Neuroscience Research 90 (2015) 15–24

Contents lists available at ScienceDirect

Neuroscience Research

journal homepage: www.elsevier.com/locate/neures

Review article

# The role of shared neural activations, mirror neurons, and morality in empathy – A critical comment

Claus Lamm[a,b,*], Jasminka Majdandžić[a,b,**]

[a] Social, Cognitive and Affective Neuroscience Unit, Department of Basic Psychological Research and Research Methods, Faculty of Psychology, University of Vienna, Austria
[b] Cognitive Science Research Platform, University of Vienna, Austria

ARTICLE INFO

*Article history:*
Received 1 September 2014
Received in revised form
19 September 2014
Accepted 22 September 2014
Available online 20 October 2014

*Keywords:*
Empathy
Morality
Mirror neurons
Social neuroscience
Simulation
Shared representations

ABSTRACT

In the last decade, the phenomenon of empathy has received widespread attention by the field of social neuroscience. This has provided fresh insights for theoretical models of empathy, and substantially influenced the academic and public conceptions about this complex social skill. The present paper highlights three key issues which are often linked to empathy, but which at the same time might obscure our understanding of it. These issues are: (1) shared neural activations and whether these can be interpreted as evidence for simulation accounts of empathy; (2) the causal link of empathy to our presumed mirror neuron system; and (3) the question whether increasing empathy will result in better moral decisions and behaviors. The aim of our review is to provide the basis for critically evaluating our current understanding of empathy, and its public reception, and to inspire new research directions.



**Contents**



Empathy is a complex social phenomenon whose many facets have fascinated scholars from various fields and laymen for centuries. Only recently, the field of Social Neuroscience has begun to shed light on the neural underpinnings of this phenomenon. A dominant part of this growing understanding can certainly be attributed to the increased availability and precision of neuroimaging methods such as functional magnetic resonance imaging (fMRI). If one were to trace back the “birthday” of the social neuroscience of empathy, one would certainly end up at the seminal fMRI study by Singer and colleagues (Singer et al., 2004) which showed that experiencing pain and empathizing with the pain of others evoke overlapping neural activations in cingulate and insular cortices. This study not only attracted enormous public and scientific interest (with over 1200 citations by peer-reviewed ISI-listed journals, at the time of writing this article), but also helped to jumpstart the field of social neuroscience, which was then still in its infancy but now has become one of the most thriving fields of neuroscientific

* Corresponding author at: Department of Basic Psychological Research and Research Methods, Faculty of Psychology, University of Vienna, Liebiggasse 5, 1010 Vienna, Austria. Tel.: +43 1427747130.
** Corresponding author at: Department of Basic Psychological Research and Research Methods, Faculty of Psychology, University of Vienna, Liebiggasse 5, 1010 Vienna, Austria.
*E-mail addresses:* claus.lamm@univie.ac.at (C. Lamm), jasminka.majdandzic@univie.ac.at (J. Majdandžić).

http://dx.doi.org/10.1016/j.neures.2014.10.008

inquiry. Since this publication, about 10 years ago (and the publication of other equally influential papers around the same time: Decety and Jackson, 2004; Jackson et al., 2005; Morrison et al., 2004; Preston and de Waal, 2002; Wicker et al., 2003) we have seen a tremendous increase in scientific publications revolving around the question of the neural computations and networks that enable us to share the feelings of others. As shown by a Pubmed search of ["empathy" and ("brain" or "neural" or "neuroscience")], performed on September 18, 2014, the handful of papers available in 2004 have now increased to 1300 listed papers, with 2245 of these papers published within the preceding year.

The goal of the present review is not, however, to provide an exhaustive summary of what we have learned from these investigations. For this, a correspondingly high number of recent reviews are available (e.g. Bernhardt and Singer, 2012; Decety et al., 2012; Keysers and Gazzola, 2014a; Shamay-Tsoory, 2011; Singer and Lamm, 2009; Zaki and Ochsner, 2012). Rather, in our extended commentary, we would like to put the spotlight on three issues that in our view currently encumber the field of empathy research. Our intention is not so much an in-depth scientific discourse on these issues, or to criticize the field, since most likely most of our colleagues are equally aware of them, or see them in a very similar way. Rather, we aim to provide some basis for a discussion on how to overcome some common misconceptions and their implications, targeting also science communicators and the interested public who in their enthusiasm have at times misinterpreted and miscommunicated the insights on empathy generated by social neuroscience.

The three issues we discuss are (a) the functional interpretation of shared neural activations and what they tell us about the mechanisms of empathy; (b) the role of mirror neurons in empathy; and (c) the relationship of empathy with morality. In order to give some context to our arguments, we first briefly summarize some of the main insights generated by the neuroscientific study of empathy.

## 1. The neural networks involved in empathy

One of the major conceptual findings of Singer et al.'s "seed study", which was probably also one of the reasons it had such a strong impact, was the observation that empathy recruits similar neural networks as the direct experience of the emotion one is showing empathy for. Confirming similar work in the domain of disgust (Wicker et al., 2003), their study showed that the anterior insular (AI) cortex and the anterior midcingulate cortex (aMCC according to Vogt, 2005 but in some studies and in most initial work referred to as anterior cingulate cortex, ACC) were activated when observing the pain of others. This finding has since been confirmed by numerous subsequent studies, as documented by image-based and coordinate-based meta-analyses which have quantitatively integrated and summarized the available data (Fan et al., 2011; Lamm et al., 2011). Notably, AI and aMCC are part of the so-called pain neuromatrix, the network of brain areas that is activated when one undergoes painful stimulation oneself (Derbyshire, 2000). Resemblance between neural activity during direct emotion experiences and specific aspects of empathy (in particular motor resonance, see discussion below) was also observed in studies using other methods, such as electroencephalography (EEG), motor evoked potential transcranial magnetic stimulation (MEP-TMS), and even presurgical intracranial electrophysiology (e.g. Avenanti et al., 2005; Bufalari et al., 2007; Hutchison et al., 1999; Perry et al., 2010). The similarity between neural activations for self- and other-related emotion experiences has motivated the interpretation that recruiting mental representations that normally underlie direct emotion experiences is a central mechanism enabling empathy and affective resonance. In other words, it has been suggested that we are able to understand and share the emotions of others by (partially) processing them with our very own emotion system(s). This has also fostered interpretations placing processes such as simulation and self-projection at the core of empathy – mechanisms of empathy that had already been proposed before the availability of functional neuroimaging evidence (Gallese and Goldman, 1998). Importantly, this view of empathy as a simulative process did not emerge in a vacuum. Rather, it was influenced by similar findings and interpretations in the motor domain, such as the discovery of mirror neurons, and claims that these neurons, which fire both when the individual performs an action and when it observes its execution by others, lie at the root of understanding others' actions (see Ferrari and Rizzolatti, 2014, for a recent review). Similarly, in social cognition, simulation and self-projection have been interpreted repeatedly as core mechanism of mentalizing, i.e., considering others' beliefs, intentions or thoughts (Goldman and Sebanz, 2005; Mitchell, 2009). We will critically discuss these claims and the available evidence for it below.

## 2. Different mechanisms and neural routes to empathy

Empathy has come in (too) many different definitions and descriptions – and this has certainly also infected the field of social neuroscience (see Batson, 2011, for an excellent overview). Yet, several social neuroscientists have argued for a definition that requires at least a partial, isomorphic sharing of the feeling(s) of another person to be classified as empathy (e.g. Bernhardt and Singer, 2012; Decety and Lamm, 2006; Decety et al., 2012; Gonzalez-Liencres et al., 2013; Singer and Lamm, 2009). From the viewpoint of social neuroscience, this interpretation is mainly based on the fact that AI and MCC are brain structures associated with the affective-motivational aspects of pain, Outside of the domain of pain, these areas are associated with functions strongly linked to emotional experiences as well – such as conjoint interoception and homeostatic regulation (Medford and Critchley, 2010). To understand the neuro-psychological mechanisms of empathy, it is not sufficient to focus on its affective components, though. Indeed, numerous investigations have consistently shown that motor and cognitive functions play important roles in the instigation and modulation of empathy. For instance, observing someone else getting his hand jammed in a door or cutting his finger (Jackson et al., 2005, 2006), getting an injection in his hand, or undergoing acupuncture (e.g. Avenanti et al., 2005; Cheng et al., 2007; Lamm et al., 2007b; Perry et al., 2010) has been shown to elicit "motor resonance" processes, which in turn may trigger the affective response to the other's pain. Notably, early but influential models of empathy had already proposed the notion of a tight perception-action coupling in the brain, and the automatic motor resonance resulting from it, as a core mechanism subserving empathy (Preston and de Waal, 2002). Likewise, observing others being touched engages our somatosensory system, seemingly enabling us to code the affective qualities of vicariously perceived touch (see Bufalari and Ionta, 2013; Keysers et al., 2010 for reviews). In the cognitive domain, the ability to deliberately adopt the perspective of others and to imagine their feelings, even without direct observation, can be an equally potent instigator of affective responses (Jackson et al., 2006; Lamm et al., 2007a) and ensuing prosocial behaviors (Hein et al., 2010, 2011). This capacity has mainly been assigned to brain structures associated with theory of mind and mentalizing, such as the medial prefrontal cortex, precuneus, and temporo-parietal junction (e.g. Shamay-Tsoory, 2011), i.e., with processes that are primarily engaged when reflecting on non-affective mental states of others.

These observations have led to the introduction of terms such as motor empathy and cognitive empathy, pitting them against affective empathy. However, it seems more useful in terms of

conceptual clarity, and more in line with the definition of empathy as a phenomenon of affective sharing, to see motor, somatosensory and cognitive processes as key *mechanisms* (supported by distinct neural pathways) of evoking an empathic response, rather than as disparate components of the empathic response itself. For instance, we have recently demonstrated that the same network of AI and MCC thought to underlie the affective component of empathy can be engaged by distinct "motor" and "cognitive" neural routes (Lamm et al., 2011). While evoking affective empathic responses by means of pictures of another person's body parts in painful conditions recruited parietal and premotor structures related to action observation, showing participants abstract visual cues that indicated the occurrence of painful electrical stimulation engaged brain areas related to mentalizing and perspective taking. This view does by no means devaluate the importance of motor, somatosensory of cognitive pathways to empathy, or their usefulness in theoretical models of it. Rather, it intends to avoid a "double assignment" of identical phenomena to distinct concepts. For instance, with respect to "cognitive empathy", distinguishing affective perspective taking (i.e., perspective taking to understand affect) from cognitive perspective taking (perspective taking to understand cognition) seems much cleaner and more parsimonious than calling the former "empathy" and the latter "theory of mind" (see also Sebastian et al., 2012; Singer, 2006).

## 3. Three central but often misunderstood issues in the neuroscience of empathy

After this brief review, we would now like to discuss three in our view central issues that frequently come up when discussing the neural mechanisms of empathy, and which are highly relevant for our current understanding (or misunderstanding) of empathy.

### 3.1. What do shared neural activations reflect?

As outlined above, there is compelling evidence that similar neural structures are activated when empathizing with someone and when directly experiencing the emotion one is empathizing with. However, what role these shared activations play in empathy is much less obvious. The range of possible explanations parallels the diversity of viewpoints in the debate on the functional role of mirror neurons that we will discuss in a later section (Csibra, 2008; Gallese and Goldman, 1998; Hickok, 2009; Uithol et al., 2011). Just as scientists have disagreed on whether mirror neuron responses *serve* action understanding or *reflect* action understanding, affective neural responses, such as the activations in aMCC and AI during empathy for pain, could either be interpreted as a *route* to understanding others' feelings on a more elaborate level, or as a mere *sign* of it. Although most scholars seem to have endorsed the first interpretation, and we have positioned ourselves close to this camp in the past as well (e.g., Lamm and Singer, 2010; Singer and Lamm, 2009) we do not think that we have sufficient evidence to unequivocally decide between these two explanations, or any intermediate version of them. Rather, we would like to emphasize the need for caution in interpreting findings of shared neural activations in a particular theoretical direction. In addition, we will outline below some of the factors that prevent a clear-cut interpretation of shared activations, and discuss some ideas on how to possibly overcome these obstacles.

One obvious methodological limitation is that fMRI activations that overlap between two different conditions do not necessarily imply that the same neural processes are engaged (e.g. see Grill-Spector and Malach, 2001). This limitation stems from both the imprecision inherent to the hemodynamic response which is only an indirect measure of neural activity, and the coarse spatial resolution of fMRI, with each measurement voxel covering thousands of neurons. These neuron populations might show a different firing and interaction pattern under different conditions, but their net metabolic activity might be comparable, producing hemodynamic responses that are indistinguishable with fMRI. Similar limitations apply to other methods such as EEG (in particular for frequency-based analyses) and MEP-TMS, where different neural inputs can create similar electrophysiological output, and where measures are also based on mass neural activation and not on firing patterns of single neurons. These physiological and methodological limitations are a hindrance to decide whether shared neural activations imply shared emotion *representations* in the strict (functional-psychological) sense of the term. One possible remedy is to use more fine-grained data acquisition in combination with multivariate imaging analysis methods that operate on the level of information rather than on the level of activations, as classical univariate fMRI analyses do. For instance, multivariate pattern analysis (MVPA) has recently been used to support claims that directly experiencing pain and perceiving pain in others rely on the same local activation patterns (Corradi-Dell'Acqua et al., 2011). This promising first step however awaits independent confirmation and extension to approaches that incorporate different types of pain, and a better control of domain-general aspects such as salience (Valentini and Koch, 2012).

A related methodological issue is that methods such as fMRI, EEG or MEP-TMS are correlational by nature (see e.g. Logothetis, 2008), and therefore only provide information on neural responses co-occurring with the experience of empathy. In order to achieve mechanistic interpretations, such as the one that shared activations are a necessary condition for subjective experiences of empathy, they need to be complemented by neuropsychological lesion studies or by neurostimulation studies, which provide more causal evidence than correlational methods. An increasing number of neuropsychological studies on empathy have recently been published (for an excellent review see Hillis, 2014). However, their significance so far is limited by the fact that there are no patients with circumscribed lesions of anterior insula and midcingulate cortex only (or more generally speaking of areas specifically related to distinct emotions and empathy alike), and by the fact that many studies were based on either self-report measures of trait empathy, or used experimental paradigms whose dependent variables captured processes other than empathy in the sense of affect sharing. Future studies are therefore needed that bridge the fields of neuropsychology with social neuroscience more exhaustively. Nevertheless, recent research has yielded some promising causal evidence, for instance, data suggesting that damage to the anterior insula results in reduced affective perspective taking (e.g. Leigh et al., 2013). As for neurostimulation in healthy volunteers, there are currently no methods available that convincingly allow non-invasive stimulation of structures such as aMCC and AI, which are located deeply under the cortical mantle. However, there is an increasing number of tDCS and TMS studies targeting motor, cognitive or sensory processes related to empathy or playing a part in triggering it (e.g. Catmur et al., 2011; Hetu et al., 2012; Lev-Ran et al., 2012; Santiesteban et al., 2012). Recently, we also used TMS of the right supramarginal gyrus (rSMG) to disrupt self-other distinction, and could demonstrate that this results in egocentrically biased empathic judgments (Silani et al., 2013). Whether this effect is limited to judgments or also extends to a decrease of affect sharing still needs to be shown though, and is currently investigated by our group (see also Tomova et al., 2014). We are therefore optimistic that future studies will continue to expand our knowledge on causal mechanisms. Moreover, an increasing number of developmental social neuroscience studies started to tap into causality questions by exploiting the fact that some areas of the brain related to empathy show changes in functionality over the lifespan. For

instance, Steinbeis and colleagues were able to demonstrate that age-related differences in function and structure of the rSMG predict differences in overcoming egocentric empathic bias in children vs. adults. Interestingly, this study also showed higher activation in anterior insula (as an indication of affect sharing) in adults, whose rSMG was fully developed (Steinbeis et al., 2014).

A further limitation in addressing the role of shared activations is the fact that the vast majority of studies have investigated empathy for negative affect, and in particular pain. This prevents testing one of the central predictions of the simulative shared representations account: If vicarious activation of the neural networks involved in the direct experience of a certain emotion really reflects empathic sharing of the other person's affect, then different networks should be engaged when empathizing with emotions that are represented differently on this level. For instance, empathizing with positive emotions such as joy and happiness should result in activation in structures engaged during the direct experience of positive affect, such as the ventral striatum and the medial orbitofrontal cortex. Furthermore, different activation patterns should also emerge when empathizing with different types of emotions, analogous to the activation differences related to the first-person experience of those emotions. Unfortunately, only few studies compared different emotions or emotions of differing valence within one design, and they produced mixed results. For instance, Perry et al. investigated empathy for distress and for joy (Perry et al., 2012), and observed largely overlapping activation patterns to these different emotions, both including the insular cortex and medial prefrontal cortex. However, activations were more pronounced when empathizing with distress rather than joy. On the other hand, Morelli et al. (2014) who used a combination of contextual descriptions and pictorial presentations of others' pain, anxiety or happiness showed that while pain and anxiety engaged bilateral aIns and aMCC, happiness activated the ventromedial prefrontal cortex (vmPFC). Only recently we were able to confirm and extend these findings (Lamm et al., submitted for publication). Using an experimental paradigm which induced pleasant and unpleasant affective states in participants by means of visuo-tactile stimulation, and then asking them to empathize with another person undergoing the same types of stimulation, we showed that sharing pleasant touch recruits a different network than sharing unpleasant touch. Specifically, empathizing with pleasant touch and directly experiencing it activated the medial orbitofrontal cortex, while directly and vicariously experienced unpleasant touch activated the anterior insula. Hence, one of the main preconditions of a simulative explanation seems to be supported: the vicarious engagement of distinct neural networks for emotions of different valence, paralleling the networks related to the self-related experience of these emotions. On the other hand, we should bear in mind a further major limitation, namely that our brain organization might not respect the commonsense categories that we have devised for our emotions; rather, different emotions seem to be subserved by broadly distributed, largely overlapping functional networks that might be involved in a range of further emotional or non-emotional mental states (Lindquist and Barrett, 2012).

Finally, another issue that makes it hard to decide how to functionally interpret shared activations is the distinction between self-centered and other-oriented responses. More specifically, as highlighted already a long time ago by social psychologists, witnessing the suffering of others can trigger both a self-oriented "personal distress" response, and an other-oriented "empathic concern" response (e.g. Batson et al., 1987). To complicate things, within the latter concept, another distinction to be made is the one between empathy in the sense of "feeling as" another person (i.e. representing her feelings as if they were our own), and "feeling for" her, which is an empathic (concern) response more akin to concepts such as sympathy, compassion, or care for the other (see Batson, 2011; Singer and Lamm, 2009 for a differentiation between these terms). Hence, activation in the anterior insula or the MCC when witnessing the pain of others might be related to any of these three phenomena. To personal distress, because seeing someone else suffer causes an aversive response resulting in increased autonomic arousal and withdrawal motivations, whose bodily concomitants are interocepted in the anterior insula, and homeostatically regulated in the MCC (see Craig, 2009; Medford and Critchley, 2010 for reviews). To empathy in the sense of "feeling as", because we "replicate" the other's neural activation of these structures related to the affective-motivational component of pain. And to empathic concern as "feeling for" because the activations might also be related to a salient motivation to relieve the suffering of the other person, stemming either from the vicarious "feeling as" emotional response, or from a cognitive evaluation of the other's state and how one should act upon it. Notably, these phenomena are not mutually exclusive but will in real life occur conjointly, implying that the neural responses in these brain areas are probably a "mixed bag", which makes it difficult to identify which activity is related to which psychological phenomenon. Not surprisingly, then, the available evidence to address these distinctions has produced inconclusive results. For instance, correlations with trait and state measures of empathic concern and personal distress have been extremely volatile. While some early studies demonstrate that AI activation correlates with empathic concern, though only when using rather liberal statistical thresholds (e.g. Lamm et al., 2007a; Singer et al., 2004, 2006), others have failed to replicate this (see review in Lamm et al., 2011), or report correlations for personal distress only (Cheetham et al., 2009). Moreover, perspective taking manipulations aimed at selectively amplifying feelings of personal distress or empathic concern resulted either in comparable responses to the two in the anterior insula but a stronger response in middle insula for personal distress (Lamm et al., 2007a), or stronger responses in the anterior insula and the MCC with higher personal distress (Jackson et al., 2006). More recently however, an interventional study has demonstrated differences in responses between participants who had trained to "empathize with" the suffering of others (without regulating their negative affect) and those who had trained to "show compassion for" them (with affect regulation) (Klimecki et al., 2014). While the former type of training probably resulted in empathic responses in the sense of "feeling as" and increased personal distress, the latter likely engaged processes related to "feeling for" and empathic concern to a stronger extent. Interestingly, differences in positive and negative affect and distinct neural activation patterns are in line with these predictions. Empathy training resulted in higher negative affect and engaged AI and MCC in those subdivisions meta-analytically identified and referred to throughout the present paper as related to empathy. Compassion training in contrast resulted in relatively higher positive affect and increased activation in areas associated with positive affect. This would therefore speak for an interpretation of the activation in AI and MCC that is consistently found in studies on empathy as reflecting "feeling as", which is in line with a shared representation account. Nevertheless, from the perspective of the suffering person, the "positive affect" response induced by compassion might be the better option, as compassion strongly motivates helping behavior while empathy in some cases doesn't (see also below, and Zanon et al., 2014, for a recent elegant demonstration using a life-threatening virtual reality scenario, showing that stronger engagement of AI results in less helping behavior).

Summing up, the empirical phenomenon of shared neural activations during direct and vicarious emotion experiences is a very clear and robust one. What is much less clear is its functional interpretation, with conceptual issues and methodological

and empirical limitations obstructing a clearer view. Future studies, including both theoretical approaches and new empirical directions, some of which we have outlined above, are therefore needed to enable a better integration of neural and conceptual–psychological levels of description.

### 3.2. What role do mirror neurons play in empathy?

In public talks or discussions with the media on the social neuroscience of empathy the term mirror neurons almost inevitably comes up – mostly referring to the presumed role of these neurons as the elementary building blocks or precondition of empathy. Mirror neurons are certainly among the most fascinating neuroscientific discoveries of the last decades. The finding in the beginning of the 90s (di Pellegrino et al., 1992; Gallese et al., 1996; Rizzolatti et al., 1996) that there are neurons in the macaque brain that selectively fire both when the monkey is executing an action and when it is merely observing its execution by others was groundbreaking: it showed for the first time that actions can be coded by individual neurons on a level that generalizes across motor and sensory domains and across different actors (see Ferrari and Rizzolatti, 2014 for review and historical account). As such it provided a common representational space for actions of one self and of others, and thus a fascinating possible neural implementation of "making sense of" observed actions by internally simulating them. According to influential views, automatically mirroring others' movements with our mirror neurons would allow us to subsequently retrieve the intentions that gave rise to them, thus forming the key to understanding what was going on in others' minds (e.g. Gallese and Goldman, 1998). Some years later, the first studies (as reviewed above) showing that processing one's own emotions and empathizing with those of others induced activation changes in overlapping areas, suggested that making sense of others' feelings also involves simulation processes.

It was tempting to conclude, then, that mirror neurons are the very reason why we can empathize with others – being the little work horses that pull the carriage of our empathic abilities. Moreover, the presence of mirror neurons in monkeys seemed to suggest that their function is "hard-wired", leading to suggestions that we are predetermined to resonate with the emotions of others "because of our mirror neurons". The prevalence of such assumptions in the public reception and media discourse has certainly been fueled by some rather uncritical popular science books (Bauer, 2006; Ramachandran, 2011), but also by early publications of scholars in the field of social neuroscience who linked the two phenomena quite liberally, but without much hard evidence. Yet, such a view is problematic in several ways: on the one hand, it lacks empirical support or even contradicts it; on the other, it has broad but misleading implications for our general understanding of empathy.

A first problem with the assumption that mirror neurons underlie empathy is that is lacks direct empirical support. A limited number of studies on mirror neurons have shown that certain types of neurons in the premotor and parietal areas in macaques respond to both the sight and the execution of (usually hand) actions; yet, there is no evidence that these neurons also play a role in evoking emotional responses in the monkey in response to the sight of emotional expressions in others. In addition, the findings of single-cell recordings in monkeys cannot be that easily transferred to humans, where we cannot perform such experiments. Instead, we need to rely on evidence from indirect and imprecise measures, including neuroimaging methods, studies combining TMS with motor evoked potentials (MEP), or electroencephalographic measures of sensorimotor "resonance". More recently, it could however be demonstrated using intracortical recordings that neurons with mirror neuron properties also seem to exist in humans (Mukamel et al., 2010). Surprisingly though, such neurons were not only found in "classical" motor areas, but also in e.g. the entorhinal and parahippocampal areas. Nevertheless, when studying healthy volunteers, we can only speak of common activation on the level of brain areas or aggregate electrical signals, not on the level of individual neurons. As a result, the term "mirror neurons" should be used with caution if applied to humans. More importantly, it needs to be clear that they are a "motor" phenomenon by their very definition.

Even if one assumes that mirror neurons are present in humans and are also involved in coding emotional expressions, ascribing a causal role in higher-order "interpretative" functions to them, whether it is understanding intentions or affective states, is problematic. Although mirror neurons have been shown to code very specific features of actions, some on the level of kinematics, some on a broader goal level, this does not necessarily imply that these neurons are involved in "copying" these aspects from the observed behavior by directly transforming visual input into motor code (as the term "mirroring" seems to suggest). In fact, such bottom-up "mirroring" of goal properties is implausible since there is no one-to-one mapping of perceived movements and the goals associated with them. In general, it seems unlikely that mirror neuron responses serve as input to contribute to high-level intention understanding (Csibra, 2008; Hickok, 2009; Uithol et al., 2011). In fact, more recent accounts of their function assume a reverse process: that observed actions are "interpreted" in other brain areas, and that mirror neuron responses constitute a top-down generated, predictive simulation that is used to facilitate ongoing perception (Wilson and Knoblich, 2005) – and thus *reflects* rather than contributes to action understanding (Csibra, 2008). Given these considerations, the view that mirror neurons enable us to understand others' feelings in a bottom-up fashion seems untenable, too.

This does not mean though that motor resonance processes cannot have a role in empathy. As outlined above, different pathways and mechanisms can trigger emotion resonance and the observation of actions that have emotional consequences for the actor is certainly one of them. For instance, as already shown by early work (Jackson et al., 2005), seeing others touching a hot stove or cutting their finger with a kitchen knife activates brain areas related to affective processing, including the MCC and AI. Importantly, however, these stimuli *also* activated parts of the inferior parietal cortex and ventral premotor cortex that would classically be assigned to the "mirror neuron system" (see Lamm et al., 2011, for recent meta-analytic integration). It therefore seems plausible that the "action observation" aspect of these paradigms resulted in (sensori-)motor resonance, which in turn might have facilitated emotion resonance. However, it is important to stress that this does not mean that motor resonance alone can "explain" or is synonymous with the emotion resonance. Rather, it might be a starting point that interacts with and needs to be supplemented by additional mechanisms, foremost affective resonance.

Further research on the links between motor resonance and empathic responses indicates that motor resonance does not seem to be a necessary condition for empathy. That is, empathy often occurs without the involvement of brain areas associated with mirror neurons in monkeys. For one, there are many situations in which empathic responses are evoked, without an action or even a person being perceived. For instance, simply reading in a novel or newspaper about the joy or plight of others, may elicit strong empathic sentiments. Although one could argue that these responses might be caused by imagining the other person's body by way of motor resonance processes, the available evidence indicates otherwise: for instance, learning that another person is in pain by means of an abstract visual cue (such as in Singer et al.'s seminal approach) rather than a pictorial presentation, does not activate "motor resonance areas", but areas related to mentalizing and theory of mind

(again, see Lamm et al., 2011, for a meta-analysis). Of course, findings based on fMRI cannot be seen as conclusive in this regard, but even if motor resonance is shown with seemingly more direct measures, such as suppression of mu rhythm in EEG signal over central areas, this does not necessarily reflect motor mirroring but may instead represent coding of other properties, e.g. tactile features, of observed actions (Coll et al., 2014).

Further support that automatic motor resonance is not a prerequisite for empathy comes from the arguments presented above that there are obviously many routes to trigger empathy. This clearly challenges the (naïve, yet in the general public widely held) assumption that mirror neurons are prerequisites for empathy. Research on clinical populations also speaks against this assumption, including the recently highlighted distinction between the propensity and the ability for empathy, shown in individuals with psychopathy who did not activate empathy "automatically", but were able to activate empathic responses when explicitly instructed to do so (Meffert et al., 2013). This is inconsistent with a view of empathy being dependent on bottom-up signaling from mirror neurons. Moreover, patients with congenital insensitivity to pain who lack the action-to-pain associations present in healthy persons are nevertheless able to experience empathic responses in response to the sight of actions which are painful for the actors. This demonstrates, obviously, that empathy can also be generated by mentalizing or associate learning mechanisms, rather than being solely dependent on motor resonance (Danziger et al., 2006, 2009).

In addition to not being necessary for empathy, "mirror neurons" do not seem to be sufficient for accurate empathy either. There are several cases in which relying on automatic motor resonance processes would lead to inaccurate empathic responses, such as when being exposed to people whose sensorimotor affective mapping differs from our own. We have addressed this aspect in a series of experiments in which we exposed participants to situations that would be painful for themselves, but are in fact not painful for the observed other. The consistent finding of these studies is that neural responses related to action observation are relatively unaffected by this manipulation (probably indicating that automatic motor resonance are relatively robust to such manipulations) while emotional responses are strongly modified, suggesting that mechanisms other than motor resonance mediate the empathic response and its regulation (Lamm et al., 2007b, 2010; Perry et al., 2010).

Taken together, both theoretical considerations and empirical evidence suggest that empathy neither requires, nor can be exhaustively explained by a "mirror neuron" faculty. In our opinion it is crucial to take a more judicious viewpoint here – both within the scientific community and in communication with the popular media. Not only is the assumption that mirror neurons are the underpinnings of empathy an incorrect one; the implications from such a view are also undesirable. For instance, possibly due to the well-defined properties of mirror neurons studied in macaques, it is often presumed that mirror neurons are hard-wired in their sensorimotor couplings, and that responses of mirror neurons are induced in an automatic, inflexible fashion. These presumptions are incorrect if applied to mirror neurons (which seem to be highly malleable by learning processes, see Cook et al., 2014; Heyes, 2010, for recent in-depth review) and therefore lead to equally false assertions if applied to the construct of empathy – for example, that we have a biologically hard-wired predisposition to automatically respond empathically to others. While the view that there are strong evolutionary roots and forces of automaticity in empathic responding certainly is valid (see also de Waal, 2008; Decety et al., 2012; Decety and Svetlova, 2012 for recent comparative and developmental reviews), it seems more likely that the automaticity and apparent predisposition for empathic resonance can be attributed to (early) learning experiences, culture and socialization rather than to hard-wired mirroring. This argument is also supported by a plethora of studies documenting the strong malleability of empathy by a variety of situational factors, including the relationship between people, the situational context, or the appraisal of a situation (e.g. Engen and Singer, 2013; Hein and Singer, 2008 for reviews focusing on modulation of empathy).

In sum, use of the term "mirroring" might be helpful as a loose analogy for the process of "reproducing" the affective experiences of others in our own emotion-related neural systems. However, we need to be aware that this is not the same as presuming that empathic abilities are causally linked to mirror neurons. Not only is this view empirically incorrect; it might also have broader, negative scientific and practical implications for our understanding of empathy.

### 3.3. Does increasing empathy make us "better people"?

In the public but at times also in the academic discourse, it appears to be taken for granted that empathy can act as a remedy or a stronghold against anti-social phenomena which seem to affect our society to an increasing extent – such as the selfish greed in the financial industry supposedly contributing to the global financial crisis, or the many armed conflicts we are witnessing these days, ranging from Syria over the Ukraine to Gaza. For instance, US-president Barack Obama has repeatedly spoken of an empathy deficit in our modern society, and stated that an "empathy crisis" may be at the root of the economic and political crises we are experiencing (2006, June 19). Such views have certainly been influenced by the folk intuition that empathy motivates prosocial behavior, such as helping others in need. Indeed, this intuition has received widespread support from social psychology (see Batson, 1991), as well as more recently from the field of social neuroscience. For instance, Hein et al. (2010) demonstrated that individual differences in altruistic behavior (taking over painful shocks from another person) were predicted by activation of empathy-related neural responses in left anterior insula (see also Hein et al., 2011; Masten et al., 2011; Mathur et al., 2010). At first glance, such a link between empathy and altruism might imply that increasing empathy in our society will reduce egoism and selfishness and the social conflicts associated with them (Rifkin, 2010).

However, such propositions overlook the fact that empathy is sensitive to deeply-rooted parochialism and ingroup bias (see Chiao and Mathur, 2010). This implies that it will motivate altruistic action in a way that prefers to help or cooperate with persons and groups that we perceive as closer or more similar to us. For instance, evidence for ethnicity[1] bias – a phenomenon characterizing the more negative perception of or behavior toward members of an ethnic outgroup – abounds in the social psychological literature. Recently, the fields of social and of cultural neuroscience have begun to document and understand the neurobiological bases of such bias in the domain of empathy. For instance, there is substantial and consistent evidence stemming from a variety of experimental approaches and neuroscientific methods that humans show reduced neural responses to pain being inflicted on ethnic outgroup members (e.g. Avenanti et al., 2010; Azevedo et al., 2013; Contreras-Huerta et al., 2013; Gutsell and Inzlicht, 2012; Riečanský et al., EPub ahead of print; Shamay-Tsoory et al., 2013; Sheng and Han, 2012; Sheng et al., 2014; Xu et al., 2009). Apart from ethnicity, factors such as one's attitude toward the other person, or

[1] Note that although the term "racial" has been mostly used in previous work, we prefer to use the term "ethnicity" as a more accurate and appropriate description of the phenomenon we are dealing with – i.e., differences between individuals in socio-cultural and physical, but not in biological-genetic terms. Moreover, this also intends to avoid the deterministic connotations of the term race, a concept that is still used in a derogatory way and to justify exploitative treatment of members of "inferior races" (AAPA, 1996).

whether one is in a competitive vs. cooperative relationship with her, also seem sufficient to cause an almost complete blockage of empathic responses and can even cause anti-social responses such as counter-empathy and Schadenfreude (Decety et al., 2010; Singer et al., 2006; Takahashi et al., 2009; Yamada et al., 2011).

It is important to note that these empathy biases also directly influence biases in altruistic behavior. For instance, both the studies of Hein et al. (2010) and of Mathur et al. (2010) cited above demonstrate higher altruistic helping for ingroup members, and this was motivated by different aspects of empathy. More specifically, football fans were more likely to share the pain of fans of their team, as compared to fans of their "enemy" team (Hein et al., 2010), and this difference in empathy as measured by activation differences in the anterior insula predicted a higher proportion of painful shocks taken over from fellow team fans. Similarly, members of different ethnic groups rather supported members of their own group than of other groups, and this was linked to higher activation of medial prefrontal cortical areas, which the authors interpreted as higher cognitive empathy (Mathur et al., 2010; in line with our discussion above, though, we suggest to avoid this term in favor of explaining the group differences as linked to a higher degree of mentalizing or perspective taking).

Interestingly, biased prosocial behavior can also be induced by subtle manipulations of interpersonal perception and connection. We have recently documented that the simple act of thinking about the mental states of another person predicts biased decisions during subsequent fictitious moral dilemmas (Majdandzic et al., 2012). More specifically, persons whose mental states had been reflected upon were sacrificed less often in scenarios requiring to sacrifice their life to prevent casualties in a greater number of others. This decision bias was therefore acting against the moral principles of utilitarianism, which proposes that one ought to act in a way that maximizes the net social welfare (West, 2013), as well as against the moral principle to treat all people equally (McKerlie, 2013). These moral decisions could be explained by a higher degree of connectedness to and "humanization" of the persons whose mental states one had previously considered, as compared to persons who had not been mentalized with. On a neural level, decisions concerning humanized persons were associated with higher activation in brain structures related to mentalizing, empathy/affect sharing, and emotion regulation. The fact that a higher degree of vicarious emotions caused a higher degree of sparing the humanized person was also confirmed by an independent behavioral experiment. It therefore seems that the biased decisions were triggered by processes related to both empathy and affective sharing, as well as to more cognitive aspects such as taking the other person's perspective which might have induced a higher degree of empathy.

The latter study also highlights another important aspect in discussing the links between empathy and morality. That is, some moral rules, like the principle not to harm others, might oppose other ones, like the principle of treating all people equally, or of maximizing the ratio of benefit and harm for all people, as in utilitarianism. Empathic sentiments toward certain persons making it less likely for someone to harm them might therefore, under certain circumstances, lead to amoral decisions with respect to other principles, and inflict "collateral" damage on other individuals or groups of people (see also Decety and Cowell, 2014; Ugazio et al., 2014, for a more extensive discussion of this aspect). This also suggests that persons with lower state or trait empathic concern will more easily adhere to the "rational" moral principle of utilitarianism and of treating all people equally, which was indeed confirmed by a recent behavioral study (Gleichgerrcht and Young, 2013). Interestingly, these findings might be mediated by individual differences in alexithymia, i.e. the inability to identify and describe one's own emotions (Patil and Silani, 2014); hence, such biased responding might be overcome by inducing a higher degree of emotional arousal (Patil et al., 2014).

Thus, increases in empathy do not necessarily make us behave more "morally", nor does moral behavior seem to require empathic responses as a motivation or precondition (Proctor, 2005). A further illustration of the latter idea comes from findings showing that although moral dilemmas often activate areas related to affective processing (Greene and Haidt, 2002), moral decisions based on a sense of justice recruit a markedly distinct network involving areas involved in higher-order cognition (e.g. the dorsolateral prefrontal cortex; Yoder and Decety, 2014). This further confirms the notion that moral decision making can operate outside the influence of empathy.

Finally, we need to consider that people may be able to empathize with others (in the sense of being able to feel what they are feeling, or "feeling as") and still harm them. The "tools of empathy" or knowledge about them at times may even be deliberately exploited to inflict harm in others, for instance in persons with psychopathic personality traits. Only recently, a series of social neuroscience studies has added to our knowledge of the psychopathic mind and how he or she is able to engage in such a-moral or a-social behavior. Interestingly, these studies suggest that psychopaths seem to show a lower *propensity* for empathy (in the sense of affect sharing), yet are able to feel what others are feeling when explicitly *instructed* to do so – though the exact way in which this instructing should be devised is still somewhat controversial, with different studies yielding somewhat different results (Decety et al., 2013a, 2013b; Keysers and Gazzola, 2014b for review; Lockwood et al., 2013a, 2014; Meffert et al., 2013; Pfabigan et al., 2014). Anecdotal evidence also suggests that the capacity to fully (and possibly empathically) sense the effects of one's actions, but to deliberately modify one's behavioral response to it can be exploited to do harm rather than to increase the welfare of others. This is illustrated in a horrendous way by case of Norwegian mass murderer Anders Breivik, who allegedly stated that "he was fully capable of empathy but had used a "meditation technique" to override his feelings" (Honigsbaum, 2013). Moreover, adolescents with aggressive conduct disorder seem to produce vicarious distress responses to the suffering of others (Decety et al., 2009; but see Lockwood et al., 2013b for conflicting results) suggesting that they might sense the distress of others but nevertheless engage in antisocial behavior. This is possibly due to the development of effective strategies to down-regulate these responses under certain circumstances, or, in more extreme cases, due to the pleasurable effects of doing harm (Baumeister and Campbell, 1999). Unfortunately, we do not have to go all the way to psychopathy to illustrate how empathy can be exploited for one's own and not for the greater good. For instance, we probably all know competitive situations, such as in sports, in which team tactics exploit our knowledge of how our opponents will feel and act in response to certain actions (such as the "psychological warfare" that might be applied during penalties shots in football), or conflicts with friends or loved ones in which our enhanced ability to empathize with them may provide us with all the more effective tools to hurt their feelings. Conversely, the potentially undesired consequences of upregulating one's empathic "gut" sentiments are illustrated by reports of "American mothers to feel sorry" for Boston Marathon bomber Dzhokhar Tsarnaev, allegedly because of pictures showing him with a "cute" and innocent face (Bloom, 2013).

Summing up, the available social psychological and social neuroscience evidence suggests that social decisions and behaviors driven by empathy will often be parochially biased, and that observations of these behavioral biases are paralleled by biases in the neurobiological processes thought to underlie empathy. Parochialism also seems to have strong phylogenetic roots, which motivate us to prefer and show maximum care for close kin, such as our own

offspring (Decety, 2011). As a consequence, initiatives simplistically aimed at a generalized increase of empathic sensitivity will likely not promote a more impartial society. Rather, it will likely replace egoism by its twin brother: an ingroup-favoring type of altruism – thereby widening rather than diminishing the boundaries between social groups.

Instead of training people to blindly rely on an amplification of empathy, attempts at enhancing impartiality of prosocial attitudes and actions in our society need to be tailored to include individuals that fall outside of our preferred social groups. This is indeed the approach on which many ancient spiritual techniques have built up on. For instance, Buddhist *Mettā* (Loving Kindness) meditation is aimed at overcoming our deeply rooted parochial preferences by using specific mental exercises akin to perspective taking, in order to help us expand our compassion to all sentient beings, including our adversaries. The field of contemplative neuroscience has recently begun to understand the neural and biological mechanisms of such approaches, with promising first results indicating that expanding our circle of "close others" not only has positive effects on how we perceive and interact with others, but also on our own well-being and affect (Klimecki et al., 2013, 2014; Leiberg et al., 2011; Weng et al., 2013). This might, then, be the trick president Obama has been looking for: a win-win situation that improves not only social welfare, but also our own wellbeing, by making us act in a more compassionate way toward all beings, including ourselves.

## 4. Conclusions

The intention of our review was to highlight some key issues relevant to our understanding of the phenomenon of empathy and the neural mechanisms supporting this complex and important social skill. The significance of these issues partly stems from the fact that they are intimately related to empathy and as such are keenly discussed in the public discourse, but at the same time due to their complexity might seriously obscure our insights into this topic. For instance, an overly enthusiastic tendency to interpret shared neural activations in a certain functional direction, to causally link empathy to our presumed mirror neuron system, or to assume that a move toward relying onto our empathic "gut" sentiments will make our society more prosocial, all seem to be popular outcomes of our increased scientific knowledge on empathy but might actually make us drift away from the facts. Therefore we would like to encourage researchers to more explicitly separate neural and conceptual–psychological levels of descriptions in their discussions of empathy, in order to avoid misinterpretation of the concept. Also, researchers as well as science communicators should take efforts to clearly articulate the interpretational limitations of their empirical work. And furthermore, care should be taken to apply concepts related to empathy more distinctively and appropriately (e.g., "affective", "motor", and "cognitive" empathy).

The aim of this review and commentary was to provide a foundation on which the field of social neuroscience and the interested public alike can critically evaluate the current understanding of empathy, and by which new directions may be inspired. These directions, include, for instance, the development of more rigorous experimental paradigms, (causal) research methods and theoretical models to (1) substantiate the notion of shared neural activations accounts and assess how they are causally related to behavioral manifestations of empathy; (2) clarify how motor resonance shapes and predicts affective responses and to what extent this enables affective understanding; (3) investigate the specificity of emotion sharing and its consequences by combining and cross-classifying different emotions within the same paradigm and (4) examine clinical populations with specific deficits. After all, the field of social neuroscience and in particular the investigation of empathy is still very young, and the latter has not even reached its adolescence yet. Having had such a promising and eager childhood, though, it seem reasonable to expect that with further aging, we will develop more elaborate theoretical models and more rigorous empirical tools to ultimately gain more conclusive insights.

## Acknowledgment

We gratefully acknowledge funding from the Vienna Science and Technology Fund (WWTF, CS11-005 and CS11-016).

## References

American Association of Physical Anthropologists, 1996. AAPA statement on biological aspects of race. Am. J. Phys. Anthropol. 101, 569–570.
Avenanti, A., Bueti, D., Galati, G., Aglioti, S.M., 2005. Transcranial magnetic stimulation highlights the sensorimotor side of empathy for pain. Nat. Neurosci. 8, 955–960.
Avenanti, A., Sirigu, A., Aglioti, S.M., 2010. Racial bias reduces empathic sensorimotor resonance with other-race pain. Curr. Biol. 20, 1018–1022.
Azevedo, R.T., Macaluso, E., Avenanti, A., Santangelo, V., Cazzato, V., Aglioti, S.M., 2013. Their pain is not our pain: brain and autonomic correlates of empathic resonance with the pain of same and different race individuals. Hum. Brain Mapp. 34, 3168–3181.
Batson, C.D., 1991. The Altruism Question: Toward a Social Psychological Answer. Lawrence Erlbaum Associates, Hillsdale, NJ.
Batson, C.D., 2011. These things called empathy. In: Decety, J., Ickes, W. (Eds.), The Social Neuroscience of Empathy. The MIT Press, Cambridge, MA, pp. 3–16.
Batson, C.D., Fultz, J., Schoenrade, P.A., 1987. Distress and empathy: two qualitatively distinct vicarious emotions with different motivational consequences. J. Pers. 55, 19–39.
Bauer, J., 2006. Warum ich fühle, was du fühlst: Intuitive Kommunikation und das Geheimnis der Spiegelneurone. Heyne, München.
Baumeister, R.F., Campbell, W.K., 1999. The intrinsic appeal of evil: sadism, sensational thrills, and threatened egotism. Pers. Soc. Psychol. Rev. 3, 210–221.
Bernhardt, B.C., Singer, T., 2012. The neural basis of empathy. Annu. Rev. Neurosci. 35, 1–23.
Bloom, P., 2013. Feeling sorry for Tsarnaev. The New Yorker, Retrieved from http://www.newyorker.com/news/news-desk/feeling-sorry-for-tsarnaev
Bufalari, I., Aprile, T., Avenanti, A., Di Russo, F., Aglioti, S.M., 2007. Empathy for pain and touch in the human somatosensory cortex. Cereb. Cortex 17, 2553–2561.
Bufalari, I., Ionta, S., 2013. The social and personality neuroscience of empathy for pain and touch. Front. Hum. Neurosci. 7, 393.
Catmur, C., Mars, R.B., Rushworth, M.F., Heyes, C., 2011. Making mirrors: premotor cortex stimulation enhances mirror and counter-mirror motor facilitation. J. Cogn. Neurosci. 23, 2352–2362.
Cheetham, M., Pedroni, A.F., Antley, A., Slater, M., Jancke, L., 2009. Virtual milgram: empathic concern or personal distress? Evidence from functional MRI and dispositional measures. Front. Hum. Neurosci. 3, 29.
Cheng, Y., Lin, C.P., Liu, H.L., Hsu, Y.Y., Lim, K.E., Hung, D., Decety, J., 2007. Expertise modulates the perception of pain in others. Curr. Biol. 17, 1708–1713.
Chiao, J.Y., Mathur, V.A., 2010. Intergroup empathy: how does race affect empathic neural responses? Curr. Biol. 20, R478–R480.
Coll, M.-P., Bird, G., Catmur, C., Press, C., 2014. Crossmodal repetition effects in the mu rhythm indicate tactile mirroring during action observation. Cortex (Epub ahead of print).
Contreras-Huerta, L.S., Baker, K.S., Reynolds, K.J., Batalha, L., Cunnington, R., 2013. Racial bias in neural empathic responses to pain. PLOS ONE 8, e84001.
Cook, R., Bird, G., Catmur, C., Press, C., Heyes, C., 2014. Mirror neurons: from origin to function. Behav. Brain Sci. 37, 177–192.
Corradi-Dell'Acqua, C., Hofstetter, C., Vuilleumier, P., 2011. Felt and seen pain evoke the same local patterns of cortical activity in insular and cingulate cortex. J. Neurosci. 31, 17996–18006.
Craig, A.D., 2009. How do you feel – now? The anterior insula and human awareness. Nat. Rev. Neurosci. 10, 59–70.
Csibra, G., 2008. Action mirroring and action understanding: an alternative account. In: Haggard, P., Rosetti, Y., Kawato, M. (Eds.), Sensorimotor Foundations of Higher Cognition. Attention & Performance XXII. Oxford University Press, Oxford, pp. 435–459.
Danziger, N., Faillenot, I., Peyron, R., 2009. Can we share a pain we never felt? Neural correlates of empathy in patients with congenital insensitivity to pain. Neuron 61, 203–212.
Danziger, N., Prkachin, K.M., Willer, J.C., 2006. Is pain the price of empathy? The perception of others' pain in patients with congenital insensitivity to pain. Brain 129, 2494–2507.
de Waal, F.B., 2008. Putting the altruism back into altruism: the evolution of empathy. Annu. Rev. Psychol. 59, 279–300.
Decety, J., 2011. The neuroevolution of empathy. Ann. N. Y. Acad. Sci. 1231, 35–45.
Decety, J., Chen, C., Harenski, C., Kiehl, K.A., 2013a. An fMRI study of affective perspective taking in individuals with psychopathy: imagining another in pain does not evoke empathy. Front. Hum. Neurosci. 7, 489.

Decety, J., Cowell, J.M., 2014. The complex relation between morality and empathy. Trends Cogn. Sci. 18, 337–339.
Decety, J., Echols, S., Correll, J., 2010. The Blame Game: the effect of responsibility and social stigma on empathy for pain. J. Cogn. Neurosci. 22, 985–997.
Decety, J., Jackson, P.L., 2004. The functional architecture of human empathy. Behav. Cogn. Neurosci. Rev. 3, 71–100.
Decety, J., Lamm, C., 2006. Human empathy through the lens of social neuroscience. Sci. World J. 6, 1146–1163.
Decety, J., Michalska, K.J., Akitsuki, Y., Lahey, B.B., 2009. Atypical empathic responses in adolescents with aggressive conduct disorder: a functional MRI investigation. Biol. Psychol. 80, 203–211.
Decety, J., Norman, G.J., Berntson, G.G., Cacioppo, J.T., 2012. A neurobehavioral evolutionary perspective on the mechanisms underlying empathy. Prog. Neurobiol. 98, 38–48.
Decety, J., Skelly, L.R., Kiehl, K.A., 2013b. Brain response to empathy-eliciting scenarios involving pain in incarcerated individuals with psychopathy. JAMA Psychiatry 70, 638–645.
Decety, J., Svetlova, M., 2012. Putting together phylogenetic and ontogenetic perspectives on empathy. Dev. Cogn. Neurosci. 2, 1–24.
Derbyshire, S.W.G., 2000. Exploring the pain "neuromatrix". Curr. Rev. Pain 4, 467–477.
di Pellegrino, G., Fadiga, L., Fogassi, L., Gallese, V., Rizzolatti, G., 1992. Understanding motor events: a neurophysiological study. Exp. Brain Res. 91, 176–180.
Engen, H.G., Singer, T., 2013. Empathy circuits. Curr. Opin. Neurobiol. 23, 275–282.
Fan, Y., Duncan, N.W., de Greck, M., Northoff, G., 2011. Is there a core neural network in empathy? An fMRI based quantitative meta-analysis. Neurosci. Biobehav. Rev. 35, 903–911.
Ferrari, P.F., Rizzolatti, G., 2014. Mirror neuron research: the past and the future. Philos. Trans. R. Soc. Lond. B: Biol. Sci. 369, 20130169.
Gallese, V., Fadiga, L., Fogassi, L., Rizzolatti, G., 1996. Action recognition in the premotor cortex. Brain 119 (Pt 2), 593–609.
Gallese, V., Goldman, A., 1998. Mirror neurons and the simulation theory of mind-reading. Trends Cogn. Sci. 2, 493–501.
Gleichgerrcht, E., Young, L., 2013. Low levels of empathic concern predict utilitarian moral judgment. PLOS ONE 8, e60418.
Goldman, A.I., Sebanz, N., 2005. Simulation, mirroring, and a different argument from error. Trends Cogn. Sci. 9, 320, author reply 321.
Gonzalez-Liencres, C., Shamay-Tsoory, S.G., Brune, M., 2013. Towards a neuroscience of empathy: ontogeny, phylogeny, brain mechanisms, context and psychopathology. Neurosci. Biobehav. Rev. 37, 1537–1548.
Greene, J., Haidt, J., 2002. How (and where) does moral judgment work? Trends Cogn. Sci. 6, 517–523.
Grill-Spector, K., Malach, R., 2001. fMR-adaptation: a tool for studying the functional properties of human cortical neurons. Acta Psychol. 107, 293–321.
Gutsell, J.N., Inzlicht, M., 2012. Intergroup differences in the sharing of emotive states: neural evidence of an empathy gap. Soc. Cogn. Affect. Neurosci. 7, 596–603.
Hein, G., Lamm, C., Brodbeck, C., Singer, T., 2011. Skin conductance response to the pain of others predicts later costly helping. PLoS ONE 6, e22759.
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.
Hein, G., Singer, T., 2008. I feel how you feel but not always: the empathic brain and its modulation. Curr. Opin. Neurobiol. 18, 153–158.
Hetu, S., Taschereau-Dumouchel, V., Jackson, P.L., 2012. Stimulating the brain to study social interactions and empathy. Brain Stimul. 5, 95–102.
Heyes, C., 2010. Where do mirror neurons come from? Neurosci. Biobehav. Rev. 34, 575–583.
Hickok, G., 2009. Eight problems for the mirror neuron theory of action understanding in monkeys and humans. J. Cogn. Neurosci. 21, 1229–1243.
Hillis, A.E., 2014. Inability to empathize: brain lesions that disrupt sharing and understanding another's emotions. Brain 137, 981–997.
Honigsbaum, M., 2013. Barack Obama and the 'empathy deficit'. The Observer, Retrieved from http://www.theguardian.com/science/2013/jan/04/barack-obama-empathy-deficit
Hutchison, W.D., Davis, K.D., Lozano, A.M., Tasker, R.R., Dostrovsky, J.O., 1999. Pain-related neurons in the human cingulate cortex. Nat. Neurosci. 2, 403–405.
Jackson, P., Brunet, E., Meltzoff, A., Decety, J., 2006. Empathy examined through the neural mechanisms involved in imagining how I feel versus how you feel pain. Neuropsychologia 44, 752–761.
Jackson, P., Meltzoff, A., Decety, J., 2005. How do we perceive the pain of others? A window into the neural processes involved in empathy. Neuroimage 24, 771–779.
Keysers, C., Gazzola, V., 2014a. Hebbian learning and predictive mirror neurons for actions, sensations and emotions. Philos. Trans. R. Soc. Lond. B: Biol. Sci. 369 (1644), 20130175.
Keysers, C., Gazzola, V., 2014b. Dissociating the ability and propensity for empathy. Trends Cogn. Sci. 18, 163–166.
Keysers, C., Kaas, J.H., Gazzola, V., 2010. Somatosensation in social perception. Nat. Rev. Neurosci. 11, 417–428.
Klimecki, O.M., Leiberg, S., Lamm, C., Singer, T., 2013. Functional neural plasticity and associated changes in positive affect after compassion training. Cereb. Cortex 23, 1552–1561.
Klimecki, O.M., Leiberg, S., Ricard, M., Singer, T., 2014. Differential pattern of functional brain plasticity after compassion and empathy training. Soc. Cogn. Affect. Neurosci. 9, 873–879.
Lamm, C., Batson, C.D., Decety, J., 2007a. The neural substrate of human empathy: effects of perspective-taking and cognitive appraisal. J. Cogn. Neurosci. 19, 42–58.
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.
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. J. Cogn. Neurosci. 22, 362–376.
Lamm, C., Nusbaum, H.C., Meltzoff, A.N., Decety, J., 2007b. 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.
Lamm, C., Silani, G., Singer, T., 2014. Distinct Neural Networks Underlying Empathy for Pleasant and Unpleasant Touch (submitted for publication).
Lamm, C., Singer, T., 2010. The role of anterior insular cortex in social emotions. Brain Struct. Funct. 214, 579–591.
Leiberg, S., Klimecki, O., Singer, T., 2011. Short-term compassion training increases prosocial behavior in a newly developed prosocial game. PLoS ONE 6, e17798.
Leigh, R., Oishi, K., Hsu, J., Lindquist, M., Gottesman, R.F., Jarso, S., Crainiceanu, C., Mori, S., Hillis, A.E., 2013. Acute lesions that impair affective empathy. Brain 136, 2539–2549.
Lev-Ran, S., Shamay-Tsoory, S.G., Zangen, A., Levkovitz, Y., 2012. Transcranial magnetic stimulation of the ventromedial prefrontal cortex impairs theory of mind learning. Eur. Psychiatry 27, 285–289.
Lindquist, K.A., Barrett, L.F., 2012. A functional architecture of the human brain: emerging insights from the science of emotion. Trends Cogn. Sci. 16, 533–540.
Lockwood, P.L., Bird, G., Bridge, M., Viding, E., 2013a. Dissecting empathy: high levels of psychopathic and autistic traits are characterized by difficulties in different social information processing domains. Front. Hum. Neurosci. 7, 760.
Lockwood, P.L., Seara-Cardoso, A., Viding, E., 2014. Emotion regulation moderates the association between empathy and prosocial behavior. PLOS ONE 9, e96555.
Lockwood, Patricia, L., Sebastian, Catherine, L., McCrory, Eamon, J., Hyde, Zoe, H., Gu, X., De Brito, Stéphane, A., Viding, E., 2013b. Association of callous traits with reduced neural response to others' pain in children with conduct problems. Curr. Biol. 23, 901–905.
Logothetis, N.K., 2008. What we can do and what we cannot do with fMRI. Nature 453, 869–878.
Majdandzic, J., Bauer, H., Windischberger, C., Moser, E., Engl, E., Lamm, C., 2012. The human factor: behavioral and neural correlates of humanized perception in moral decision making. PLoS ONE 7, e47698.
Masten, C.L., Morelli, S.A., Eisenberger, N.I., 2011. An fMRI investigation of empathy for 'social pain' and subsequent prosocial behavior. Neuroimage 55, 381–388.
Mathur, V.A., Harada, T., Lipke, T., Chiao, J.Y., 2010. Neural basis of extraordinary empathy and altruistic motivation. Neuroimage 51, 1468–1475.
McKerlie, D., 2013. Egalitarianism, International Encyclopedia of Ethics. Blackwell Publishing Ltd., Oxford, UK.
Medford, N., Critchley, H.D., 2010. Conjoint activity of anterior insular and anterior cingulate cortex: awareness and response. Brain Struct. Funct. 214, 535–549.
Meffert, H., Gazzola, V., den Boer, J.A., Bartels, A.A., Keysers, C., 2013. Reduced spontaneous but relatively normal deliberate vicarious representations in psychopathy. Brain 136, 2550–2562.
Mitchell, J.P., 2009. Inferences about mental states. Philos. Trans. R. Soc. Lond. B: Biol. Sci. 364, 1309–1316.
Morelli, S.A., Rameson, L.T., Lieberman, M.D., 2014. The neural components of empathy: predicting daily prosocial behavior. Soc. Cogn. Affect. Neurosci. 9, 39–47.
Morrison, I., Lloyd, D., di Pellegrino, G., Roberts, N., 2004. Vicarious responses to pain in anterior cingulate cortex: is empathy a multisensory issue? Cogn. Affect. Behav. Neurosci. 4, 270–278.
Mukamel, R., Ekstrom, A.D., Kaplan, J., Iacoboni, M., Fried, I., 2010. Single-neuron responses in humans during execution and observation of actions. Curr. Biol. 20, 750–756.
Obama Challenges Grads to Cultivate Empathy., 2006, June 19. Northwestern University News, Retrieved from http://www.northwestern.edu/newscenter/stories/2006/06/barack.html
Patil, I., Cogoni, C., Zangrando, N., Chittaro, L., Silani, G., 2014. Affective basis of judgment-behavior discrepancy in virtual experiences of moral dilemmas. Soc. Neurosci. 9, 94–107.
Patil, I., Silani, G., 2014. Reduced empathic concern leads to utilitarian moral judgments in trait alexithymia. Front. Psychol. 5, 501.
Perry, A., Bentin, S., Bartal, I.B., Lamm, C., Decety, J., 2010. "Feeling" the pain of those who are different from us: modulation of EEG in the mu/alpha range. Cogn. Affect. Behav. Neurosci. 10, 493–504.
Perry, D., Hendler, T., Shamay-Tsoory, S.G., 2012. Can we share the joy of others? Empathic neural responses to distress vs joy. Soc. Cogn. Affect. Neurosci. 7, 909–916.
Pfabigan, D.M., Seidel, E.M., Wucherer, A.M., Keckeis, K., Derntl, B., Lamm, C., 2014. Affective empathy differs in male violent offenders with high- and low-trait psychopathy. J. Pers. Disord. 28, 1–20.
Preston, S.D., de Waal, F.B.M., 2002. Empathy: its ultimate and proximate bases. Behav. Brain Sci. 25, 1–72.
Proctor, J., 2005. Frans de Waal and Evan Thompson interviewed by Jim Proctor. Primates, monks and the mind. The case of empathy. J. Conscious. Stud. 12 (7), 38–54.
Ramachandran, V.S., 2011. The Tell-Tale Brain: A Neuroscientist's Quest for What Makes Us Human. W.W. Norton, New York.

Riečanský, I., Paul, N., Kölble, S., Stieger, S., Lamm, C., 2014. Beta Oscillations Reveal Ethnicity Ingroup Bias in Sensorimotor Resonance to Pain of Others (EPub ahead of print).
Rifkin, J., 2010. The Empathic Civilization: The Race to Global Consciousness in a World in Crisis. Jeremy P. Tarcher Inc., Los Angeles.
Rizzolatti, G., Fadiga, L., Gallese, V., Fogassi, L., 1996. Premotor cortex and the recognition of motor actions. Brain Res. Cogn. Brain Res. 3, 131–141.
Santiesteban, I., Banissy, M.J., Catmur, C., Bird, G., 2012. Enhancing social ability by stimulating right temporoparietal junction. Curr. Biol. 22, 2274–2277.
Sebastian, C.L., Fontaine, N.M., Bird, G., Blakemore, S.J., Brito, S.A., McCrory, E.J., Viding, E., 2012. Neural processing associated with cognitive and affective Theory of Mind in adolescents and adults. Soc. Cogn. Affect. Neurosci. 7, 53–63.
Shamay-Tsoory, S.G., 2011. The neural bases for empathy. Neuroscientist 17, 18–24.
Shamay-Tsoory, S.G., Abu-Akel, A., Palgi, S., Sulieman, R., Fischer-Shofty, M., Levkovitz, Y., Decety, J., 2013. Giving peace a chance: oxytocin increases empathy to pain in the context of the Israeli-Palestinian conflict. Psychoneuroendocrinology 38, 3139–3144.
Sheng, F., Han, S., 2012. Manipulations of cognitive strategies and intergroup relationships reduce the racial bias in empathic neural responses. Neuroimage 61, 786–797.
Sheng, F., Liu, Q., Li, H., Fang, F., Han, S., 2014. Task modulations of racial bias in neural responses to others' suffering. Neuroimage 88, 263–270.
Silani, G., Lamm, C., Ruff, C.C., Singer, T., 2013. Right supramarginal gyrus is crucial to overcome emotional egocentricity bias in social judgments. J. Neurosci. 33, 15466–15476.
Singer, T., 2006. The neuronal basis and ontogeny of empathy and mind reading: review of literature and implications for future research. Neurosci. Biobehav. Rev. 30, 855–863.
Singer, T., Lamm, C., 2009. The social neuroscience of empathy. Ann. N. Y. Acad. Sci. 1156, 81–96.
Singer, T., Seymour, B., O'Doherty, J., Kaube, H., Dolan, R.J., Frith, C.D., 2004. Empathy for pain involves the affective but not the sensory components of pain. Science 303, 1157–1161.
Singer, T., Seymour, B., O'Doherty, J.P., Stephan, K.E., Dolan, R.D., Frith, C.D., 2006. Empathic neural responses are modulated by the perceived fairness of others. Nature 439, 466–469.
Steinbeis, N., Bernhardt, B.C., Singer, T., 2014. Age-related differences in function and structure of rSMG and reduced functional connectivity with DLPFC explains heightened emotional egocentricity bias in childhood. Soc. Cogn. Affect. Neurosci. (Epub ahead of print).
Takahashi, H., Kato, M., Matsuura, M., Mobbs, D., Suhara, T., Okubo, Y., 2009. When your gain is my pain and your pain is my gain: neural correlates of envy and schadenfreude. Science 323, 937–939.
Tomova, L., von Dawans, B., Heinrichs, M., Silani, G., Lamm, C., 2014. Is stress affecting our ability to tune into others? Evidence for gender differences in the effects of stress on self-other distinction. Psychoneuroendocrinology 43, 95–104.
Ugazio, G., Majdandzic, J., Lamm, C., 2014. Are empathy and morality linked? Insights from moral psychology, social and decision neuroscience, and philosophy. In: Maibom, H. (Ed.), Empathy in Morality. Oxford University Press, Oxford, pp. 155–171.
Uithol, S., van Rooij, I., Bekkering, H., Haselager, P., 2011. What do mirror neurons mirror? Philos. Psychol. 24, 607–623.
Valentini, E., Koch, K., 2012. Fine-grained analysis of shared neural circuits between perceived and observed pain: implications for the study of empathy for pain. J. Neurophysiol. 108, 1805–1807.
Vogt, B.A., 2005. Pain and emotion interactions in subregions of the cingulate gyrus. Nat. Rev. Neurosci. 6, 533–544.
Weng, H.Y., Fox, A.S., Shackman, A.J., Stodola, D.E., Caldwell, J.Z., Olson, M.C., Rogers, G.M., Davidson, R.J., 2013. Compassion training alters altruism and neural responses to suffering. Psychol. Sci. 24, 1171–1180.
West, H.R., 2013. Utilitarianism, International Encyclopedia of Ethics. Blackwell Publishing Ltd., Oxford UK.
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, 655–664.
Wilson, M., Knoblich, G., 2005. The case for motor involvement in perceiving conspecifics. Psychol. Bull. 131 (3), 460–473.
Xu, X., Zuo, X., Wang, X., Han, S., 2009. Do you feel my pain? Racial group membership modulates empathic neural responses. J. Neurosci. 29, 8525–8529.
Yamada, M., Lamm, C., Decety, J., 2011. Pleasing frowns, disappointing smiles: an ERP investigation of counterempathy. Emotion 11, 1336–1345.
Yoder, K., Decety, J., 2014. The good, the bad, and the just: justice sensitivity predicts neural responses neural response during moral evaluation of actions performed by others. J. Neurosci. 34 (12), 4161–4166.
Zaki, J., Ochsner, K., 2012. The neuroscience of empathy: progress, pitfalls and promise. Nat. Neurosci. 15, 675–680.
Zanon, M., Novembre, G., Zangrando, N., Chittaro, L., Silani, G., 2014. Brain activity and prosocial behavior in a simulated life-threatening situation. Neuroimage 98, 134–146.