

Understanding Other Minds: Perspectives from developmental social neuroscience

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Understanding emotional and cognitive empathy

A neuropsychological perspective

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Abstract and Keywords

Empathy is a broad concept that refers to the cognitive as well as the emotional reactions of one individual to the observed experiences of another. Questions regarding how we understand others have intrigued psychologists and philosophers for centuries. In order to answer these questions, two major theories have been proposed, known as Theory Theory and Simulation Theory. In the past two decades, these questions have been re-examined by neuropsychologists and neuroscientists. This chapter reviews the different aspects of emotional and cognitive empathy in light of converging evidence from lesion patients, electrophysiology and neuroimaging studies. Neuropsychiatric disorders, such as Autism or Schizophrenia, although heterogeneous and difficult to study, have also been examined in relation to their deficits in cognitive and emotional empathy, and some of these new findings are discussed in this chapter. Lastly, we propose a model which relates brain mechanisms such as simulation or cognitive and affective ToM, to psychological processes, the empathic responses they lead to, and deficits which may occur when these mechanisms are disrupted.

Keywords: Cognitive empathy, emotional empathy, Theory of Mind, mirror neurons, mentalizing, medial prefrontal cortex

Awful disasters descended upon FJ. She was happily married to MJ, and they had three lovely children, until one day MJ said he decided to leave her. But that was only the beginning—for the next few months he started verbally abusing her and her children (which he never did before), left the house, abandoned all his responsibilities to others—taking the children from school, answering phone calls, cutting back on work, etc. FJ refused to accept that her marriage had suddenly collapsed, and that it was beyond mending. Whatever FJ tried, she felt she just couldn't get through to MJ. For friends around them, this was another typical divorce story, but FJ kept saying she felt MJ has gradually become a completely different person—this was not the MJ she fell in love with, married and raised a family with. They went through a terrible divorce, at times involving the police and social workers in the community. A few months following the divorce, MJ was hospitalized with a chronic headache and a CT scan found a large tumor in his frontal lobe, which had probably been there and growing for the last couple of years. In a way, FJ had been right; this wasn't the MJ she married.

Probably every first year Psychology or Neuroscience student is familiar with the famous case report of Phineas Gage, which presented one of the first descriptions of aberrant social conduct following brain damage (Harlow, 1868). Neuroscience research since Harlow's time has shown that impaired empathic abilities among people with different brain lesions, tumors and pathologies may account in part for the social and behavioral disturbances often observed among such patients. These seminal studies have a two-way effect—they further our knowledge of brain networks that enable empathy, and they may contribute to future diagnosis and therapeutic development of different social deficits and pathologies. Importantly, progress has been made in the past decade, both in the psychological terminology and through neuroimaging studies, in differentiating between sub-components of empathy, i.e. emotional and cognitive empathy, and further differentiating cognitive empathy to its' affective and cognitive components. This chapter will provide a theoretical framework for understanding of emotional and cognitive empathy. Combining the findings from lesion studies, electrophysiology and imaging data of healthy and patient groups, a neuroanatomical model for empathy and its relationship with theory of mind will be proposed.

How do we normally empathize with others? A psychological background
Empathy is a broad concept, which generally denotes our ability to identify with or to feel what the other is feeling. In order to empathize with the other, we need to first understand *what* that person (**p.179**) is going through, feeling or thinking. Two main psychological approaches have dominated the discussion of how we understand others' minds—simulation-theory and theory-theory (Carruthers, 1996; Davies & Stone, 1995). Simulation-theory suggests that we use our own mental mechanisms to estimate and predict the mental processes of others. According to this theory, we automatically create within ourselves a

simulation of the others' motor acts, and in accordance with these acts, we feel the desires, preferences, and beliefs of the sort we assume the other to have. Instead of acting on that decision, it is taken "off-line," and used to predict the intention and emotions of the other (Gallese & Goldman, 1998; Goldman, 1989; Gordon, 1986; Heal, 1986; Kosslyn, 1978; Stich, 1992).

The second theory, theory-theory, posits that children, as they grow up, use the same cognitive mechanisms that adults use in science, that is they develop theories. These theories help them investigate the world around them, infer from one situation to the next, and predict others' behaviors and emotions. In other words, understanding others and empathizing with them arise from **theoretical** reasoning involving known causal laws, or knowledge of how people in general, or people with the other's specific characteristics, are likely to think or feel (Churchland, 1988; Fodor, 1987; Gopnik, 1993; Lewis, 1972; Wellman, 1990). This ability, of attributing mental states such as beliefs, intents, desires, pretending, knowledge, etc., to oneself and others, and understanding that others have beliefs, desires and intentions that may be different from one's own has also been labeled theory of mind (ToM; Frith & Frith, 1999; Premack & Woodruff, 1978).

Developmental studies indicate that emotional contagion (e.g. contagious crying) is observed very early in young babies (e.g. Simner, 1971), while cognitive perspective taking abilities, ascribing agency, understanding intentions, and joint attention skills are acquired during later cognitive development (e.g. Carpenter, Nagell, and Tomasello, 1998; Johnson, 2003). More advanced ToM skills usually appear only by 3 or 4 years of age. These include understanding "false-belief", i.e. that the other may have a belief that you know is false (e.g. (Flavell, Everett, Croft, & Flavell, 1981; Flavell, Flavell, & Green, 1983; Wimmer & Perner, 1983). In a typical false-belief task (also known as the "Sally-Anne task"), the child is asked where a person (e.g. Sally) will look for a toy that she left in a certain place and that was moved by another person (e.g. Anne) when Sally was out of the room. Understanding that Sally will not know the new location of the toy relies on the ability to distinguish between Sally's false belief and reality (Wimmer & Perner, 1983).

Current evolutionary evidence also supports the existence of several systems mediating empathy. De Waal (2008) suggests that the phylogenetically earliest system is the emotional contagion system, in which one is affected by another's emotional or arousal state. On the other hand, the cognitive empathic perspective-taking system is a more advanced system and involves higher cognitive functions including mental state attribution. Indeed, emotional contagion has been reported in rodents (Langford, Crager, Shehzad, Smith, Sotocinal, Levenstadt, et al., 2006), while only the closest living relatives of

humans, the chimpanzees, possess rudimentary traits of cognitive aspects of empathy such as theory of mind (Call & Tomasello, 2008).

Indeed, empathy is a broad concept that refers to the cognitive, as well as the emotional reactions of one individual to the observed experiences of another. Neuroimaging, lesion and behavioral studies with humans and animals have been increasingly capable of characterizing the neural basis of empathy, thus providing new insights into the question of how we understand others' minds. Recent evidence supports a model of two separate brain systems for empathy: an emotional system and a cognitive system. The capacity to experience affective reactions to the observed experiences of others or share a "fellow feeling" has been described as "**emotional empathy**". Emotional empathy may involve several related underlying processes, including, among others, emotional contagion, emotion recognition, and shared pain. This might be where a kind of "simulation theory" takes. (p.180) On the other hand, the term "**cognitive empathy**" describes empathy as a cognitive "role-taking" ability, or the capacity to engage in the cognitive process of adopting another's psychological point of view (Frith & Singer 2008). This ability may involve a "theory-theory" kind of reasoning, i.e. making inference regarding the other's affective and cognitive mental states (Shamay-Tsoory, Aharon-Peretz & Perry, 2009).

This suggests that, while the two systems may work together, they may be behaviorally, developmentally and neuroanatomically dissociable. Here, we will focus on these two systems from a neuropsychological point of view—reviewing the current research from imaging, lesion studies, and different pathologies related to empathic abilities.

Emotional empathy

According to Preston & de Waal's (2002) perception-action hypothesis, perception of a behavior in another automatically activates one's own representations for the behavior, and output from this shared representation automatically proceeds to motor areas of the brain where responses are prepared and executed. This state-matching reaction has been related to the simulation theory. The most prominent evidence for the biological feasibility of the simulation theory is the seminal discovery of mirror neurons in the macaque monkey, a particular class of visuo-motor neurons that discharge both when the monkey does a particular goal-directed action and when it observes another individual (monkey or human) doing a similar action (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992; Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Rizzolatti, Fadiga, Gallese, & Fogassi, 1996). Such neurons have been discovered primarily in areas F5 of the monkey's pre-motor cortex (roughly corresponding in humans to Brodmann (BA) areas 44 and parts of BA 45), in the rostral part of the inferior parietal lobule (IPL, particularly in area PFG) and in the anterior intraparietal sulci (AIP) (Fogassi, Ferrari, Gesierich, Rozzi, Chersi, & Rizzolatti, 2005; Rozzi, Ferrari, Bonini, Rizzolatti, & Fogassi, 2008). Given its

observation-execution properties, it was suggested that the mirror neuron system (MNS) is particularly well suited to provide the appropriate mechanism for imitation, emotional contagion, and by extension, enabling empathy.

Neuroimaging studies in humans found different brain regions that similarly to the MNS in the monkey, are activated on the one hand by motor performance and, on the other hand, by seeing similar movements made by others. Such activity was found primarily in the rostral part of the IPL, the lower part of the precentral gyrus and the posterior part of the inferior frontal gyrus (IFG) (Buccino, Binkofski, Fink, Fadiga, Fogassi, Gallese, et al., 2001; Decety, Chaminade, Grezes, & Meltzoff, 2002; Grafton, Arbib, Fadiga, & Rizzolatti, 1996; Grezes, Armony, Rowe, & Passingham, 2003; Grezes, Costes, & Decety, 1998; Iacoboni et al., 1999; Rizzolatti et al., 1996). There is consistent and strong evidence for the involvement of the IFG in emotional contagion and emotion recognition. Chakrabarti, Bullmore, & Baron-Cohen (2006) found a positive correlation between a validated measure of empathy (the empathy quotient, EQ) and IFG activation, when viewing video clips depicting basic emotions. Indeed, it has been suggested that overt facial mimicry (as measured by an electro-myograph or through observation) is related to emotional contagion and emotion understanding (Niedenthal, 2007). The existence of mirror neurons related to emotional facial expressions in the human IFG suggests that the human MNS may be used to convert observed facial expressions into a pattern of neural activity that would be suitable for producing similar facial expressions and provide the neural basis for emotional contagion (Keysers & Gazzola, 2009). Jabbi and colleagues (Jabbi, Swart, & Keysers, 2007) have reported that observing positive and disgust facial expressions activated parts of the IFG and that participants' empathy scores were predictive of their IFG activation while witnessing facial expressions. Additionally, two **(p.181)** neuroimaging studies, one which involved emotion recognition (Schulte-Ruther, Markowitsch, Fink, & Piefke, 2007) and one that involved empathizing with people suffering serious threat or harm (Nummenmaa, Hirvonen, Parkkola, & Hietanen, 2008) have further emphasized the specific role of the IFG in emotional empathy. Cortical lesions involving the IFG, particularly in BA 44, are associated with impaired emotional contagion and deficits in emotion recognition, suggesting that the IFG not only participates in tasks that involve emotional empathy, but is also **necessary** for emotional empathy. Shamay-Tsoory et al. (2009) studied eight patients with IFG damage, and compared their results with those of ventro-medial (VM) lesion patients, posterior lesion patients and healthy controls. The IFG patients differed from the other groups in the emotional empathy subscales of the Interpersonal Reactivity Index (IRI, Davis, 1983), an empathy questionnaire that differentiates between cognitive and emotional components of empathy. In addition, these patients were significantly worse in an emotional recognition task, but did not differ from healthy individuals in the ToM tasks. The authors show a double dissociation between IFG patients and VM patients,

who showed significantly different results in the cognitive subscale and ToM task (Shamay-Tsoory et al., 2009).

A different neural manifestation that has been tentatively associated with the MNS in humans is a modulation of electroencephalographic (EEG) oscillations in the 8–13 frequency range, coined mu rhythms (Pineda, 2005). Mu rhythms are desynchronized and their power attenuated when engaging in motor activity (Gastaut, 1952) and, crucially, also while observing actions executed by someone else (Cochin, Barthelemy, Lejeune, Roux, & Martineau, 1998; Cochin, Barthelemy, Roux, & Martineau, 1999; Cohen-Seat, Gastaut, Faure, & Heuyer, 1954; Gastaut & Bert, 1954; Muthukumaraswamy, Johnson, & McNair, 2004). The visual-motor coupling suggested by this pattern led several investigators to suggest that it reflects a “resonance system”, simulating others’ motor actions (see Pineda, 2005, for a review). In the last few years, several studies linked mu suppression to higher social behaviors, such as understanding others social interactions (Oberman, Pineda, & Ramachandran, 2007), intentions (Perry, Troje, & Bentin, 2010) and empathy (Cheng, Lee, Yang, Lin, Hung, & Decety, 2008a; Cheng, Yang, Lin, Lee, & Decety, 2008b, Perry et al., 2010).

However, not only motor regions exhibit mirror-like mechanisms. **Tactile processing** mechanisms have also been shown to have mirror properties. The second and third somatosensory cortices can be vicariously recruited by the sight of other people being touched, performing actions or experiencing somatic pain (see Keysers, Kaas, & Gazzola, 2010, for a review). **Smelling** a foul odor engages the same sub-regions of the anterior insula as does watching another person express smell-induced disgust (Wicker, Keysers, Plailly, Royet, Gallese, & Rizzolatti, 2003). **Shared pain** appears to involve regions related to the first hand experience of pain, such as parts of the pain matrix. Specifically, a network including the anterior cingulate cortex (ACC) and the insula was reported to respond to both felt and observed pain (Decety, Yang, & Cheng, 2010). Activation in the ACC and insula has been also found to correlate with the participant’s judgments of the subjective severity of pain experienced by others on the basis of the other’s facial pain expression (Saarela, Markowitsch, Fink, & Piefke, 2007). This indicates that empathizing with people in pain is associated with hemodynamic activity in the brain that is similar to the activity that occurs when people feel pain themselves.

Finally, emotional empathy is also crucially dependent to the limbic system, and particularly on the amygdala. This brain structure, which is constructed of a complex collection of nuclei, is known for its substantial role in both experiencing and recognizing emotions, especially fear, and in social behavior and reward learning in general (see Adolphs, 2010, for a review). Adolphs and colleagues (Adolphs, Tranel, Hamann, Young, Calder, Phelps, et al., 1999)

examined a cohort of nine individuals with rare bilateral amygdala damage. Compared with controls, the subjects as a **(p.182)**

group were significantly impaired in recognizing fear, although individual performances ranged from severely impaired to essentially normal. An extensive line of research has been done on a specific patient, SM, with bilateral amygdala damage, who does not seem to recognize fear in others or to experience the feeling of fear or danger in herself (Adolphs, Tranel, Damasio, & Damasio, 1994). Impaired emotion recognition has also been linked to abnormal amygdala activation in psychiatric illnesses in which the behavioral relevance of social stimuli is abnormally evaluated, ranging from phobias (e.g. Larson, Schaefer, Siegle, Jackson, Anderle, & Davidson, 2006) to depression (e.g. Pezawas, 2005) schizophrenia (e.g. Das, Kemp, Flynn, Harris, Liddell, Whitford et al., 2007; Gur Calkins, Gur, Horan, Nuechterlein, & Seidman, 2007) and autism (e.g. Spezio, Adolphs, Hurley, & Piven, 2007). Interestingly, a similar impairment has also been found in first-degree relatives of people with autism and may constitute part of an “endophenotype” for impaired amygdala function in autism (Dalton Nacewicz, Alexander, & Davidson, 2007; Adolphs, Spezio, Parlier, & Piven, 2008).

To conclude, the core process enabling emotional empathy appears to be the generation of corresponding (to the target) emotional response (e.g. through the insula in shared pain, the amygdala in fear), and the corresponding motor representation related to the emotion (e.g. IFG, mu rhythms). The neural networks that participate in this system are detailed in Figure 11.1. Although this system appears to be bottom-up, it seems that top-down processes can modulate this automatic system and perhaps aspects of higher order cognitive process, as well as cognitive empathy, interact with emotional empathy in such

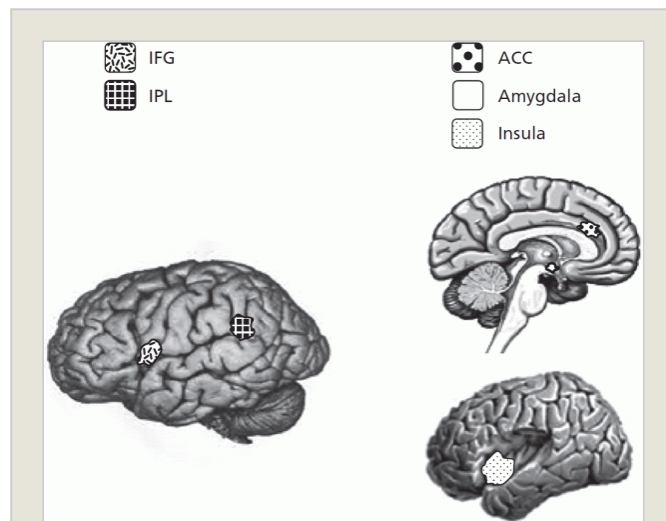


Figure 11.1 Brain regions associated with emotional empathy. See also Plate 3.

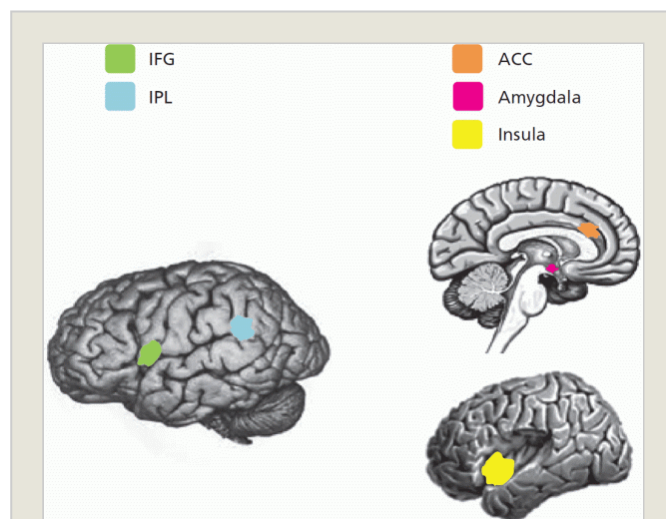


Plate 3 Brain regions associated with emotional empathy. See also Fig. 11.1.

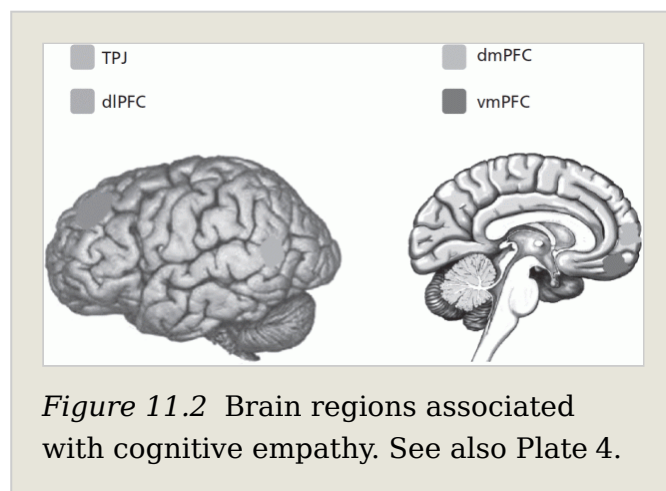
cases (e.g. Lamm, Meltzoff, & Decety, 2010; Perry, Bentin, Ben-Ami Bartal, Lamm, & Decety, 2010).

Cognitive empathy

Some complex forms of empathy involve the ability to create a theory about the other's mental state and cognitively take the perspective of others. This process of understanding another person's perspective, termed "cognitive empathy" is very much related to having ToM abilities. While one **(p.183)** dimension of ToM relates to others' beliefs and desires, another dimension concerns the emotional and social meaning of others' intentions (Brothers & Ring, 1992). Brothers & Ring (1992) referred to these dimensions as "cold" and "hot" aspects of ToM, and suggested that both forms of cognition contribute to understanding others' actions. The distinction between "cold" aspects of mental representations (cognitive ToM) as opposed to "hot" aspects of mental representations (affective ToM), has been further extended and examined in lesion studies (e.g. Shamay-Tsoory Tomer, Berger, Goldsher, & Aharon-Peretz, 2005; Shamay-Tsoory & Aharon-Peretz, 2007) and functional imaging studies (Hynes, Baird, & Grafton, 2006; Völlm, Taylor, Richardson, Corcoran, Stirling, McKie, S., et al., 2006). These have differentiated between cognitive and affective ToM, referring to reasoning about beliefs vs. reasoning about emotions, respectively. If we take the example of "false belief," cognitive false belief requires understanding what someone thinks about what someone else thinks (belief about belief), while affective false belief refers to understanding what someone thinks about what someone else feels (belief about emotions). Consistent with the possibility that ToM skills comprise several distinct processes that meet different cognitive demands, recent studies have identified a set of brain regions involved in ToM: the medial prefrontal cortex (mPFC), the superior temporal sulcus (STS), the temporoparietal junction (TPJ) and the temporal poles (TP) (Frith & Singer, 2008; Van Overwalle & Baetens, 2009). A recent review of imaging studies of ToM, (Carrington & Bailey, 2009) found that 93% of the 40 studies reviewed report activation in the mPFC. The TPJ region was active in 58% of the studies reviewed and the STS (including the IPL) in 50% of the studies. Based on a separate meta-analysis, Van Overwalle & Baetens (2009) proposed that the TPJ is mainly responsible for transient mental inferences about other people (e.g. their goals, desires, and beliefs), while the mPFC subserves the attribution of more enduring traits and qualities about the self and other people. The ventromedial prefrontal cortex (vmPFC) role in self-reflection has been lately shown in an fMRI study by Mitchell and colleagues (Mitchell, 2009), placing it as a key region necessary for evaluating the similarities and differences distinguishing the mental states of oneself from others. It is possible that situations that involve affective ToM entail more self-reflection as compared with situations involving cognitive ToM, which are more detached. Therefore the vmPFC, which is highly connected to the amygdala, appears to be particularly necessary for affective mentalizing, as opposed to neutral or cognitive forms of mentalizing, along with

mentalizing about others who are different from oneself, which have been linked to the modulation of dorsal regions of the mPFC (dmPFC; e.g. Mitchell, Macrae and Banaji, 2006). In addition, Kalbe and colleagues (Kalbe, Schlegel, Sack, Nowak, Dafotakis, Bangard, et al., 2010) have recently reported impaired cognitive ToM, following 1Hz repetitive Transcranial Magnetic Stimulation (TMS) which interfered with cortical activity of the dorsolateral PFC (dlPFC).

The frontal lobes, especially regions of the PFC, have been associated with executive aspects of cognition, social and moral behavior ever since description of frontal lobe syndromes-related changes in personality, social behavior, and emotional regulation emerged in the nineteenth century (Eslinger, Flaherty-Craig, & Benton, 2004). There is ample evidence that the orbitofrontal cortex (OFC) mediates affective information, emotional stimuli, and social behavior. Lesions in the OFC result, among other things, in impaired empathy (Eslinger, 1998; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004) and deficits in complex ToM abilities (Stone Baron-Cohen, & Knight, 1998). Shamay-Tsoory, Tibi-Elhanani, & Aharon-Peretz (2006) compared the performance of patients with lesions localized either in the vmPFC (part of the OFC), dorsolateral, TPJ, or superior parietal to healthy controls, with a battery of naturalistic affective and cognitive ToM stories (about false beliefs, false attribution, irony and lies). Compared with controls, patients with VM damage were impaired at providing appropriate mental state explanations for the affective ToM stories (see also Shamay-Tsoory et al., 2009). In addition, these patients' **(p.184)**



performance in the affective ToM was significantly impaired as compared with their performance in cognitive ToM stories. Furthermore, ratings of levels of emotionality of each story suggested that levels of affective load correlated with number of errors in the stories in the VM group, indicating that the more the emotional load involved in the story the greater the difficulty posed for the subjects in this group (see Figure 11.2).

To conclude, it appears that cognitive empathy involves higher order cognitive functions that involve self-other differentiation, and cognitive and affective ToM. Self-other distinction and affective ToM involve a network in which the OFC (specifically vmPFC), and the TPJ to some extent, are core regions. The dlPFC and dmPFC also seem to be involved in cognitive ToM, although more studies are needed to further dissociate brain regions necessary for each sub-component of ToM.

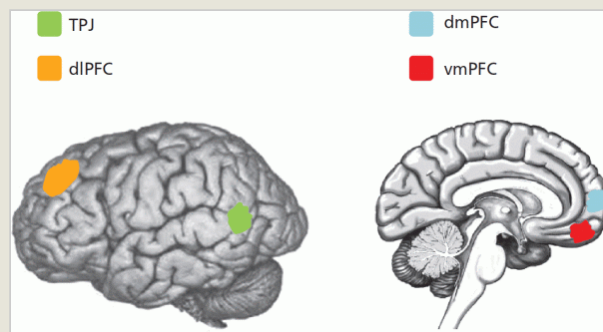


Plate 4 Brain regions associated with cognitive empathy. See also Fig. 11.2.

Neuropsychiatric deficits in empathy

There has been a marked increase in empathy and ToM studies in psychiatric populations that examine the pattern of brain activity in response to social perception tasks among patients. The failure of most studies to find clear activation or deactivation in specific brain regions, indicates the difficulties in recruiting a homogenous patient group, adjusting the behavioral measures and finding the appropriate control groups. Nonetheless, studying empathy in psychiatric populations may have important contributions to both basic research and to diagnosis and therapy. Some of these contributions are discussed below.

Autism

Autistic spectrum disorders (ASD) include a wide range of deficits in social cognitive skills, including deficits in ToM abilities, i.e. in understanding thoughts and intentions of others, and differentiating between one's own knowledge and the knowledge of the other (e.g. Baron Cohen, 2000). In an fMRI study, Lombardo and colleagues (Lombardo, Chakrabarti, Bullmore, Sadek, Pasco, Wheelwright, et al., 2010) have demonstrated that while typical individuals recruit the ACC and the vmPFC in response to self and other-referential processing respectively, in autism, vmPFC responded equally to self and other. Furthermore, the magnitude of neural self-other distinction in ventromedial prefrontal cortex was strongly related to the magnitude of early childhood social impairments in autism. Individuals whose vmPFC made little to no distinction between **(p.185)** mentalizing about self and other were the most socially impaired in early childhood. In a different fMRI study (Schulte-Ruther, et al.,

2011), individuals with autism and healthy control subjects were asked to identify the emotional state observed in a facial stimulus (other-task) or to evaluate their own emotional response (self-task). Activations in these tasks were found in vmPFC only in control subjects, while more dorsal activation was found in ASD subjects. During the self-task, ASD subjects activated an additional network of frontal and inferior temporal areas. The authors concluded that subjects with ASD may use an atypical cognitive strategy to gain access to their own emotional state in response to other people's emotions. The differences between the results of these two studies may be due to the different tasks used, or to a different subgroup of ASD individuals. However, both studies strengthen the notion that this complex disorder is correlated with abnormalities in brain regions that enable our understanding of others, and the differentiation between self and others.

Mirror neuron system dysfunction and autism

To examine MNS abnormalities in individuals with ASD, Dapretto and colleagues studied high functioning children with ASD and matched controls, in an fMRI experiment involving imitation and observation of emotional expressions. Although there were no behavioral differences between the groups, children with ASD showed no mirror activity in the IFG. Moreover, activity in this area was inversely related to symptom severity in the social domain (Dapretto, Davies, Pfeifer, Scott, Sigman, Bookheimer, et al., 2006; see also Uddin, Davies, Scott, Zaidel, Bookheimer, Iacoboni, et al., 2008; Schulte-Ruther, Greimel, Markowitsch, Kamp-Becker, Remschmidt, Fink, et al., 2010 for similar results).

In line with this, studies of EEG mu suppression in individuals with ASD show normal mu suppression while self-performing hand movements, but no suppression when passively viewing someone else performing the same movements (Martineau, Schmitz, Assaiante, Blanc, & Barthelemy, 2004; Oberman, Hubbard, McCleery, Altschuler, Ramachandran, & Pineda 2005; Oberman, Ramachandran, & Pineda, 2008; but see Raymaekers, Wiersema, & Roeyers, 2009). Oberman and colleagues (2008) found a positive correlation between the amount of mu suppression and the putative ability of the observer to identify with the agent moving on the screen: Both ASD and typically developed individuals showed greater suppression to familiar hands (of family members) compared with those of strangers. These studies strengthen the notion that ASD patients may have dysfunction in mirror neuron systems, however, further studies are needed to validate this claim and especially to understand what is the cause (MNS dysfunction, Autism) and what is the effect.

Schizophrenia

Schizophrenia constitutes a complex mental disorder, which includes what is typically referred to as "positive symptoms" (delusions, hallucinations, disorganized speech, disorganized or catatonic behavior), and/or "negative symptoms" (affective flattening, alogia, or anhedonia). Patients suffering from

schizophrenia show impaired emotional and social behavior, such as misinterpretation of social situations and lack of ToM skills. Differentiating between cognitive and affective ToM, Shamay-Tsoory and colleagues (2007) showed that patients with schizophrenia made significantly more errors in the affective conditions, compared with controls. Furthermore, correlation analysis indicated that impaired affective ToM in these patients correlated with their level of negative symptoms. These results indicate that individuals with high level of negative symptoms of schizophrenia may demonstrate selective impairment in their ability to attribute affective mental states. **(p.186)**

Following this study, the performance of 24 patients with schizophrenia was compared with the responses of patients with localized lesions in the VM or dorsolateral PFC, patients with non-frontal lesions, and healthy control subjects. Patients with schizophrenia and those with VM lesions made similar errors on “affective ToM” tasks showed normal results in cognitive ToM conditions. The authors concluded that the pattern of mentalizing impairments in schizophrenia resembled those seen in patients with lesions of the frontal lobe, particularly with VM damage, providing support for the notion of a disturbance of the fronto-limbic circuits in schizophrenia (Shamay-Tsoory, Aharon-Peretz, & Levkovitz, 2007). Strengthening this notion, an fMRI study using a modified emotional-Stroop task, showed that patients with schizophrenia were significantly less efficient than the healthy controls during the emotional incongruent Stroop trials, and when emotionally incongruent trials were compared with congruent trials, relative deactivations of the subgenual cingulate gyrus and the vmPFC observed in the healthy controls were not found in the patient group.

Importantly, activities of these regions inversely correlated with emotional interference to performance efficiency and response accuracy respectively in the patient group (Park, Park, Chun, Kim, & Kim, 2008). Recently, an important study showed that ToM skills are related to gray matter volume (GMV) in the vmPFC in Schizophrenia (Hooker, Bruce, Lincoln, Fisher, & Vinogradov, 2011). The authors used voxel-based morphometry and a multi-method behavioral assessment of ToM processing, including performance-based, self-report, and interview-rated ToM assessments, to investigate whether ToM skills were related to vmPFC GMV. As expected, compared with healthy participants, schizophrenia participants had worse ToM performance and lower self-reported ToM processing in daily life. Importantly, schizophrenia participants had less vmPFC GMV than healthy participants. Moreover, among schizophrenia participants, all three measures of ToM processing were associated with vmPFC GMV, such that worse ToM skills were related to less VMPFC GMV. This association remained strong for self-reported and interview-rated ToM skills, even when controlling for the influence of global cognition. Together, these studies suggest a strong correlation between abnormalities in vmPFC, and cognitive empathy skills in schizophrenia patients.

MNS dysfunction and schizophrenia

The discovery of the MNS had led to much excitement, and was seen as an opportunity to further our understanding of different disorders of social cognition and empathy. It has been suggested in the last few years that both the positive and the negative symptoms of schizophrenia may be due to a dysfunctional MNS (e.g. Arbib & Mundhenk, 2005; Buccino & Amore, 2008; Burns, 2006), although only little support has been found for these hypotheses (Enticott, Hoy, Herring, Johnston, Daskalakis, & Fitzgerald, 2008; Singh, Pineda, & Cadenhead, 2011) and more research is needed in order to understand the relationship between these brain networks and the complex disorder of schizophrenia.

Psychopathy

Patients with OFC lesions described above have sometimes been described as expressing “acquired sociopathy” (Blair & Cipolotti, 2000; Tranel, Bechara, & Denburg, 2002), a term denoting aberrant behavior, high levels of aggression, and a callous disregard for others following OFC lesions. Similarly, criminal offenders with psychopathic tendencies show impaired emotional and social behavior, such as lack of emotional responsiveness to others and deficient empathy. To assess the emotional and cognitive aspects of ToM in people with psychopathic tendencies, Shamay-Tsoory, Harari, Aharon-Peretz, & Levkovitz (2010) used a task that examines affective vs. cognitive ToM processing in separate conditions. ToM abilities of criminal offender diagnosed with antisocial **(p.187)** personality disorder with high psychopathy features was compared with that of participants with localized lesions in the OFC or dorsolateral participants with non-frontal lesions, and healthy control subjects. Individuals with psychopathy and those with OFC lesions were impaired on the “affective ToM” conditions, but not in “cognitive ToM” conditions, compared with the control groups. This study suggests that mentalizing impairments in psychopathy resembles remarkably that seen in participants with lesions of the frontal lobe, particularly with OFC damage, and provides support for the notion of amygdala-OFC dysfunction in psychopathy (Blair, 2007; Finger, Marsh, Mitchell, Reid, Sims, Budhani, et al., 2008). However, it should be noted that regardless of the similarities between acquired frontal lesions and developmental psychopathy, comparison between these groups should be treated with caution as essential differences exist between these individuals. Importantly, while both groups may demonstrate reactive aggression, instrumental aggression is typically reported in developmental psychopathy, but rarely reported after OFC damage (Mitchell, Avny & Blair, 2006).

Frontotemporal dementia

Frontotemporal dementia (FTD) is a progressive neurodegenerative syndrome with diverse clinical presentations. Among the most prominent features are progressive aphasia and bizarre affect with a “personality change.” These may include disinhibition and impulsivity, distractibility and impersistence, and

perseverative behavior, in addition to social deficits such as lack of empathy, emotional unconcern, apathy, and irritability (for a review, see Grossman, 2002). Shany-Ur and colleagues (Shany-Ur, Poorzand, Grossman, Growdon, Jang, Ketelle, et al., 2012) investigated whether face-to-face testing of comprehending insincere communication would effectively discriminate among different neurodegenerative disease patients. The authors examined the ability to comprehend lies and sarcasm from a third-person perspective, using contextual cues, in 102 patients with either FTD, Alzheimer's disease, progressive supranuclear palsy (PSP) or vascular cognitive impairment, and 77 healthy older adults. Participants answered questions about videos depicting social interactions involving deceptive, sarcastic, or sincere speech using the Awareness of Social Inference Test, which assesses poor understanding of emotional expressions and difficulty integrating contextual information that is part of normal social encounters (McDonald, Flanagan, Rollins, 2002). All subjects equally understood sincere remarks, but FTD patients displayed impaired comprehension of lies and sarcasm compared with normal controls. In other groups, impairment was not disease-specific, but was proportionate to general cognitive impairment. Analysis of the task components revealed that only FTD patients were impaired on perspective taking and emotion reading elements and that both FTD patients and PSP patients had impaired ability to represent others opinions and intentions (i.e. ToM). Moreover, test performance correlated with informants' ratings of subjects' empathy, perspective taking and neuropsychiatric symptoms in everyday life. All patient groups exhibited some deficiencies in these complex social communication tasks, which require multiple cognitive and emotional processes. However, FTD patients showed uniquely focal and severe impairments at every level of ToM and emotion reading skills, showing an inability to identify even obvious examples of deception and sarcasm. These results suggest that FTD may target a specific neural network necessary for perceiving social salience and social outcomes (Shany-Ur et al., 2012).

Interactions between the two empathy systems

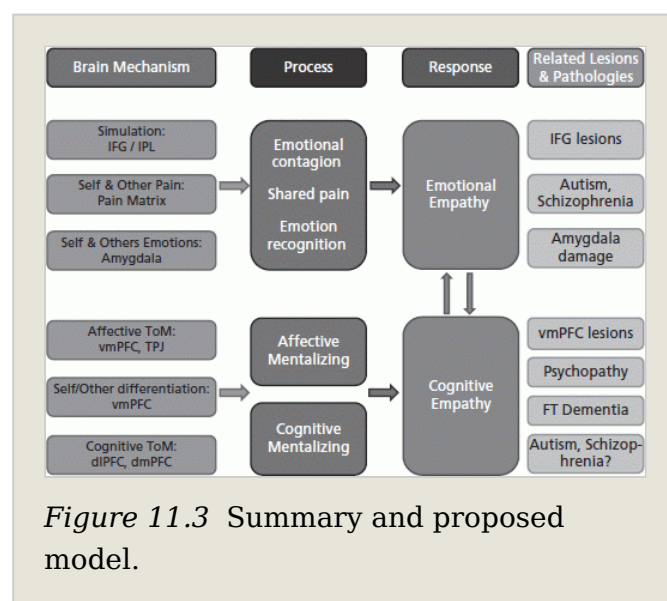
The differentiation between these two systems enabling empathy suggests that under normal circumstances every interaction with another may trigger independently both an emotional response (**p.188**) (emotional empathy) as well as cognitive evaluation of his state of mind or perspective (cognitive empathy). The other's emotions are "shared" through brain areas involved in resonance or simulation, such as the human MNS. In addition, the ability to accurately infer the other's perspective and imagine their state of mind is activated, requiring self-other decoding and ToM abilities. Both functional neuroimaging and lesion studies in humans indicate that the vmPFC plays a crucial role in the network performing cognitive empathic function. This system is phylogenetically younger and is unique to primates and human adults.

Although both emotional and cognitive components of empathy may operate partly autonomously, it is likely that every empathic response will evoke both components to some extent, depending on the social context. Zaki and colleagues showed that empathically accurate, as compared with inaccurate, judgments depended both on the activation of structures within the human MNS, thought to be involved in emotional empathy, and on the activation of regions implicated in mental state attribution, or cognitive empathy, such as the medial prefrontal cortex. These data demonstrate that activity in these two sets of brain regions tracks with the accuracy of attributions made about another's internal emotional state (Zaki, Bolger, & Ochsner, 2009). Moreover, in a second study, the authors manipulated the degree to which one could use non-verbal and contextual cues in order to infer about the target's emotions. They found that conditions in which biasing of neural activity was seen toward the MNS, tracked with perceivers' behavioral reliance on non-verbal cues; In contrast, conditions in which biasing was seen toward what they called a "mental state attribution system", including the TPJ and mPFC, was tracked with perceivers' reliance on contextual cues when drawing inferences about targets' emotions. Future studies may further this focus on the interactions between the two systems and the different conditions that may affect the activation of each. Different variables such as the level of emotions involved, the past experiences of the empathizer, gender, relationship with the protagonist and the perceived similarity between

(p.189) the individual and the protagonist may differentially activate the emotional and the cognitive systems. Research of complex conditions, such as autism and schizophrenia, which are characterized by (among others) their deficits in social cognition and empathic abilities, may highly benefit from the progress in this field. Exploring these questions using a combination of several research tools such as lesion studies, neuroimaging, electrophysiology, genetics and animal research may prove to be essential in characterizing the relationship between these two systems and the conditions in which each system is activated. See Figure 11.3 for a proposed model.

References

Bibliography references:



Adolphs, R. (2010). What does the amygdala contribute to social cognition? *Annals of the New York Academy of Sciences* 1191: 42–61.

Adolphs, R., Spezio, M. L., Parlier, M., & Piven, J. (2008). Distinct face processing strategies in parents of autistic children. *Current Biology* 18: 1090–3.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature* 372: 669–72.

Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., Anderson, A., Lee, G. P., & Damasio, A. R. (1999). Recognition of facial emotion in nine subjects with bilateral amygdala damage. *Neuropsychologia* 37: 1111–17.

Arbib, M. A., & Mundhenk T. N. (2005). Schizophrenia and the mirror-neuron system: an essay. *Neuropsychologia* 43: 268–80.

Baron-Cohen, S. (2000). Theory of mind and autism: A fifteen year review. In: S. Baron-Cohen, H. Tager-Flusberg & D. J. Cohen (Eds), *Understanding Other Minds: Perspectives from Developmental Cognitive Neuroscience* (pp. 1–20). Oxford: Oxford University Press.

Blair R. J. (2007). Dysfunctions of medial and lateral orbitofrontal cortex in psychopathy. *Annals of the New York Academy of Sciences* 1121: 461–79.

Blair R. J., & Cipolotti L. (2000). Impaired social response reversal. A case of “acquired sociopathy”. *Brain* 123: 1122–41.

Brothers, L., & Ring, B. (1992). A neuroethological framework for the representation of minds. *Journal of Cognitive Neuroscience* 4: 107–18.

Buccino, G., & Amore M. (2008). Mirror neurons and the understanding of behavioural symptoms in psychiatric disorders. *Current Opinion in Psychiatry* 21: 281–5.

Buccino, G., Binkofski, F., Fink, G. R., Fadiga, L., Fogassi, L., Gallese, V., et al. (2001). Action observation activates premotor and parietal areas in a somatotopic manner: an fMRI study. *European Journal of Neuroscience* 13(2): 400–4.

Burns, J. (2006). The social brain hypothesis of schizophrenia. *World Psychiatry* 5: 77–81.

Call, J., & Tomasello, M. (2008). Does the chimpanzee have a theory of mind? 30 years later. *Trends in Cognitive Science* 12: 187–92.

- Carpenter, M., Nagell, K., & Tomasello, M. (1998). Social cognition, joint attention, and communicative competence from 9 to 15 months of age. *Monographs in Social Research in Child Development* 63: 176.
- Carrington, S. J., Bailey, A. J. (2009). Are there theory of mind regions in the brain? A review of the neuroimaging literature. *Human Brain Mapping* 30: 2313–35.
- Carruthers, P., & Smith, P. K. (Eds). (1996). *Theories of Theories of Mind*: Cambridge University Press.
- Chakrabarti, B., Bullmore, E. T., & Baron-Cohen, S. (2006). Empathizing with basic emotions: Common and discrete neural substrates. *Social Neuroscience* 1(3–4): 364–84.
- Cheng, Y., Lee, P. L., Yang, C. Y., Lin, C. P., Hung, D., & Decety, J. (2008a). Gender differences in the mu rhythm of the human mirror-neuron system. *PLoS ONE* 3(5): e2113.
- Cheng, Y., Yang, C-Y., Lin, C-P., Lee, P-L., & Decety, J. (2008b). The perception of pain in others suppresses somatosensory oscillations: A magnetoencephalography study. *NeuroImage* 40(4): 1833–40. **(p.190)**
- Cochin, S., Barthelemy, C., Roux, S., & Martineau, J. (1999). Observation and execution of movement: similarities demonstrated by quantified electroencephalography. *European Journal of Neuroscience* 11(5): 1839–42.
- Cochin, S., Barthelemy, C., Lejeune, B., Roux, S., & Martineau, J. (1998). Perception of motion and qEEG activity in human adults. *Electroencephalography and Clinical Neurophysiology* **107**(4): 287–95.
- Churchland, P. N. (1988). *Matter and Consciousness*. Cambridge: MIT Press.
- Davis, M. H. (1980). A multidimensional approach to individual differences in empathy. *JSAS Catalog of Selected Documents in Psychology* 10, 85.
- Dalton, K. M., Nacewicz, B. M., Alexander, A. L., & Davidson, R. J. (2007). Gaze-fixation, brain activation, and amygdala volume in unaffected siblings of individuals with autism. *Biological Psychiatry* 61: 512–20.
- Dapretto, M., Davies M. S., Pfeifer J. H., Scott A. A., Sigman M., Bookheimer S. Y., et al. (2006). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, 9, 28–30.
- Davis, M. H. (1983). Measuring individual differences in empathy: Evidence for a multidimensional approach. *Journal of Personality and Social Psychology* **44**(1): 113–26.

- Davies, M., & Stone, T. (Eds). (1995). *Mental Simulation*. Oxford: Basil Blackwell.
- Das, P., Kemp, A. H., Flynn, G., Harris, A. W., Liddell, B. J., Whitford, T. J., et al. (2007). Functional disconnections in the direct and indirect amygdala pathways for fear processing in schizophrenia. *Schizophrenia Research* 90: 284–94.
- de Waal, F. B. (2008). Putting the altruism back into altruism: the evolution of empathy. *Annual Review of Psychology* 59: 279–300.
- Decety, J., Chaminade, T., Grezes, J., & Meltzoff, A. N. (2002). A PET exploration of the neural mechanisms involved in reciprocal imitation. *NeuroImage* 15(1): 265–72.
- Decety J., Yang C. Y., & Cheng Y. (2010). Physicians down-regulate their pain empathy response: An event-related brain potential study. *NeuroImage* 50: 1676–82.
- di Pellegrino, G., Fadiga, L., Fogassi, L., Gallese, V., & Rizzolatti, G. (1992). Understanding motor events: a neurophysiological study. *Experimental Brain Research* 91(1): 176–80.
- Enticott P. G., Hoy K. E., Herring S. E., Johnston P. J., Daskalakis Z. J., & Fitzgerald P. B. (2008). Reduced motor facilitation during action observation in schizophrenia: A mirror neuron deficit? *Schizophrenia Research* 102: 116–21.
- Eslinger, P. J. (1998). Neurological and neuropsychological bases of empathy. *European Neurology* 39: 193–9.
- Eslinger, P. J., Flaherty-Craig, C. V., & Benton, A. L. (2004). Developmental outcomes after early prefrontal cortex damage. *Brain and Cognition* 55(1): 84–103.
- Finger E. C., Marsh A. A., Mitchell D. G. V., Reid M. E., Sims C., Budhani S., et al. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives of General Psychiatry* 65, 586–94.
- Flavell, J. H., Everett, B. A., Croft, K., & Flavell, E. R. (1981). Young children's knowledge about visual perception: Further evidence for the Level 1-Level 2 distinction. *Developmental Psychology* 17, 99–103.
- Flavell, J. H., Flavell, E. R., & Green, F. L. (1983). Development of the appearance—reality distinction. *Cognitive Psychology* 15(1): 95–120.
- Fodor, J. A. (1987). *Psychosemantics*. Cambridge: MIT Press.

- Fogassi, L., Ferrari, P. F., Gesierich, B., Rozzi, S., Chersi, F., & Rizzolatti, G. (2005). Parietal lobe: from action organization to intention understanding. *Science* 308(5722): 662–7.
- Frith, C. D., & Frith, U. (1999). Interacting minds—a biological basis. *Science* 286(5445): 1692–5.
- Frith C. D., & Singer T. 2008. The role of social cognition in decision making. *Philosophical Transactions of the Royal Society of London, B, Biological Sciences* 363: 3875–86.
- Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain* 119(Pt 2): 593–609. **(p.191)**
- Gallese, V., & Goldman, A. (1998). Mirror neurons and the simulation theory of mind-reading. *Trends in Cognitive Sciences* 2(12): 493–501.
- Gastaut, H. (1952). Etude électrocorticographique de la réactivité des rythmes rolandiques. *Revue Neurologique* 87(2): 176–82.
- Gastaut, H. J., & Bert, J. (1954). EEG changes during cinematographic presentation. *Electroencephalography and Clinical Neurophysiology* 6: 433–44.
- Goldman, A. (1989). Interpretation psychologized *Mind Language* 4, 161–85.
- Gopnik, A. (1993). How we know our minds: the illusion of first-person knowledge of intentionality. *Behaviour & Brain Sciences* 16: 1–14.
- Gordon, R. (1986). Folk psychology as simulation. *Mind Language* 1, 158–71.
- Grafton, S. T., Arbib, M. A., Fadiga, L., & Rizzolatti, G. (1996). Localization of grasp representations in humans by positron emission tomography.2. Observation compared with imagination. *Experimental Brain Research* 112(1): 103–11.
- Grezes, J., Armony, J. L., Rowe, J., & Passingham, R. E. (2003). Activations related to “mirror” and “canonical” neurones in the human brain: An fMRI study. *NeuroImage* 18(4): 928–37.
- Grezes, J., Costes, N., & Decety, J. (1998). Top down effect of strategy on the perception of human biological motion: a PET investigation. *Cognitive Neuropsychology*, **15**: 553–82.
- Grossman, M. (2002). Frontotemporal dementia: a review. *Journal of the International Neuropsychology Society* 8, 566–83.

Gur, R. E., Calkins, M. E., Gur, R. C., Horan, W. P., Nuechterlein, K. H., Seidman, L. J., Stone, W. S. (2007). The Consortium on the Genetics of Schizophrenia: neurocognitive endophenotypes. *Schizophrenia Bulletin* **33**(1), 49–68.

Gur, R. E., Loughhead, J., Kohler, C. G., Elliott, M., Lesko, K., Ruparel, K., Wolf, D. H., Bilker, W. B., & Gur, R. C. (2007). Limbic activation associated with misidentification of fearful faces and flat affect in schizophrenia. *Archives of General Psychiatry* **64**, 1356–66.

Harlow, J. M. (1868). Recovery from the passage of an iron bar through the head. *Publications of the Massachusetts Medical Society* **2**: 327–47.

Heal, J. (Ed.). (1986). *Replication and functionalism*. Cambridge: Cambridge University Press.

Hooker, H. I., Bruce, L., Lincoln, S. H., Fisher, M., & Vinogradov, S. (2011). Theory of mind skills are related to gray matter volume in the ventromedial prefrontal cortex in schizophrenia. *Biological Psychiatry* **70**(12): 1169–78.

Hynes, C. A., Baird, A. A., & Grafton, S. T. (2006). Differential role of the orbital frontal lobe in emotional vs. cognitive perspective-taking. *Neuropsychologia* **44**, 374–83.

Iacoboni, M., Woods, R. P., Brass, M., Bekkering, H., Mazziotta, J. C., & Rizzolatti, G. (1999). Cortical mechanisms of human imitation. *Science* **286**(5449): 2526–8.

Jabbi, M., Swart, M., Keysers, C. (2007). Empathy for positive and negative emotions in the gustatory cortex. *NeuroImage* **34**: 1744–53.

Johnson, S. (2003). Detecting agents. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences* **358**(1431): 549.

Kalbe, E., Schlegel, M., Sack, A. T., Nowak, D. A., Dafotakis, M., Bangard, C., Brand, M., Shamay-Tsoory, S., Onur, O. A., & Kessler, J. (2010). Dissociating cognitive from affective theory of mind: a TMS study. *Cortex* **46**, 769–80.

Keysers, C., & Gazzola, V. (2007). Integrating simulation and theory of mind: from self to social cognition. *Trends in Cognitive Sciences* **11**(5): 194–6.

Keysers, C., Kaas, J. H., & Gazzola, V. (2010). Somatosensation in social perception. *National Review of Neuroscience* **11**: 417–28.

Kosslyn, S. (1978). Measuring the visual angle of the mind's eye. *Cognitive Psychology* **10**, 356–89.

Lamm, C., Meltzoff, A. N., & Decety, J. (2010). How do we empathize with someone who is not like us? A functional magnetic resonance imaging study. *Journal of Cognitive Neuroscience* 22, 362–76. **(p.192)**

Langford D. J., Crager S. E., Shehzad Z., Smith S. B., Sotocinal S. G., Levenstadt J. S. Chanda M. L., Levitin D. J., & Mogil J. S. (2006). Social modulation of pain as evidence for empathy in mice. *Science* 312, 1967–70.

Larson, C. L., Schaefer, H. S., Siegle, G. J., Jackson, C. A., Anderle, M. J., & Davidson, R. J. (2006). Fear is fast in phobic individuals: amygdala activation in response to fear-relevant stimuli. *Biological Psychiatry* 60, 410–17.

Lewis, D. (1972). Psychophysical and theoretical identifications. *Australasian Journal of Philosophy* 50, 249–58.

Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., Sadek, S. A., Pasco, G., Wheelwright, S. J., Suckling, J., & Baron-Cohen, S. (2010). Atypical neural self-representation in autism. *Brain* 133, 611–24.

Martineau, J., Schmitz, C., Assaiante, C., Blanc, R., & Barthelemy, C. (2004). Impairment of a cortical event-related desynchronization during a bimanual load-lifting task in children with autistic disorder. *Neuroscience Letters* 367(3): 298–303.

McDonald, S., Flanagan, S., & Rollins, J. (2002). *The Awareness of Social Inference*. TestSuffolk: Thames Valley Test Co., Ltd.

Mitchell, J. P. (2009). Inferences about mental states. *Philosophical Transactions of the Royal Society London, B, Biological Sciences* 364, 1309–16.

Mitchell, J. P., Macrae, C. N., & Banaji, M. R. (2006) Dissociable medial prefrontal contributions to judgments of similar and dissimilar others. *Neuron* 50(4): 655–63.

Mitchell, D., Avny, S., & Blair, R. J. (2006). Divergent patterns of aggressive and neurocognitive characteristics in acquired versus developmental psychopathy. *Neurocase* 12(3): 164–78

Muthukumaraswamy, S. D., Johnson, B. W., & McNair, N. A. (2004). Mu rhythm modulation during observation of an object-directed grasp. *Brain Research in Cognitive Brain Research* 19(2): 195–201.

Niedenthal, P. (2007). Embodying emotion. *Science* 316(5827): 1002.

Nummenmaa, L., Hirvonen, J., Parkkola, R., & Hietanen, J. K. (2008). Is emotional contagion special? An fMRI study on neural systems for affective and cognitive empathy. *NeuroImage* 43, 571–80.

- Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. A. (2005). EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Brain Research: Cognitive Brain Research* 24(2): 190-8.
- Oberman, L. M., Pineda, J. A., & Ramachandran, V. S. (2007). The human mirror neuron system: A link between action observation and social skills. *Social Cognitive Affective Neuroscience* 2(1): 62-6.
- Oberman, L. M., Ramachandran, V. S., & Pineda, J. A. (2008). Modulation of mu suppression in children with autism spectrum disorders in response to familiar or unfamiliar stimuli: The mirror neuron hypothesis. *Neuropsychologia* 46(5): 1558-65.
- Park, I. H., Park, H. J., Chun, J. W., Kim, E. Y., & Kim, J. J. (2008). Dysfunctional modulation of emotional interference in the medial prefrontal cortex in patients with schizophrenia. *Neuroscience Letters* 440, 119-24.
- Perry, A., Bentin, S., Ben-Ami Bartal, I., Lamm, C., & Decety, J. (2010). "Feeling" the pain of those who are different from us—modulation of EEG in the mu/alpha range. *Cognitive, Affective and Behavioral Neuroscience* 10, 493-504.
- Perry, A., Troje, N. F., & Bentin, S. (2010). Exploring motor system contributions to the perception of social information: Evidence from EEG activity in the mu/alpha frequency range. *Social Neuroscience* 5(3): 272-84.
- Pezawas, L., Meyer-Lindenberg, A., Drabant, E. M., Verchinski, B. A., Munoz, K. E., Kolachana, B. S., Egan, M. F., Mattay, V. S., Hariri, A. R., & Weinberger, D. R. (2005). 5-HTTLPR polymorphism impacts human cingulate-amygdala interactions: a genetic susceptibility mechanism for depression. *National Neuroscience* 8: 828-34.
- Pineda, J. A. (2005). The functional significance of mu rhythms: translating "seeing" and "hearing" into "doing". *Brain Research and Brain Research Reviews* 50(1): 57-68. **(p.193)**
- Premack, D., & Woodruff, G. (1978). Chimpanzee problem-solving: a test for comprehension. *Science* 202(4367): 532-5.
- Preston, S. D., & de Waal, F. B. M. (2002). Empathy: Its ultimate and proximate bases. *Behavioral and Brain Sciences* 25(1): 1-20.
- Raymaekers, R., Wiersema, J., & Roeyers, H. (2009). EEG study of the mirror neuron system in children with high functioning autism. *Brain Research* 1304: 113-21.

Rizzolatti, G., Fadiga, L., Gallese, V., & Fogassi, L. (1996). Premotor cortex and the recognition of motor actions. *Brain Research and Cognitive Brain Research* 3(2): 131–41.

Rozzi, S., Ferrari, P. F., Bonini, L., Rizzolatti, G., & Fogassi, L. (2008). Functional organization of inferior parietal lobule convexity in the macaque monkey: electrophysiological characterization of motor, sensory and mirror responses and their correlation with cytoarchitectonic areas. *European Journal of Neuroscience* 28(8): 1569–88.

Saarela, M. V., Hlushchuk, Y., Williams, A. C., Schurmann, M., Kalso, E., & Hari, R. (2007). The compassionate brain: humans detect intensity of pain from another's face. *Cerebral Cortex* 17: 230–7.

Schulte-Ruther, M., Markowitsch, H. J., Fink, G. R., & Piefke, M. (2007). Mirror neuron and theory of mind mechanisms involved in face-to-face interactions: a functional magnetic resonance imaging approach to empathy. *Journal of Cognitive Neuroscience* 19, 1354–72.

Schulte-Ruther, M., Greimel, E., Markowitsch, H. J., Kamp-Becker, I., Remschmidt, H., Fink, G. R., & Piefke, M. (2010). Dysfunctions in brain networks supporting empathy: An fMRI study in adults with autism spectrum disorders. *Social Neuroscience* 6: 1–21.

Shamay-Tsoory, S. G., & Aharon-Peretz, J. (2007). Dissociable prefrontal networks for cognitive and affective theory of mind: a lesion study. *Neuropsychologia* 45: 3054–67.

Shamay-Tsoory, S. G., Aharon-Peretz, J., & Levkovitz, Y. (2007). The neuroanatomical basis of affective mentalizing in schizophrenia: comparison of patients with schizophrenia and patients with localized prefrontal lesions. *Schizophrenia Research* 90, 274–83.

Shamay-Tsoory, S. G., Aharon-Peretz, J., & Perry, D. (2009). Two systems for empathy: a double dissociation between emotional and cognitive empathy in inferior frontal gyrus vs. ventromedial prefrontal lesions. *Brain* 132, 617–27.

Shamay-Tsoory, S. G., Harari, H., Aharon-Peretz, J., & Levkovitz, Y. (2010). The role of the orbitofrontal cortex in affective theory of mind deficits in criminal offenders with psychopathic tendencies. *Cortex* 46(5): 668–77.

Shamay-Tsoory, S. G., Shur, S., Barcai-Goodman, L., Medlovich, S., Harari, H., & Levkovitz, Y. (2007). Dissociation of cognitive from affective components of theory of mind in schizophrenia. *Psychiatry Research* 149(1–3): 11–23.

- Shamay-Tsoory, S. G., Tibi-Elhanani, Y. & Aharon-Peretz, J. (2006). The ventromedial prefrontal cortex is involved in understanding affective, but not cognitive theory of mind stories. *Social Neuroscience* 1(3-4): 149-66.
- Shamay-Tsoory, S. G., Tomer, R., Berger, B. D., Goldsher, D., & Aharon-Peretz, J. (2005). Impaired “affective theory of mind” is associated with right ventromedial prefrontal damage. *Cognitive and behavioral neurology* 18(1): 55-67.
- Shamay-Tsoory, S. G., Tomer, R., Goldsher, D., Berger, B. D., & Aharon-Peretz, J. (2004). Impairment in cognitive and affective empathy in patients with brain lesions: anatomical and cognitive correlates. *Journal of Clinical Experimental Neuropsychology* 8, 1113-27.
- Shany-Ur, T., Poorzand, P., Grossman, S., Growdon, M. E., Jang, J. Y., Ketelle, R. S., Miller, B. L., & Rankin, K. P. (2012). Comprehension of insincere communication in neurodegenerative disease: Lies, sarcasm, and theory of mind. *Cortex* **48**(10): 1329-41.
- Simner, M. L. (1971). Newborn’s response to the cry of another infant. *Developmental Psychology* 5: 136-50.
- Singh, F., Pineda, J., & Cadenhead, K. S. (2011). Association of impaired EEG mu wave suppression, negative symptoms and social functioning in biological motion processing in first episode of psychosis. *Schizophrenia Research* 130, 182-6. **(p.194)**
- Spezio, M. L., Adolphs, R., Hurley, R. S., & Piven, J. (2007). Abnormal use of facial information in high-functioning autism. *Journal of Autism and Development Disorders* 37: 929-39.
- Stich, S. A. N., & Nichols, S. (1992). Folk psychology: simulation vs. tacit theory. *Mind and Language* 7: 29-65.
- Stone, V. E., Baron-Cohen, S., & Knight, R. T. (1998). Frontal lobe contributions to theory of mind. *Journal of Cognitive Neuroscience* 10(5): 640-56.
- Tranel, D., Bechara, A., & Denburg, N. L. (2002). Asymmetric functional roles of right and left ventromedial prefrontal cortices in social conduct, decision-making, and emotional processing. *Cortex* 38: 589-612.
- Uddin, L. Q., Davies, M. S., Scott, A. A., Zaidel, E., Bookheimer, S. Y., Iacoboni, M., et al. (2008). Neural basis of self and other representation in autism: An FMRI study of self-face recognition. *PLoS ONE* 3: 3526.
- Van Overwalle F., & Baetens K. (2009). Understanding others’ actions and goals by mirror and mentalizing systems: a meta-analysis. *NeuroImage* 48(3): 564-84.

Völlm, B. A., Taylor, A. N. W., Richardson, P., Corcoran, R., Stirling, J., McKie, S., et al. (2006). Neuronal correlates of theory of mind and empathy: A functional magnetic resonance imaging study in a non-verbal task. *NeuroImage* 29: 90–8.

Wellman, H. (1990). *The Child's Theory of Mind*. Cambridge: MIT Press.

Wicker, B., Keysers, C., Plailly, J., Royet, J. P., Gallese, V., & Rizzolatti, G. (2003). Both of us disgusted in My insula: the common neural basis of seeing and feeling disgust. *Neuron* 40(3): 655–64.

Wimmer, H., & Perner, J. (1983). Beliefs about beliefs: representation and constraining function of wrong beliefs in young children's understanding of deception. *Cognition* 13(1): 103–28.

Zaki, J., Bolger, N., & Ochsner, K. (2009). Unpacking the informational bases of empathic accuracy. *Emotion* 9: 478–87.