

# What (If Anything) Is Shared in Pain Empathy? A Critical Discussion of De Vignemont and Jacob's Theory of the Neural Substrate of Pain Empathy

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In a recent article in *Philosophy of Science*, De Vignemont and Jacob defend the view that empathy involves interpersonal similarity between an empathizer and a target person with respect to internal affective states. Focusing on empathy for pain, they propose a theory of the neural substrate of pain empathy. We point out several flaws in their interpretation of the data and argue that currently available data do not differentiate between De Vignemont and Jacob's model and alternative models. Finally, we offer some suggestions about how this might be achieved in future research.

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In recent years, controversy has erupted in the philosophy of mind, developmental psychology, and cognitive neuroscience concerning how best to conceptualize empathy. Much of this controversy centers on the issue of how to articulate the common intuition that empathy involves the sharing of emotional experiences. On one side of the debate, researchers (e.g., Eisenberg and Fabes 1990; Decety and Jackson 2004; De Vignemont and Singer 2006; Batson 2009; Keysers, Kaas, and Gazzola 2010; De Vignemont and Jacob 2012) maintain that empathy involves interpersonal similarity between an empathizer and a target person with respect to their internal affective

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states. On the other side, another group of researchers (Goldie 1999; Gallagher 2012) have pointed out that emotional experiences are usually directed toward intentional objects and proposed that in empathy one person engages with a target person's experience and thereby comes to have an experience with the same intentional object.<sup>1</sup> On this latter view, empathy involves the sharing of the intentional component of emotional experience rather than the sharing of internal affective states. Still other theorists (e.g., Zahavi 2011; Zahavi and Overgaard 2012) deny that empathy involves the sharing of emotional experiences and, thus, do not postulate any kind of interpersonal similarity at all.

One distinct advantage of the approach taken by De Vignemont and colleagues is that their model endeavors to make sense of recent findings in neuroscience and is therefore constrained by the relevant empirical data. In order to do so, they home in on a specific type of empathy, namely, empathy for pain, which is particularly well researched in the empirical literature. We embrace this aspect of their approach—bearing in mind, of course, that it will be important to exercise great caution in extrapolating from a detailed study of pain empathy to other types of empathy or to empathy in general. But we also point out several flaws in their interpretation of the data they refer to and also criticize their overly selective review of data relevant to the debate. We hope that our criticism can contribute to the further refinement of their model of empathy as well as the alternatives and to the development of experimental methods of distinguishing among these competing theoretical options.

De Vignemont and Jacob (2012) refer to the common view that the experience of pain derives from the processing and integration of nociceptive inputs and complex emotional and cognitive processes, implicating the participation of several pain-specific brain structures that may be functionally distinct. The neural network involved in pain processing is often referred to as the 'pain matrix', the primary components of which are sometimes said to be a sensory-discriminative and an affective-motivational network (e.g., Singer et al. 2004; Aydede 2006). On this view, primary and secondary somatosensory and posterior insular cortices are thought to serve the processing of sensory-discriminative features of pain stimuli, such as location, duration, and stimulus intensity. In the affective-motivational domain, anterior cingulate and anterior insular cortices are thought to mediate these aspects of pain processing, for example, the unpleasantness of pain.

In their review, De Vignemont and Jacob (2012) suggest that these two components can be dissociated and that this provides a basis for dis-

1. There are important differences among these proposals. But for present purposes, the only feature of their proposals that is relevant is the claim that empathy involves interpersonal similarity with respect to intentional structure rather than with respect to internal affective states. Snow (2000) requires both kinds of interpersonal similarity.

tinguishing pain empathy from the phenomenon of contagious pain. Specifically, they suggest that contagious pain is more likely to recruit the sensory-discriminative component, whereas empathy is more likely to recruit the affective component of the pain matrix. In accordance, they argue that empathy is ‘other-centered’ insofar as it involves a concern for the other person’s affective state, whereas contagion is ‘self-centered’. In support of this position, they refer to research suggesting that in pain empathy the affective neural components are selectively activated (Singer et al. 2004; Botvinick et al. 2005). They further propose a mechanism explaining how exactly pain empathy is presumed to occur. When one person perceives another person receiving a painful stimulus (or a cue anticipating a painful stimulus), De Vignemont and colleagues thus argue that the affective component of the observer’s pain matrix is likely to activate. The observer’s brain then generates the expectation that there is a painful stimulus impinging upon her body and that the sensory-discriminative component of her pain matrix will therefore be active (because this would normally be the case when the affective part of her pain matrix is active). But, of course, this is not the case, and so the observer makes the judgment that it is not her but the other person who is experiencing pain.

In the following, we express four separate concerns with this proposal. The first is conceptual. Specifically, it is not clear to us what the final inference in the procedure proposed by De Vignemont and Jacob is supposed to add: after all, the procedure begins with the observer perceiving or otherwise resolving the information that someone else is in pain. So why does the observer need to register that there is no activation in the sensory-discriminative component of her own pain matrix in order to infer that it is the other person who is in pain? Indeed, given that background information about the person receiving the painful stimulation modulates the activation of the affective component of the observer’s pain matrix (as reviewed in Singer and Lamm [2009]), it seems that the ‘other-centeredness’ is there from the start. Hence, it is just as likely that the understanding that it is the other who is in pain precedes the activation of the pain matrix rather than being generated by it.<sup>2</sup>

Second, why should specifically the affective component but not the sensory component be involved in empathy? De Vignemont and Jacob’s (2012) idea seems to be that since empathy is other-centered it should not involve representations of one’s own body—which is presumably what

2. Given that participants in the experiments discussed by De Vignemont and Jacob are always aware that it is others’ bodies, and not their own, that are being subjected to painful stimulation, it is in fact also unclear how the results bear upon contagious pain at all. Indeed, De Vignemont and Jacob agree (307 and elsewhere) that contagion does not involve ascription of the painful experience to the other person.

activation of the sensory-discriminative component of the pain matrix would reflect. But it would then seem to follow from this that the affective component should not be activated either, since one's affective representations are, after all, also general representations of one's own affective state. In fact, if empathy involves imagining the experience (including thoughts, feelings, and sensations) of the target person, it is not clear that the sensory aspect should be any less relevant than the affective component. In other words, if there can be vicarious affective representations, why not also vicarious sensory representations? And indeed, there is research suggesting that the sensory component is also active under some circumstances when one observes others in pain (Keyesers et al. 2010). The difference between these results and the results of Singer et al. (2004) and Botvinick et al. (2005) may be explained by the fact that the former involved participants actually observing other people receiving painful stimuli to specific parts of their bodies, whereas the latter involved observers receiving cues that another person was receiving a painful stimulus. In other words, in the latter cases, the observers did not actually see the relevant body parts, so it is no surprise that somatotopically organized bodily representations play less of a role in these cases. Moreover, taking a sidelong glance to the developmental literature, Roth-Hanania, Davidov, and Zahn-Waxler (2011) conducted a longitudinal study in which they found that 8-month-olds who touched the part of their own body corresponding to the body part of another person whom they observed to be in pain were subsequently, at 16 months, more likely to engage in prosocial behavior toward someone in pain than 16-month-olds who had not shown this response at 8 months. This suggests that a somatotopical sensory representation of an observed painful experience plays some role in generating an other-centered representation of the experience.

Third, De Vignemont and Jacob fail to provide adequate justification for the claim that the sensory component of pain is predominant in contagious pain. We are aware of no evidence that contagion involves the preferential activation of the sensory-discriminative component of pain. Moreover, it seems clear that other contagious affective states, such as contagious fear and contagious sadness, involve an affective experience of the emotion one takes on (and presumably not any particular sensory-discriminative component). So why should the situation be reversed in contagious pain?

Fourth, it is currently unclear to what extent activation of the pain matrix is specifically related to pain phenomena. The notion of pain as emerging from the integrated activity of specific and dissociable modules (e.g., sensory vs. affective components of the pain matrix) has been questioned in light of recent neuroscientific findings. Indeed, the concept of the 'pain matrix' has been criticized on the grounds that the different brain structures gathered together under this label in fact make up a functional catch-

all that is not specific to pain at all but, rather, to stimulus salience regardless of sensory modality (e.g., Iannetti and Mouraux 2010). Loud sounds, strong nonpainful vibrations, and sudden visual inputs have been shown to induce overlapping activations in the anterior cingulate cortex and anterior insula, among other brain areas (Mouraux et al. 2011), suggesting that this neural network may be involved in bottom-up attentional mechanisms induced by any stimulus that may represent a threat for the individual (Legrain et al. 2011).

Thus, the very idea of the pain matrix, upon which De Vignemont and Jacob's account is based, can be called into question. Indeed, the alternative interpretation of the data in terms of the registration of salience would actually fit well with the idea that empathy involves taking the perspective of another person and engaging with the intentional object(s) of their experience. For if Jim is in pain, the painful stimulus will be salient to him, and if Sue takes Jim's perspective and engages with his experience, then the painful stimulus will also become salient to Sue. And just as Jim is likely to be attending to it, Sue will be likely to direct her attention to it, too. Hence, these data fit just as well with the alternative model proposed by Goldie and Gallagher, according to which empathy involves one person engaging with another person's emotional experience and thereby coming to have an experience with the same intentional object. On this latter view, what is shared in empathy is the intentional component of affective experience rather than any internal affective states.

Clearly, in order to resolve these issues, it will first of all be necessary to isolate pain-specific neural activity. And in fact some recent results may point in a fruitful direction. An investigation using multivariate decoding techniques identified a neurological signature of physical pain (Wager et al. 2013), providing empirical evidence that physical pain experience can be predicted by the overall pattern of activity within a specific and distributed neural network, at least in part overlapping with the classic pain matrix. The pattern recognition model in that experiment successfully predicted subjective pain ratings in a different group of participants under similar conditions and under the administration of an analgesic opioid drug, but very interestingly not under a social pain condition related to a recent romantic breakup. Indeed, although both physical and social pain induced activation of similar brain areas, the same algorithm failed in predicting the experienced social pain. These results underscore the importance of caution in the interpretation of neural activity in the so-called pain matrix. Importantly, they suggest that pain phenomena are not reflected by activity in any one area, and the level of activation of the same brain region cannot be considered a sufficient proof of similar functions or brain mechanisms. On the contrary, similarity in the activation profile of a brain region in different circumstances may hide subtly different activity within that region's sub-

populations. Thus, understanding the brain mechanisms underpinning pain, as well as any other sensory or cognitive process, including empathy, requires going beyond the localization of function to any one area or network of areas and actually describing their functional interactions and computational mechanisms.

And indeed, a detailed neural model of pain could help in adjudicating among the competing models of empathy. For in order to determine whether empathy in general, or pain empathy in particular, involves interpersonal similarity of internal affective states, of intentional objects, or of nothing at all, it would be very useful to be able to distinguish the processes underpinning the affective components of experiences such as pain from the processes underpinning the intentional structure of those experiences (such as the modulation of attention). Once this is achieved, a subsequent step could be to investigate which, if any, of these components are shared. This is not to say that neuroscientific techniques and computational modeling are the only tools that will be useful in making progress toward understanding empathy. Indeed, we suspect that behavioral techniques may be especially useful in investigating the extent to which the contents of thought, or intentional objects, are shared by a person experiencing pain and a person who learns of this and empathizes, since it is plausible that the neural differences between distinct thoughts may be highly subtle and the intersubject variability quite large.

Although we have identified several flaws in the model put forth by De Vignemont and Jacob (2012), we must emphasize that we support their efforts to link up the philosophical discussion with ongoing neuroscientific research and hope that this brief commentary contributes a further step in this direction. Clearly, determining an optimal framework for understanding empathy remains an interpretative challenge, requiring further attention from both neuroscientific and philosophical perspectives.

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