A Biopsychological Perspective on Empathy:

Processes of Affect-sharing and Mentalizing

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Abstract

The biopsychological study of empathy has intensified dramatically in recent years. Focusing on affect-sharing and mentalizing components of empathy, this paper describes three facets of empathy that are currently under discussion. First, contextual factors impacting empathic responses are discussed. Second, implicated brain regions are examined, along with the methods of their discovery. Finally, the role of mirror neurons in affect-sharing mechanisms is evaluated. Important directions for future research are also discussed.

Introduction

Along with other interpersonal capacities, empathy seems to have played a major role in the success of humans as a species (Zaki & Ochsner, 2012). The ability to put oneself in another's shoes is suggested to be crucial for the development of close interpersonal relationships; it allows individuals to share experiences on a more satisfying level.

But even though empathy is thought to be foundational for the human experience, it has no single definition, either in everyday language or in research settings. Zaki & Ochsner (2012) suggest that empathy can be broken down into the following three components: internal experience sharing ("affect-sharing"), cognitively putting oneself in another's shoes ("mentalizing"), and prosocial concern ("sympathy"). Biopsychologists sometimes ignore the second or third component or both, but affect-sharing is considered to be an essential component of empathy. It should be noted that this affect-sharing is of a type that distinguishes the self from the other: empathizing people can share another's feelings without blurring their identities (Bernhardt & Singer, 2012; Decety & Meyer, 2008). This paper will address three facets of empathy: contextual determinants, implicated brain regions, and the potential role of mirror neurons. In discussing empathy's contextual determinant and implicated brain regions, this paper will address both the affect-sharing and mentalizing components of empathy. Mirror neurons have only been implicated in affect-sharing (Preston & de Waal, 2002); thus, the third section of this paper will not address mentalizing.

Contextual factors are known to influence the experience of empathy. Empathic brain activity has been observed to increase when empathy is felt for fair vs. unfair game-players (Singer et al., 2006) and for those within one's group relative to those outside (Harris & Fiske, 2006; Hein, Silani, Preuschoff, Batson, & Singer, 2010). Empathic brain activity is also greater for individuals whose personalities are more empathetic (Jabbi, Swart, & Keesers, 2007; Saarela

et al., 2007) and less alexithymic (Bird et al., 2010), as measured by self-report questionnaires. Other contextual determinants include a person's beliefs about another's subjective experience (Lamm, Nusbaum, Meltzoff, & Decety, 2007) and, possibly, oxytocin levels in the empathizing person (Riem et al., 2011).

A great deal of work has been done to locate the brain's "empathy centres." This research has relied heavily on neuroimaging techniques, especially functional magnetic resonance imaging (fMRI). From these studies, it has been argued that the affect-sharing component of empathy stems from brain activity in regions including the anterior insula (AI), dorsal-anterior/anterior-midcingulate cortex (dACC/aMCC), premotor cortex (PMC), inferior parietal lobe (IPL), and posterior superior temporal sulcus (Bernhardt & Singer, 2012; Fan, Duncan, deGreck, & Northoff, 2011; Lamm & Singer, 2010; Zaki & Ochsner, 2012). Mentalizing, on the other hand, seems to stem from brain activity largely localized to the aMCC, temporoparietal junction (TPJ), temporal pole (TP), medial prefrontal cortex (MPFC), and precunius (Fan et al., 2011; Zaki & Ochsner, 2012). However, the logic behind the identification of these particular regions, along with the logic behind fMRI studies in general, may not be fully sound. Also, fMRI studies looking for empathy-linked regions have often been limited in their applicability by the artificial nature of the empathy cues employed, and by the failure to investigate how neuroimaging data correspond to behavioural outcomes (Zaki & Ochsner, 2012).

It has been argued that mirror neurons allow the affect-sharing component of empathy to occur by activating one's own physical representations of any observed emotional state (Preston & de Waal, 2002). This seems to fit with the known function of mirror neurons, which fire when a given action is either performed or observed (Kohler et al., 2002). Blair (2010) disagrees with a pure form of this hypothesis, based on the fact that context can heavily impact empathic responses. Still, others have argued that mirror neurons could provide a mechanism for some

close affective link between the self and the other (Uddin, Iacoboni, Lange, & Keenan, 2007).

This paper will examine and critique the evidence in three areas of empathy research: (a) the contextual factors impacting empathic responses, (b) the brain regions and systems implicated in the experience of empathy, and (c) the relationship between empathy and mirror neurons.

Contextual Factors Modulating Empathy

Empathy seems to be affected by characteristics of empathizers (e.g., their personality). It also appears to be modulated by characteristics of the objects of empathy (e.g., perceived fairness, ingroup vs. outgroup status), and by the empathizer's beliefs about another person's subjective experience. Oxytocin levels in the empathizer may also impact the degree of empathy felt.

The degree of empathy you experience might depend on who you are. Although everyone experiences empathy to some extent, some people certainly seem more empathetic than others. Neuroimaging empathy research has supported the idea (previously established by behavioural research) that empathy is not only an affective experience but a personal trait that can be reliably measured for individuals.

Empathy as a trait can be measured using self-report questionnaires like the Interpersonal Reactivity Index (Davis, 1983) or the Balanced Emotional Empathy Scale (Mehrabian, 1997). These scales appeared and were verified before the dramatic rise of neuroimaging empathy research, but the two approaches to studying empathy seem to integrate well. Individuals who score high on Davis' scale also appear to have greater activation of empathy-linked brain regions in empathy-inducing situations. This correlation between self-reported personal traits and fMRI-measured brain activity has been observed in empathy for pain (Saarela et al., 2007) and for gustatory pleasure or aversion (Jabbi et al., 2007).

So because neuroimaging studies show varying degrees of activity in empathy-linked regions across individuals, and because the degree of activity is correlated with Interpersonal Reactivity Index empathy measures, it stands to reason that there are true individual differences in the strength of an empathic response. In other words, an empathy trait exists and can be reliably measured. It should be noted, however, that this line of evidence is only as strong as the premise that the experience and degree of empathy can be reliably represented by activity in empathy-linked regions in neural imaging studies. (The strength of fMRI-based evidence will be evaluated in the next section.) However, even without taking fMRI studies into account, it is relatively well-established that there are reliable individual differences in empathy when it is measured by self-report or other behavioural measures. These differences exist both when measuring affect-sharing alone (Mehrabian, Young, & Sato, 1988) and when measuring combined affect-sharing and mentalizing (Baron-Cohen & Wheelwright, 2004; Davis, 1983). Again, affect-sharing refers to the process of sharing another person's internal emotional state whereas mentalizing refers to the process of cognitively putting oneself in another person's shoes.

An individual's degree of trait empathy could depend in part on her ability to identify and describe her own emotions. Alexithymic people are those who have difficulty with this at a subclinical level (Nemiah, 1977). Individuals with stronger alexithymia experienced weaker empathic responses (Bird et al., 2010) when the strength of an empathic response was measured by the degree of fMRI activity in empathy-linked brain regions. Interestingly, the regions shown to be affected by alexithymia are thought to mediate only the affect-sharing facet of empathy. This suggests that even empathic responses that seem primarily emotional may require a certain degree of cognitive self-awareness regarding one's emotions (Bernhardt & Singer, 2012). Ten percent of people are estimated to have alexithymia (Salminen, Saarijarvi, Aarela, Toikka, &

Kauhanen, 1999), and the proportion is higher among those with autism spectrum disorder (Hill, Berthoz, & Frith, 2004).

The degree of empathy experienced could also depend on characteristics of the recipient of empathy, such as their perceived fairness or unfairness. In one study, men seeing someone in pain were less likely to feel pained themselves (that is, to engage in affect-sharing) if the other was perceived as an unfair person (Singer et al., 2006). Affect-sharing was measured with neural imaging in this study as well. Female participants did not show the same fairness-based empathy modulation, for reasons that are not entirely clear. The authors suggest that the stronger male response may have resulted from the use of physical pain situations, rather than financial or psychological pain situations which hypothetically could have elicited stronger reactions from females. Follow-up studies are needed to test this idea.

The recipient's ingroup vs. outgroup status also seems to affect the degree to which empathy is experienced. fMRI studies suggest that men show decreased affect-sharing responses towards the pain of a person who cheers against their favourite soccer team (Hein et al., 2010). This study also measured the sacrificial helping behaviour that was motivated by empathic feelings, and found that outgroup members received less help. Women were not included as participants in this study.

The degree of empathic response is also impacted by an empathizer's beliefs about another person's subjective experience. These beliefs can be adjusted by explicitly informing the empathizer about a situation. For instance, affect-sharing accompanying a painful needle prick was decreased when the person in the empathic role was told that the pricked person was anesthetized (Lamm et al., 2007).

Lastly, it has been argued that oxytocin release can increase empathy; this effect was observed in a neuroimaging study in which women listened to the sound of a baby crying (Riem

et al., 2011). Increased activation of empathy-linked regions was observed for women in this situation who had been administered oxytocin as opposed to a placebo.

Studies involving hormones require careful controls and are often difficult to interpret. This is because of the extensive (though still certainly incomplete) list of factors that influence hormone levels, and because an intricate interdependence exists among the hormones themselves. Fortunately, Riem and colleagues analyzed the above findings quite carefully; for example, they controlled for the mother's menstrual stage and use of contraceptives.

It would make sense for oxytocin to impact the experience of empathy like this, as it is known to be highly involved in social bonding (see MacDonald & MacDonald, 2010). However, Singer and colleagues (2008) found no effect of oxytocin on fMRI activation of the empathy-linked areas in males. It is unclear whether this discrepancy is due to a sex difference or to an erroneous conclusion from one group.

Some may think of empathy, particularly its affect-sharing component, as a simple, gut-level response to another person's emotion. However, it seems clear from the data above that the experience of empathy may be more nuanced than this. The degree of empathy a person feels can be modulated by situational factors in complex ways, some of which require a certain level of cognitive analysis.

Neuroimaging Studies: Findings and Limitations

In neuroscience, it seems that the study of empathy has been reduced in many cases to the identification of its associated brain regions. “Mapping” empathy is a common endeavour; this is typically done by using fMRI to determine which regions are active when someone is experiencing empathy.

It seems that the affect-sharing and mentalizing components of empathy are localized to largely separate regions of the brain. The anterior insula (AI), dorsal-anterior/anterior-

midcingulate cortex (dACC/aMCC), premotor cortex, inferior parietal lobe, and posterior superior temporal sulcus have all been linked to affect-sharing (Bernhardt & Singer, 2012; Fan et al., 2011; Lamm & Singer, 2010; Zaki & Ochsner, 2012). Of these areas, the AI and dACC/aMCC seem to be the two areas most consistently implicated (Lamm et al., 2011).

Many studies have identified these affect-sharing regions by looking for areas of the brain which are active both when a subject experiences an emotion and when they watch others experience the same emotion (Zaki & Oschner, 2012). Many of the areas listed above fulfil this criteria with reasonable consistency. There is, in general, a significant degree of spatial overlap between brain activity patterns for feeling and observing. This overlap can be seen for the feeling and observation of many affective states, including fear, pleasant affect, and even social exclusion (see Bernhardt & Singer, 2012). The regions of overlap can be implicated in affect-sharing because researchers accept the concept of "neural resonance" – the idea that certain cells and cell networks involved in experiencing a given emotion are also involved in empathically feeling the emotions of others (see Bernhardt & Singer, 2012; Zaki & Oschner, 2012). The concept of neural resonance does not seem particularly far-fetched, given the subjective similarities between feeling an emotion personally and vicariously.

However, even if the neural resonance concept is valid, there may be problems with the assumptions behind the method for determining affect-sharing regions (in addition to the potential problems with placing too much faith in fMRI data, which will be discussed briefly later). Specifically, there could be a problem with the assumption that the regions of overlap are involved in the mechanism that allows the overlap. It is not necessarily true, for example, that the regions involved in both feeling and observing an emotion are responsible for inducing an individual to feel another person's emotion. It is possible, perhaps probable, that these regions are involved in both processes simply because they are responsible for some of the processes'

components. This is even more likely if these regions otherwise appear to have diffuse, nonspecific functions.

A reasonable question, then, regards the general functions of the AI and dACC/aMCC. It turns out that neither has a clear single function, but the AI has been linked generally to human awareness or consciousness (Craig, 2009) and the dACC/aMCC to cognition and motor control (Bush et al., 2002). The breadth and nonspecificity of these functions could affirm our reticence to directly map affect-sharing to these regions. If the AI is involved in human awareness, for example, perhaps we should accept that function as a sufficient explanation for the fact that it is activated while feeling or observing emotional states; perhaps there is no need to characterize it as an "empathy-linked area." Many empathy researchers are aware of the broad functions of empathy-linked areas (see Bernhardt & Singer, 2012), but certain practices suggest otherwise, such as the use of AI activity to directly represent the degree of empathy in many of the experiments described in the previous section.

Another problem with the mapping of affect-sharing involves the assumption that spatially overlapping activity between feeling and observing indicates shared representations at the neuronal level. A single fMRI voxel covers thousands of neurons (Bernhardt & Singer, 2012), so there are no guarantees that voxel activation across multiple situations indicates activity in the same cell networks. The ideal solution to this particular problem would be to obtain single-cell recording data in the human AI and dACC/aMCC (Single-cell recording data from non-human animals has questionable applicability for affect-sharing, due to our limited ability to deduce a rat's subjective experience.). However, single-cell recording is still relatively unusual in our species and typically involves recording from the medial temporal lobes of epileptic patients (Quian Quiroga, 2009) rather than the AI or dACC/aMCC.

The process of mentalizing is said to be localized in the temporoparietal junction (TPJ),

temporal pole, medial prefrontal cortex, and posterior cingulate cortex (Zaki & Ochsner, 2012). Activity is observed in these regions when people make cognitive judgements about the feelings of others (Zaki & Oschner, 2012). Such activation is not surprising. In particular, it makes sense that mentalizing tasks (in which subjects imagine what things would be like from another person's perspective) would activate regions like the TPJ, which is known to be involved in mental imagery and out-of-body experiences (Blanke et al., 2005) and theory of mind (Saxe & Kanwisher, 2003). Mentalizing is quite similar to using theory of mind, but more specific; mentalizing here refers to understanding the emotional experience of others, rather than understanding their perspective in general. Perhaps the process of mentalizing recruits cell networks related to theory of mind (Lamm et al., 2011).

Before ending this section, a few difficulties should be mentioned regarding the interpretation of fMRI studies, which are currently the most common type of experiment used to discover the neural processes of human empathy (Lamm et al., 2011). fMRI works by assuming that the strength of the blood oxygen level-dependent (BOLD) signal represents the degree of brain activity in any given region. However, we know that the magnitude and time onset of the BOLD response to brain activity can differ across individuals, and even across brain regions within individuals (Ramsey et al., 2010). Also, fMRI studies generally report regions of activation by giving the spatial averages of data from many subjects; thus, large individual differences in spatial activation patterns could give misleading results (Ramsey et al., 2010). In addition, the interpretation of fMRI data often relies heavily on questionable statistical procedures (Ramsey et al., 2010). Lastly, the stimuli used to evoke empathic responses in many fMRI studies have been somewhat artificial; for example, a typical cue might have been an isolated picture of a facial expression or a one-sentence description of an empathy-inducing situation (Zaki & Oschner, 2012). This paradigm may not adequately represent naturalistic

settings, in which empathy responses based on a person's evaluation of a wide variety of social cues (Zaki & Oschner, 2012). This approach been misleading in multiple ways. For instance, affect-sharing and mentalizing have been seen as entirely dissociated, since typically only one of these systems is activated by artificial stimuli; however, under more naturalistic paradigms it appears that these systems are more tightly integrated than previously thought (Zaki & Oschner, 2012).

However, fMRI studies do have some value. They have elucidated regions of the brain that are involved in our experience of empathy, even if these regions are not entirely responsible for it. The empathy-linked nature of these regions has been corroborated by the correlation of their high activation with high empathy scores on the Interpersonal Reactivity Index, as mentioned above. fMRI studies have also clearly demarcated the empathic systems for affect-sharing and mentalizing, which do have distinct functions (Zaki & Oschner, 2012). These studies also provide reasonable starting points for future research. Possible directions for this research will be discussed at the end of this paper.

Mirror Neurons and Affect-Sharing

Mirror neurons are cells that fire when a goal-directed action is either performed or observed (Rizzolati & Craighero, 2004). They were first discovered in rhesus monkeys, but exist in humans as well (Mukamel, Ekstrom, Kaplan, Iacoboni, & Fried, 2010; Rizzolati & Craighero, 2004). Some have argued that mirror neurons provide the basis for the affect-sharing component of empathy (Preston & de Waal, 2002). In this section, we will briefly evaluate this argument, looking first at its theoretical basis and subsequently discussing relevant neuroimaging and behavioural data.

The reason mirror neurons have been been linked with empathy is because, on some level, they allow us to experience what we observe. The perception-action model is a way of

explaining how this allows affect-sharing (Preston & de Waal, 2002). The central tenet of the perception-action model is that when individuals observe someone in a given emotional state, the observation activates their physical representations of that state (that is, the cell networks that fire in the normal experience of that state) (Preston & de Waal, 2002). Mirror neurons could be involved somehow in this activation step. These physical representations then activate their normally associated bodily responses, and together these factors cause the observer to experience the observed emotion – that is, to engage in affect-sharing (Preston & de Waal, 2002). Decety & Moriguchi (2007) provide an example: when we look at another person smiling, our facial smiling muscles are activated (this may involve mirror neurons). The facial smiling muscles are not activated strongly enough to produce an observable smile, but they are activated strongly enough to make us feel happy due to facial feedback. This is believed to be an automatic process (Decety & Jackson, 2004).

This theory runs into a few problems, particularly in its simplest form. Blair (2010) argues that the perception-action model doesn't have room for the many contextual variables which can modulate affect-sharing. For example, certain situations cause people to feel pleasure at another's pain (Dvash, Gilam, Ben-Ze'ev, Hendler, & Shamay-Tsoory, 2010). It is unclear how mirror neurons could be involved in this, unless their role in engaging physical representations of an emotional state is quite nuanced (Blair, 2010). Blair argues that mirror neurons are not necessary to describe affect-sharing, which he views as a conditioned response to social cues.

fMRI data has also contributed to the current discussion of mirror neurons and affect-sharing. In a recent review, Bernhardt & Singer (2012) point out that there is some overlap between the regions of the brain linked to affect-sharing and those believed to house the human mirror neuron system. Of course, fMRI activation of the mirror neuron region cannot confirm the involvement of mirror neurons, but may suggest it.

Another approach to the study of mirror neurons and empathy has been to assess the correlation between covert motor imitation of others' movements and empathy scores on the Interpersonal Reactivity Index (Baird, Scheffer, & Wilson, 2011). Mirror neurons have been proposed to explain covert motor imitation, though this assertion has been disputed (see Hicock, 2009; Rizzolati & Craighero, 2004). The goal of these studies is generally to show that people who show a higher degree of empathy also show a higher degree of covert muscle imitation, and from this to argue that the mirror neuron system must be involved in empathy as well as imitation. The two studies using this approach have obtained conflicting results, possibly because of varied procedures for assessing the degree of motor imitation (see Baird et al., 2011). Also, the Interpersonal Reactivity Index may not have been the best testing tool to use in this study, since it tests for a relatively broad concept of empathy that includes more than simply affect-sharing (Davis, 1983). However, it is also possible that there is simply no imitation-empathy correlation to be found.

It seems that the evidence is not yet strong enough to conclude that mirror neurons do or do not mediate affect-sharing. The theoretical basis for the involvement of mirror neurons seems reasonable, though perhaps perception-action theory simplifies their role too much. Functional neuroimaging cannot yet give us conclusive answers, and correlational studies looking at imitation and empathy have not given a cohesive set of results to interpret.

Conclusions and Future Directions

The biopsychological study of empathy has come a long way since its beginning. We are much more well-informed about contextual factors, associated brain regions, & mirror neuron involvement than we were ten years ago.

We know, for instance, that the degree of empathy is associated with the empathizer's personality and degree of alexithymia. It is affected by characteristics of the object of empathy,

including perceived fairness and ingroup vs outgroup membership. Explicit beliefs about how another person is experiencing a situation can also play a role in determining the degree of empathy experienced towards them, as can (disputably) oxytocin levels.

We also know something about the regions that are active when people are experiencing affect-sharing and mentalizing. Key areas appear to include the AI and dACC/aMCC (for affect-sharing) and the TPJ and medial prefrontal cortex (for mentalizing). However, our knowledge about this comes from fMRI studies, which may have questionable validity.

The involvement of mirror neurons in affect-sharing is still an open question, but neuroimaging data does show activity in the areas we would expect to see if the mirror neuron system was involved. However, there may be problems with using a pure perception-action hypothesis to explain mirror neuron involvement.

Biopsychologists are beginning to study empathy in many novel ways that have not been mentioned yet; these branch out from the functional neuroimaging studies that have been standard (Zaki & Oschner, 2012). This seems like an excellent development for the field. It does make sense to investigate the neural underpinnings of empathy, but finding the regions in which activity occurs may not be an entirely satisfactory way to do this. Additionally, focusing empathy research on neuroimaging studies can cause researchers to ignore processes taking place in the rest of the body.

Exciting recent areas of empathy research include experiments using genetics, pharmacology, and transcranial magnetic stimulation (see Zaki & Oschner, 2012). Other researchers have used an electroencephalographic framework to test for event-related potentials, or ERPs (Fan & Han, 2008). ERPs have much higher temporal resolution than fMRI, and these experiments have shown that affect-sharing brain activity typically occurs before mentalizing brain activity (Fan & Han, 2008).

Many fMRI studies are also improving in their methods (Zaki & Oschner, 2012). For example, some researchers are using more naturalistic stimuli and are collecting behavioural and self-report data in addition to observing regions of neural activity (see Zaki & Oschner, 2012). As mentioned in a previous section, these studies have shown affect-sharing and mentalizing to be more tightly integrated than was previously thought, co-occurring in almost every empathic situation. Armed with this knowledge, neuroscientists studying empathy can relate their work more directly to the vast empathy literature of psychologists, who have typically been less insistent about breaking empathy into subcomponents.

Work remains to be done, both in the novel directions of empathy research mentioned here and in this paper's primary focus areas (contextual factors, associated brain regions, and the role of mirror neurons). In this field, many open questions and opportunities remain.

References

Baird, A.D., Scheffer, I.E., & Wilson, S.J. (2011). Mirror neuron system involvement in empathy: A critical look at the evidence. *Social Neuroscience, 6,* 327-335. doi:10.1080/17470919.2010.547085

Baron-Cohen, S., & Wheelwright, S. (2004). The empathy quotient: An investigation of adults with Asperger Syndrome or high functioning autism, and normal sex differences. *Journal of Autism and Developmental Disorders, 34,* 163-175. doi:10.1023/B:JADD.0000022607.19833.00

Bernhardt, B.C., & Singer, T. (2012). The neural basis of empathy. *Annual Review of Neuroscience, 35,* 1-23. doi:10.1146/annurev-neuro-062111-150536

Bird, G., Silani, G., Brindley, R., White, S., Frith, U., & Singer, T. (2010). Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain, 133,* 1515-1525. doi:10.1093/brain/awq060

Blair, R.J.R. (2010). Empathy, moral development, and aggression: A cognitive neuroscience perspective. In W. F. Arsenio, E. A. Lemerise (Eds.), *Emotions, aggression, and morality in children: Bridging development and psychopathology* (pp. 97-114). Washington, DC: American Psychological Association. doi:10.1037/12129-005

Blanke, O., Mohr, C., Michel, C.M., Pascual-Leone, A., Brugger, P., Seeck, M., . . . Thut, G. (2005). Linking out-of-body experience and self processing to mental own-body imagery at the temporoparietal junction. *The Journal of Neuroscience, 25,* 550-557. doi:10.1523/JNEUROSCI.2612-04.2005

Bush, G., Vogt, B.A., Holmes, J., Dale, A.M., Greve, D., Jenike, M.A., & Rosen, B.R. (2002). *Proceedings of the National Academy of Sciences of the United States of America, 99,* 523-528. doi:10.1073/pnas.012470999

Craig, A.D. (2009). How do you feel – now? The anterior insula and human awareness. *Nature Reviews Neuroscience, 10,* 59-70. doi:10.1038/nrn2555

Cox, C.L., Uddin, L.Q., Di Martino, A., Castellanos, F.X., Milham, M.P., & Kelly, C. (2012). The balance between feeling and knowing: Affective and cognitive empathy are reflected in the brain's intrinsic functional dynamics. *Social Cognitive and Affective Neuroscience, 7,* 727-737. doi:10.1093/scan/nsr051

Davis, M.H. (1983). Measuring individual differences in empathy: Evidence for a multidimensional approach. *Journal of Personality and Social Psychology, 44,* 113-126.

Decety, J., & Ickes, W. (Eds.). (2011). The social neuroscience of empathy. Cambridge, MA: MIT Press.

Decety, J., & Jackson, P. L. (2004). The functional architecture of human empathy. *Behavioral and Cognitive Neuroscience Reviews, 3,* 71–100. doi:10.1177/1534582304267187

Decety, J., & Meyer, M. (2008). From emotion resonance to empathic understanding: A social developmental neuroscience account. *Development and Psychopathology, 20,* 1053-1080. doi:10.1017/S0954579408000503

Decety, J., & Moriguchi, Y. (2007). The empathic brain and its dysfunction in psychiatric populations: Implications for intervention across different clinical conditions. *BioPsychoSocial Medicine, 1,* 22–65. doi:10.1186/1751-0759-1-22

Dvash, J., Gilam, G., Ben-Ze'ev, A., Hendler, T., Shamay-Tsoory, S.G. (2010). The envious brain: The neural basis of social comparison. *Human Brain Mapping, 31,* 1741-1750. doi:10.1002/hbm.20972

Fan, Y., Duncan, N.W., deGreck, M., & Northoff, G. (2011). Is there a core neural network in empathy? An fMRI based quantitative meta-analysis. *Neuroscience & Behavioral Reviews, 35,* 903-911. doi:10.1016/j.neubiorev.2010.10.009

Fan, Y., & Han, S. (2008). Temporal dynamic of neural mechanisms involved in empathy for pain: An event-related brain potential study. *Neuropsychologia, 46,* 160-173. doi:10.1016/j.neuropsychologia.2007.07.023

Gallese, V., Keysers, C., & Rizzolatti, G. (2004). A unifying view of the basis of social cognition. *Trends in Cognitive Sciences, 8,* 396-403. doi:10.1016/j.tics.2004.07.002

Harris, L.T., & Fiske, S.T. (2006). Dehumanizing the lowest of the low: Neuroimaging responses to extreme out-groups. *Psychological Science, 17,* 847-853. doi:10.1111/j.1467-9280.2006.01793.x

Hein, G., Silani, G., Preuschoff, K., Batson, C.D., & Singer, T. (2010). Neural responses to ingroup and outgroup members' suffering predict individual differences in costly helping. *Neuron, 68,* 149-160. doi:10.1016/j.neuron.2010.09.003

Hickok, G. (2009). Eight problems for the mirror neuron theory of action understanding in monkeys and humans. *Journal of Cognitive Neuroscience, 21,* 1229-1243. doi:10.1162/jocn.2009.21189

Hill, E., Berthoz, S., & Frith, U. (2004). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives. *Journal of Autism and Developmental Disorders, 34,* 229-235. doi:10.1023/B:JADD.0000022613.41399.14

Jabbi, M., Swart, M., & Keysers, C. (2007). Empathy for positive and negative emotions in the gustatory cortex. *Neuroimage, 34,* 1744-53. doi:10.1016/j.neuroimage.2006.10.032

Kohler, E., Keysers, C., Umilta, M.A., Fogassi, L., Gallese, V., & Rizzolatti, G. (2002). Hearing sounds, understanding actions: Action representation in mirror neurons. *Science, 297,* 846-848. doi:10.1126/science.1070311

Lamm, C., Decety, J., & Singer, T. (2011). Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. *NeuroImage, 54,* 2492-2502. doi:10.1016/j.neuroimage.2010.10.014

Lamm, C., Nusbaum, H.C., Meltzoff, A.N., & Decety, J. (2007). What are you feeling? Using functional magnetic resonance imaging to assess the modulation of sensory and affective responses during empathy for pain. *PloS One, 12,* e1292. doi:10.1371/journal.pone .0001292

Lamm, C., & Singer, T. (2010). The role of anterior insular cortex in social emotions. *Brain Structure and Function, 214,* 579-591. doi:10.1007/s00429-010-0251-3

MacDonald, K., & MacDonald,T.M. (2010). The peptide that binds: A systematic review of oxytocin and its prosocial effects in humans. *Harvard Review of Psychiatry, 18,* 1-21. doi:10.3109/10673220903523615

Mehrabian, A. (1997). Relations among personality scales of aggression, violence, and empathy: validational evidence bearing on the risk of eruptive violence scale. *Aggressive Behavior, 23,* 433-445. doi:10.1002/(SICI)1098-2337(1997)23:6<433::AID-AB3>3.0.CO;2-H

Mehrabian, A., Young, A.L., & Sato, S. (1988). Emotional empathy and associated individual differences. *Current Psychology, 7,* 221-240. doi:10.1007/BF02686670

Mukamel, R., Ekstrom, A.D., Kaplan, J., Iacoboni, M., & Fried, I. (2010). Single neuron responses in humans during execution and observation of actions. *Current Biology, 20,* 750-756. doi:10.1016/j.cub.2010.02.045

Nemiah, J.C. (1977). Alexithymia. Theoretical considerations. *Psychotherapy and Psychosomatics, 28,* 199-206.

Preston, S., & de Waal, F.B.M. (2002). Empathy: Its ultimate and proximate bases. *Behavioral and Brain Sciences, 25,* 1-72. doi:10.1017/S0140525X02000018

Quian Quiroga, R. (2009). Single-neuron recordings in epileptic patients. *Advances in Clinical Neuroscience and Rehabilitation, 9,* 8-10.

Ramsey, J.D., Hanson, S.J., Hanson, C., Halchenko, Y.O., Poldrack, R.A., & Glymour, C. (2010). Six problems for causal inference from fMRI. *NeuroImage, 49,* 1545-1558. doi:10.1016/j.neuroimage.2009.08.065

Riem, M.E., Bakermans-Kranenburg, M.J., Pieper, S., Tops, M., Boksem, M.A.S., Vermeiren, R.R.J.M., . . . Rombouts, S.A.R.B. (2011). Oxytocin modulates amygdala, insula, and inferior frontal gyrus responses to infant crying: A randomized controlled trial. *Biological Psychiatry, 70,* 291-297. doi:10.1016/j.biopsych.2011.02.006

Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience, 27,* 169-192. doi:10.1146/annurev.neuro.27.070203.144230

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,* 230-237. doi:10.1093/cercor/bhj141

Salminen, J.K., Saarijarvi, S., Aarela, E., Toikka, T., & Kauhanen, J. (1999). Prevalence of alexithymia and its association with sociodemographic variables in the general population of Finland. *Journal of Psychosomatic Research, 46,* 75-82. doi:10.1016/S0022-3999(98)00053-1

Saxe, R., & Kanwisher, N. (2003). People thinking about thinking people: The role of the temporo-parietal junction in “theory of mind”. *NeuroImage, 19,* 1835-1842. doi:10.1016/S1053-8119(03)00230-1

Singer, T., & Lamm, C. (2009). The social neuroscience of empathy. *The Year in Cognitive Neuroscience, 1156,* 81-96. doi:10.1111/j.1749-6632.2009.04418.x

Singer, T., Seymour, B., O'Doherty, J.P., Stephan, K.E., Dolan, R.J., & Frith, C.D. (2006). Empathic neural responses are modulated by the perceived fairness of others. *Nature, 439,* 466-469. doi:10.1038/nature04271

Singer, T., Snozzi, R., Bird, G., Petrovic, P., Silani, G, Heinrichs, M., & Dolan, R.J. (2008). Effects of oxytocin and prosocial behavior on brain responses to direct and vicariously experienced pain. *Emotion, 8,* 781-791. doi:10.1037/a0014195

Uddin, L.Q., Iacoboni, M., Lange, C., & Keenan, J.P. (2007). The self and social cognition: The role of cortical midline structures and mirror neurons. *Trends in Cognitive Science, 11,* 153-157. doi:10.1016/j.tics.2007.01.001

Zaki, J., & Ochsner, K. (2012). The neuroscience of empathy: progress, pitfalls and promise. *Nature Neuroscience, 15,* 675-680. doi:10.1038/nn.3085