representing the mental states of others. However, these regions are also known to play roles in mechanisms of memory, emotion and decision-making. Hence the question: does Theory of Mind, processing of transgressions of social norms, and other mechanisms of social cognition draw upon neural processes specialized for social information, or do they depend on known mechanisms of memory, emotion, decision-making and other basic functions?

Indeed, the dorsal part of the medial prefrontal region as a whole (including the anterior cingulate) forms a bottleneck region where systems concerned with movement, emotion, attention and working memory interact (Damasio, 1994). Patients with bilateral lesions of this region suffer from ‘akinetic mutism’, in which the patient ceases to attend to external stimuli, movements become very limited, and facial expressions are blank reflecting no signs of emotion and feeling (Damasio, 1994; McPherson and Cummings, 2002). Similarly, the temporal pole region overlaps with systems involved in memory retrieval. Patients with such lesions have difficulties retrieving memories of unique entities (Damasio et al., 1996).

A similar question can be asked with regard to the observed activity in the lateral aspect of the left orbitofrontal cortex (BAs 47 and 10), which has been shown to respond to...
expressions of anger. This region is also implicated in mechanisms of ‘impulse control or response inhibition’ (Rahman et al., 2001). There are several mechanisms of impulse control that can be measured by different tasks and linked to different prefrontal regions (Bechara et al., 2002). Some researchers describe these mechanisms of inhibitory control as ‘executive functions’ or ‘executive processes’ of working memory. It is important to note that although all these mechanisms are inter-related, decision-making was shown as distinct from working memory and mechanisms of impulse control both cognitively and anatomically (Bechara et al., 2002).

Despite its known role in emotion and social behaviour (Adolphs et al., 1994), activity was not observed in the amygdala, which is consistent with studies showing failure of amygdala activation under similar conditions, i.e. when the emotion is triggered by ‘mental imagery’ (Damasio et al., 2000).

Thus processing transgressions of social norms involve neural systems implicated in Theory of Mind and in responding to emotional expressions of others. However, the same neural systems are involved in working memory, impulse control and decision-making. Can social cognition be decomposed into these more basic mechanisms, which are rooted in neuroscience, in both human and animal studies? What are the manifestations at the social level if any of these mechanisms were impaired? Many instances of social cognition involve ‘judgement/decision-making’ about social conventions, intentions of others, trust, morals, ethics, and so on. Is the mechanism for these judgments specialized for social information and independent of the one described earlier in VM patients? Or can we tie all these instances to known mechanisms of decision-making, albeit that judgment may take different forms, just like working memory has different modules of (e.g. spatial, object, etc.) and impulse control has different mechanisms? These are important questions to consider because introducing functional terms that are mere descriptions of the social behaviour modelled by a given task without tying the behaviour to its root components, or using different languages to describe the

Fig. 1 A BA map with its different areas designated by numbers. Courtesy of Hanna Damasio.
same neural system in two different fields, would inevitably lead to confusion and poor understanding of the neurology of social cognition.

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References


