

“Feeling” the pain of those who are different from us: Modulation of EEG in the mu/alpha range

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We explored how apparently painful stimuli and the ability to identify with the person on whom the pain is inflicted modulate EEG suppression in the mu/alpha range (8–12 Hz). In a 2×2 design, we presented pictures of hands either experiencing needle pricks or being touched by a Q-tip. In the *dissimilar-other* condition, the hand was assigned to a patient suffering from a neurological disease in which Q-tips inflicted pain, whereas needle pricks did not. In the *similar-other* condition, the hand was assigned to a patient who responded to stimulation in the same way as the healthy participant. Participants were instructed to imagine the feeling of the person whose hand was shown and to evaluate his or her affective state. Pain conditions elicited greater EEG suppression than did nonpain conditions, particularly over frontocentral regions. Moreover, an interaction between pain and similarity revealed that for similar others, the pain effect was significant, whereas in the dissimilar-other group, suppression was equally large in the pain and no-pain conditions. We conclude that mu/alpha suppression is elicited both automatically, by observing a situation that is potentially painful for the observer, and by empathy for pain, even if the other person is different from oneself.

Empathy, the ability to share the emotions of others, is a human aptitude intriguing philosophers, psychologists, sociologists, and neuroscientists alike. It is thought to have a key role in much of human social interaction and to be a proxy for motivating prosocial behavior (Decety & Michalska, 2010). Although it has been defined in many ways (e.g., Batson, 2009; Decety & Meyer, 2008; de Vignemont & Singer, 2006), here we will use the term *empathy* broadly, to describe an intersubjective induction process by which emotions are shared, while preserving the knowledge about their personal origin (Decety & Jackson, 2004; Decety & Meyer, 2008).

Much effort has been invested in the past decade in identifying the neural mechanisms that mediate empathic abilities. As part of this endeavor, an impressive body of behavioral and physiological studies has been marshaled under the perception–action coupling account of empathy (Preston & de Waal, 2002). The core

assumption of this account is that perceiving a target’s emotion and/or action activates automatically the corresponding representations of that emotion and/or action in the observer, which, in turn, activates somatic and autonomic responses. The mechanism of this process is not straightforwardly bottom-up, however. For example, Decety and Lamm (2006) proposed that empathy involves both emotion sharing (bottom-up information processing) and executive control, which modulates the shared affective experience (top-down information processing). According to this model, these bottom-up and top-down processes involve several specific and interacting neural networks (such as the anterior cingulate cortex [ACC] and the anterior insula). Feedback loops from the prefrontal cortex may play a crucial role in taking into account one’s own mental state and intentions in order to react (or not) to the affective states of others (Decety & Lamm, 2006).

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Similarly, other authors have emphasized that empathy is not an all-or-none process, suggesting that several factors can, jointly or separately, modulate empathic brain responses. For example, during the evaluation of an emotional cue, contextual appraisal may determine whether an empathic brain response is generated. Other salient top-down controlled factors that might affect empathy are attention, perspective taking, and the relationship between empathizer and agent (for reviews, see de Vignemont & Singer, 2006; Hein & Singer, 2008; Singer & Lamm, 2009). Particularly relevant to the present study is that all these factors might be important for regulating empathy for pain.

Pain is one of the feelings that we strongly experience in ourselves and to which we strongly react when perceived in others. A number of functional magnetic resonance imaging (fMRI) studies have compared the hemodynamic responses elicited by the perception or the imagination of others in painful situations versus neutral situations. These studies associated specific activation of a brain circuit including the ACC, insula, and somatosensory cortex with empathy for pain (Akitsuki & Decety, 2009; Benuzzi, Lui, Duzzi, Nichelli, & Porro, 2008; Botvinick et al., 2005; Cheng et al., 2007; Decety, Michalska, & Akitsuki, 2008; Gu & Han, 2007; Jackson, Brunet, Meltzoff, & Decety, 2006; Jackson, Meltzoff, & Decety, 2005; Lamm, Batson, & Decety, 2007; Lamm, Nusbaum, Meltzoff, & Decety, 2007; Moriguchi et al., 2007; Ogino et al., 2007; Saarela et al., 2007; Singer et al., 2004; Zaki, Ochsner, Hanelin, Wager, & Mackey, 2007). Other fMRI studies, however, have not revealed empathy-related activations in somatosensory regions (e.g., Jackson et al., 2005; Morrison, Lloyd, di Pellegrino, & Roberts, 2004; Singer et al., 2004).

Yet evidence for a coupling between observing pain inflicted in others and the response of the observer's somatosensory system has also been provided by studies examining more direct measures of neural activity. For example, recording electrophysiological manifestations of brain activity, Bufalari, Aprile, Avenanti, Di Russo, and Aglioti (2007) found that viewing video clips showing painful or not painful tactile stimuli delivered to others increased or decreased, respectively, the amplitude of an early component of the somatosensory evoked potential (P45), which is presumed to reflect activity in the primary somatosensory cortex (S1). Furthermore, transcranial magnetic stimulation (TMS) studies have demonstrated that the sensory and motor cortices are also modulated by observation of pain in others (Avenanti, Buetti, Galati, & Aglioti, 2005; Avenanti, Minio Paluello, Bufalari, & Aglioti, 2006). The latter results provide strong support for the implication of a sensory discriminative component of pain during empathy, as well as a motor component, which fits neatly with the perception-action account (for a review and account of the differences that exist between directly and vicariously experienced pain, see Jackson, Rainville, & Decety, 2006). The interaction between multiple neural networks involved in affective and cognitive components of empathy has been explored in the last few years. For example, Gu and Han (2007), using fMRI,

showed that neural correlates of empathic processes of pain can be altered by task demand and stimulus reality (cartoon vs. real pictures). Jackson, Brunet, et al. (2006) compared different perspectives of feeling pain and showed that the perspectives of both one's self and others were associated with activation in the neural network involved in pain processing; however, the self perspective yielded higher pain ratings and involved the pain matrix more extensively in the secondary somatosensory cortex, the ACC, and the insula, whereas adopting the perspective of the other was associated with specific increase in the posterior cingulate and the right temporo-parietal junction. These results showed the similarities between self and other pain representations but also highlighted some distinctiveness between these two representations, which may be what allows us to distinguish empathic responses to others from our own personal distress (Decety & Lamm, 2009).

Additional evidence for the involvement of a sensory-motor system in the perception of others' pain has been provided by electroencephalographic (EEG) and magnetoencephalographic (MEG) studies that have shown that watching painful situations, as compared with non-painful situations, suppressed EEG/MEG oscillations in the 10-Hz range on both sides of the Rolandic fissure (Cheng, Lee, et al., 2008; Cheng, Yang, Lin, Lee, & Decety, 2008; Mu, Fan, Mao, & Han, 2008). These oscillations have been labeled *mu* (Rolandic) rhythms (Kuhlman, 1978) when measured over somatomotor regions and have tentatively been distinguished from the *alpha* rhythms (Berger, 1929; see also, e.g., Goodman, Beatty, & Mulholland, 1980) that are measured primarily (but not exclusively) over the occipital cortex (for a review, see Klimesch, 1999).

The alpha frequency dominates the EEG when the brain rests (*idling rhythms*; e.g., Pfurtscheller, Stancak, & Neuper, 1996), and their (event-related) desynchronization (ERD) probably reflects enhancement of neural activity induced by a perceptual event, which leads to asynchronous neural firing. It should be noted, however, that, although in the same frequency range, the modulation of mu rhythms differs from that of the alpha waves not only on their presumably more anterior sources, but also on functional dimensions. Whereas alpha is modulated primarily by visual stimulation, mu rhythms are desynchronized and their power attenuated when motor activity is engaged in (Gastaut, 1952) and, crucially, also while actions executed by someone else are *observed* (Cochin, Barthelemy, & Martineau, 1998; Cochin, Barthelemy, Roux, & Martineau, 1999; Cohen-Seat, Gastaut, Faure, & Heuyer, 1954; Gastaut & Bert, 1954; Muthukumaraswamy, Johnson, & McNair, 2004). The visual-motor coupling suggested by this pattern led several investigators to suggest that it reflects a *resonance system*, which might be the biological basis for the simulation theory (see Pineda, 2005, for a review). In the last few years, several studies of typical participants have linked mu suppression to higher social information processing, including social skills (Oberman, Pineda, & Ramachandran, 2007), theory

of mind (Perry, Troje, & Bentin, 2010; Pineda & Hecht, 2009), and empathy (Cheng, Lee, et al., 2008; Cheng, Yang, et al., 2008). Moreover, abnormal mu suppression has been linked in several studies to autistic spectrum disorders when such participants viewed the actions of others (Martineau, Schmitz, Assaiante, Blanc, & Barthelemy, 2004; Oberman et al., 2005; Oberman, Ramachandran, & Pineda, 2008; but see Raymaekers, Wiersema, & Roeyers, 2009, and Yang-Teng, Decety, Chia-Yen, Lui, & Cheng, 2010, for nonreplication of this finding).

All of the studies above presented affective situations that are experienced in a similar way by the observer and the observed target (e.g., a hand being pricked, a foot being stepped on). However, we know from experience that it is possible to empathize with those who are different from us, such as people with different cultural backgrounds, animals, cartoon characters, and even artificial objects (Abell, Happé, & Frith, 2000; Heider & Simmel, 1944). Indeed, the roots of this tendency can be found very early in life (Hamlin, Wynn, & Bloom, 2007; Montgomery & Montgomery, 1999). A recent fMRI study indicated that inferring the affective state of someone who was different from the observer recruited neural structures that are also involved in inferring the affective states of someone who is similar to the observer (Lamm, Meltzoff, & Decety, 2010). Notably, these authors showed that empathy in a situation that was aversive only for the observer, but neutral for the target, also recruited areas involved in high-level appraisals of both one's own traits and affective states and those of others (dorsomedial prefrontal cortex), as well as areas involved in cognitive control (right inferior frontal cortex). These results suggest that inferring the affective state of someone who is not like us might rely on the same neural processes as inferring the affective states of someone who is similar to us. However, it has been shown that these tendencies may be overcome by executive functions (e.g., Lamm et al., 2010). Evidently, more work is needed to shed additional light on the mechanism enabling us to understand dissimilar others.

In the present study, we investigated the modulation of mu suppression in painful and nonpainful situations by the similarity between the observer and the target. Like Lamm et al. (2010), we presented situations in which different groups of targets experienced painful and nonpainful stimulation. Whereas the targets in one group (similar others) showed affective responses to stimulation similar to those of the participants themselves, the targets in the other group differed in their affective responses to the same object and were, therefore, defined as dissimilar others. This scenario was implemented by presenting pictures of hands experiencing needle pricks or being touched by a soft object (a Q-tip). One group of hands was described as belonging to patients who responded to these situations in the same way as the participants (feeling pain in response to needle pricks and no pain in response to touch), whereas members of the second group were described as patients who felt pain when touched by a Q-tip and no pain when pricked with a needle (see

Figure 1). The participants were instructed to imagine the feelings of the targets in order to share and evaluate their affective states. The valence of the shared feelings could therefore be either neutral (in the case of nonpainful stimulations) or negative (in the case of painful stimulations). We expected differences between EEG desynchronization in the 8- to 12-Hz range in response to the observed painful and nonpainful conditions over somatosensory areas (mu suppression). In addition, assuming a similarity effect (similar/dissimilar other), we also expected to find greater suppression when the participants viewed similar others (whom the participants could more easily identify with) than when they viewed dissimilar others. For situations that were aversive to the participants but not aversive to the targets, we expected stronger mu suppression, in line with Lamm et al.'s (2010) findings of stronger responses to such stimuli.

METHOD

Participants

Thirty right-handed healthy volunteers between 18 and 34 years of age participated in the study (age, $M = 21.5$ years, $SE = 4.5$; 18 women). All the participants gave informed written consent and were paid for participation. They reported no history of neurological, psychiatric, or major medical disorder and no current use of psychoactive medications. The study was approved by the local Ethics Committee (University of Chicago) and was conducted in accordance with the Declaration of Helsinki. The participants did not consume caffeine on the day of the experiment. One participant withdrew from the experiment in the middle, and 1 was excluded from the data analysis due to noisy data, resulting in a final sample size of 28 participants.

Stimuli, Task, and Design

The participants were presented with color photos of left hands being either pricked by a needle or touched with a Q-tip (Figure 1). As part of the design, the participants were informed that one group of hands belonged to patients who suffer from a rare neurological disease, which causes them to feel pain when touched by a soft object (such as a Q-tip), but to experience a touch-like sensation and no pain when being pricked by a needle (dissimilar others). The second group of hands belonged to patients described as suffering from a disease (tinnitus aurium) that was unrelated to their somatic sensation and nociception. Therefore, needle pricks caused pain, whereas touch by the Q-tip did not (similar others). Thus, the same stimuli could be perceived as either painful or not painful, depending on the target (similar or dissimilar other). The combination of stimulation and target groups therefore constituted a 2×2 factorial design (pain \times similarity).

The stimuli were presented in 48 blocks of 8 trials each, for a total of 384 trials. An instruction screen at the beginning of each block displayed the patient's face and the patient group he or she belonged to. The participants were informed that the situations they were about to see had been experienced by this patient, and they were instructed to vividly imagine the affect of the targets resulting from the displayed situations.

Each trial consisted of a hand photograph presented for 2 sec, followed by a fixation cross for another 2 sec. Each of the four (pain \times similarity) conditions was presented on 96 trials. In order to ensure that the participants were paying attention and complying with the task and to explore the participants' performance, a number of pseudorandomized trials (20% of the total trials) was followed by a response screen. Responses required either rating the amount of pain felt by the target on a visual analog scale (VAS) or performing

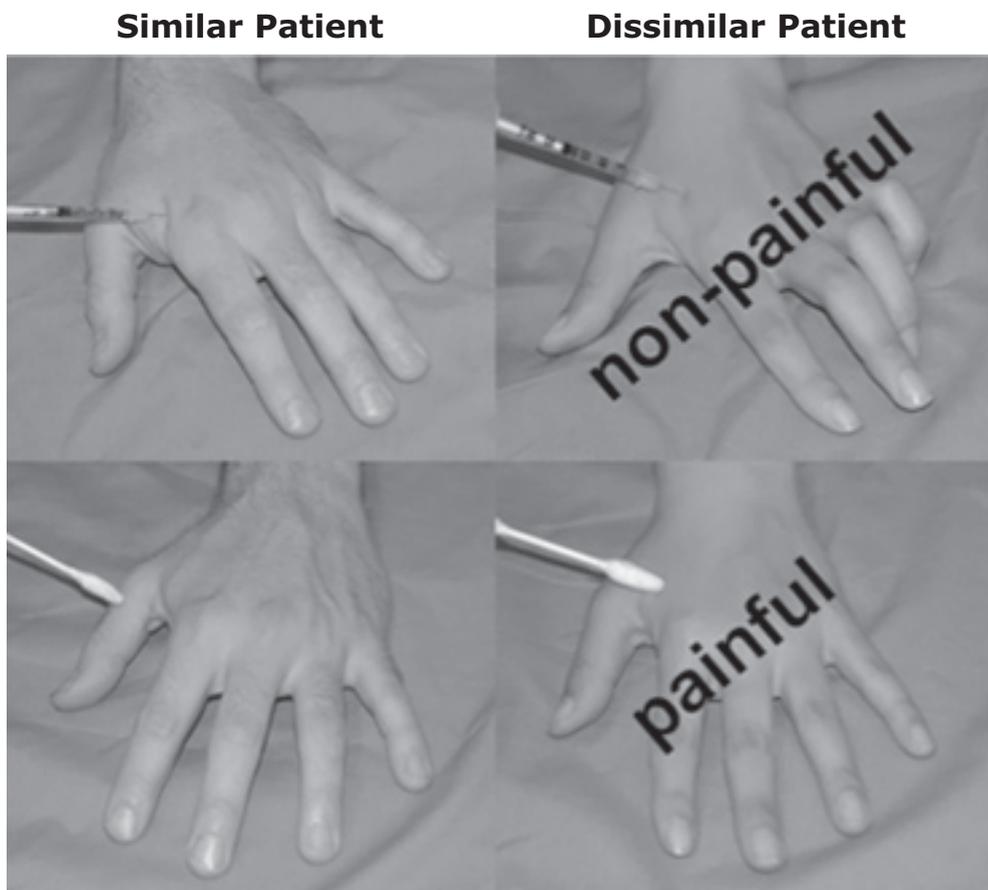


Figure 1. The stimuli presented in this experiment. Note the distinction between the similar-other and dissimilar-other conditions.

a forced choice matching task. The VAS scale was used to record subjective pain ratings reflecting the imagined pain felt by the target (with scores ranging from 0 = *no pain* to 100 = *very severe pain*). In the forced choice task, a face expression was presented, and the participants had to judge whether the expression (neutral or pained) matched the image displayed on the previous trial (e.g., a painful expression for a similar-other patient following a needle prick would be a match). The rationale for using two different response types, as well as response omissions, was to minimize response preparation during performance of the pain imagery task, such as preparing the VAS response before stimulus offset. The participants executed four practice blocks before beginning the actual experiment (see Figure 2).

Following the EEG experiment, emotional responses in the four experimental conditions were assessed using a procedure proposed by Batson, Early, and Salvarini (1997). The participants were shown one trial in each condition and rated the degree to which they experienced 20 emotional states while imagining the target's pain (e.g., alarmed, concerned, compassionate, distressed; 0 = *not at all*, 6 = *extremely*). Ratings of emotional states were aggregated by calculating indices of empathic concern and personal distress (see Batson et al., 1997, for details). These indices were analyzed using a repeated measures ANOVA with factors of similarity (similar other, dissimilar other), pain (pain, no pain), and emotion (personal distress and empathic concern). In addition, a sadness index (mean rating of feeling low-spirited, low, heavy-hearted, and sorrowful) was calculated and analyzed using a separate repeated measures ANOVA (factors: similarity and pain). Lastly, at the end of the ex-

periment, the participants were debriefed as to whether they had any doubts about whether the described neurological conditions actually exist.

Data Acquisition and Analysis

EEG recording. EEG signals were recorded using a Geodesic Sensor Net with 128 sensors and Net Station, Version 4.1.2, software (Electrical Geodesics, Eugene, OR). Electrode impedances were kept under 60 k Ω . Electrode Cz was used as the reference. The EEG was amplified (band-pass, 0.1–100 Hz) and digitized at a sampling rate of 250 Hz. Data were segmented into 2.5-sec epochs, starting at 500 msec before stimulus onset until stimulus offset.

EEG data processing. The EEG data were analyzed using Brain Vision Analyzer software (Brain Products; www.brainproducts.com) and house-made MATLAB routines. Raw EEG data were initially 0.5-Hz high-pass filtered (24 dB) to reduce DC drifts and were rereferenced offline to the average of the two mastoids. Eye movements were corrected using an ICA procedure (Jung et al., 2000). Remaining EEG artifacts exceeding $\pm 100 \mu\text{V}$ were detected, and the data during an epoch of 300 msec symmetrically encompassing the event were excluded from the analysis.

Mu/alpha suppression. To reduce the data to a manageable size, we spatially averaged the data recorded at three adjacent sites in eight regions of interest (ROIs). These regions were determined to cover left and right lateral sites along the anterior–posterior dimension. The ROIs were frontal–right (FR), frontal–left (FL), central–right (CR), central–left (CL), parietal–right (PR), parietal–left (PL), occipital–right (OR), and occipital–left (OL) sites (Figure 3). The

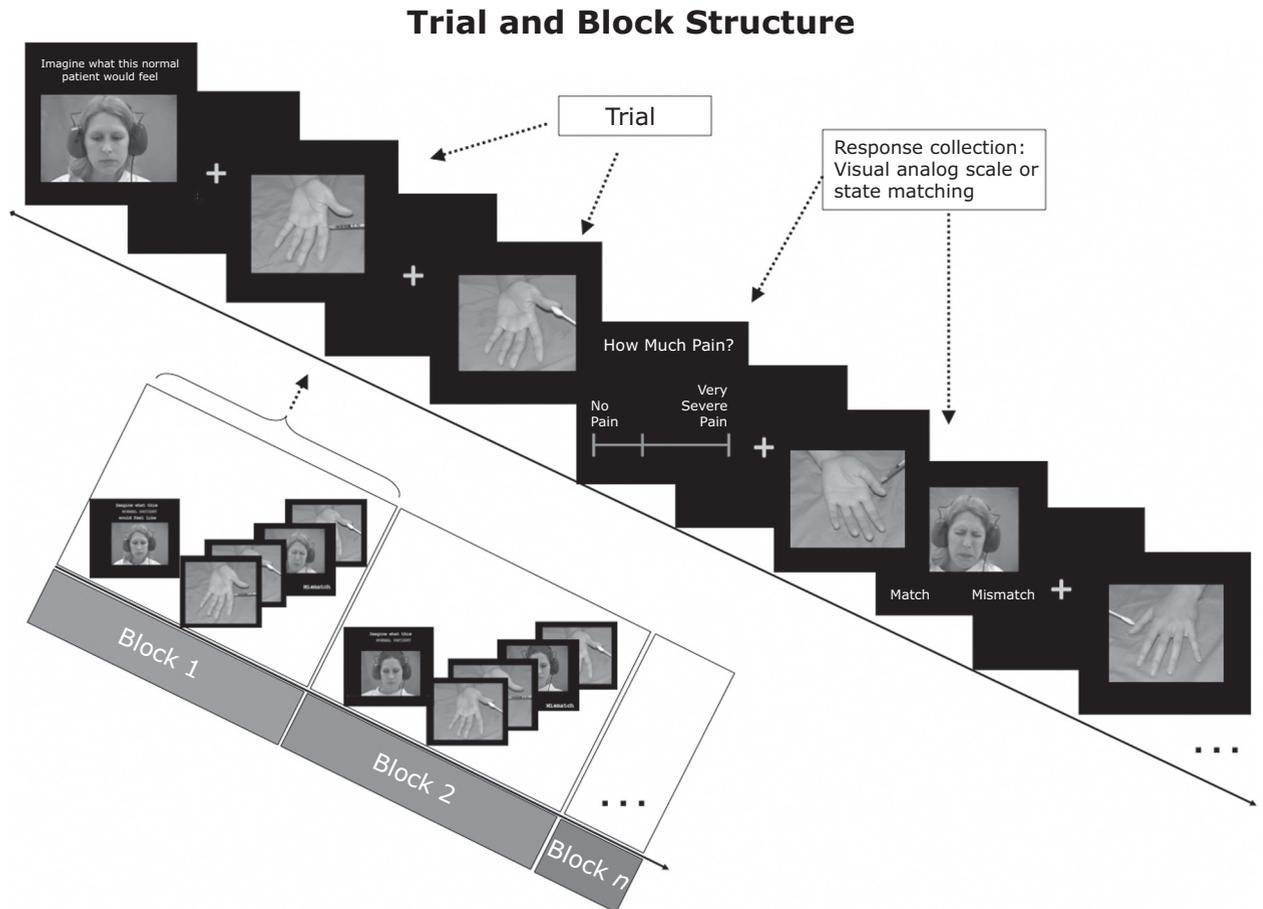


Figure 2. The sequence of events and the experimental design used in this study.

eccentricity of these regions was determined on the basis of prior mu suppression studies.

The EEG power in the 8- to 12-Hz frequency range during a period of 2 sec starting at the stimulus onset was then integrated using a fast Fourier transform performed at 0.5-Hz intervals (on the basis of 2,048 points per segment and using a Hanning window). A mu suppression index was calculated for each experimental condition. This index was the logarithm of the ratio of the EEG power while the participants watched a stimulus, relative to the EEG power during the 500 msec before stimulus onset while the participants fixated on a cross presented at the center of the screen. The ratio (as opposed to subtraction) was used in order to reduce variance across participants, such as that imposed by variability in absolute EEG power resulting from individual differences in, for example, scalp thickness and electrode impedance (Pineda & Oberman, 2006). Since ratio data are inherently not normally distributed as a result of lower bounding, a log transform was applied to the computed ratio data. Hence, a log ratio of less than zero indicates suppression in the EEG amplitude, a value of zero indicates no change, and values greater than zero indicate enhancement. This suppression index was computed separately for each of the eight regions described above.

RESULTS

Behavioral Results and Dispositional Measures

Pain ratings using the VAS were in line with the actual affective conditions of the target stimuli. The conditions

that were defined as painful for the patient were rated as more painful than those defined as painless [$M (SD)$ pain = 84 (5.3), no pain = 5 (3.2); $F(1,28) = 690.7$, $MS_e = 1,611$, $p < .0001$], with no difference between similar and dissimilar others and no interaction [$F < 1$; $F(1,28) = 2.7$, $p = .108$]. An analysis of response times (RTs) revealed that they were longer for rating dissimilar than for rating similar others [$M (SD)$ dissimilar = 3,542 msec (148), similar = 3,248 msec (141); $F(1,28) = 18.3$, $MS_e = 136,962$, $p < .0001$]. The RTs were similar for the pain and no-pain conditions, and there was no significant interaction between the two factors [$F < 1$; $F(1,28) = 2.9$, $p = .101$]. Although the interaction between pain and similarity was not significant either for rating scores or for RTs to these ratings, there was a trend in this direction. Therefore, to establish these trends and explore whether the evaluation of pain in the similar-other and dissimilar-other conditions was, indeed, similar, we compared the pain and no-pain conditions separately for the two types of targets. For similar-other targets, there was, as was expected, a robust effect of pain condition on pain rating [84.2 (13.7) and 3.8 (5.3) for pain and no pain, respectively; $F(1,28) = 656.7$, $p < .0001$] and also a trend for shorter RTs to pain than to no-pain stimuli

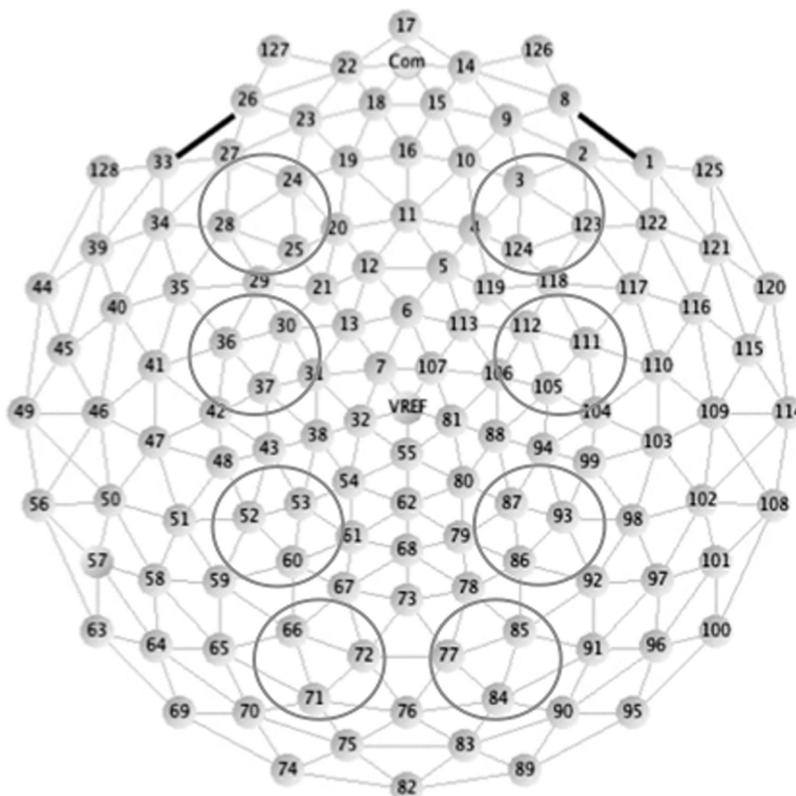


Figure 3. The dense head coverage of recording sites and the regions of interest selected for statistical analysis.

[$M(SD)$ pain = 3,115 msec (874), no pain = 3,382 msec (815); $F(1,28) = 3.8, p = .06$]. The effect of pain on pain rating was also significant for dissimilar-other targets, albeit the difference between pain and no-pain ratings was smaller [83.4 (13) and 7.11 (8.9) for pain and no pain, respectively; $F(1,28) = 520, p < .0001$], and there was no effect of pain on RTs [$M(SD)$ pain = 3,425 msec (752), no pain = 3,659 msec (1,128); $F(1,28) = 1.378, p = .25$].

Matching the facial expression associated with the emotional state of a patient to its actual affective state was also consistent with the predefined pain condition, and there was no significant effect of either pain or similarity and no interaction (all F s close to or lower than 1). However, the RTs were longer for dissimilar others than for similar others [1,728 msec (71), 1,583 msec (68); $F(1,28) = 23.3, MS_e = 25,942, p < .0001$]. The RTs were also longer for the nonpainful conditions than for the painful ones [1,696 msec (68), 1,615 msec (74); $F(1,28) = 4.5, MS_e = 41,663, p < .05$], with no significant interaction ($F < 1$).

The three-way ANOVA with the empathic concern and personal distress indices yielded significant main effects of both similarity and pain [$F(1,28) = 7.3, MS_e = 1.6, p < .05$; $F(1,28) = 96.5, MS_e = 2.8, p < .001$]. In addition, all two way-interactions were significant [index \times similarity, $F(1,28) = 4.7, MS_e = 0.194, p < .05$; index \times pain, $F(1,28) = 6.9, MS_e = 0.4, p < .05$; similarity \times

pain, $F(1,28) = 20.0, MS_e = 0.73, p < .001$]. These effects reflect the fact that the feelings of empathic concern and personal distress were in line with the valence of the targets' affective states. The significant similarity \times pain interaction showed, in addition, that distress and concern were higher when participants watched needle pricks of dissimilar others, as compared with the equally nonpainful stimulation with a Q-tip of similar others. Furthermore, a significant three-way interaction [index \times similarity \times pain, $F(1,28) = 17.3, MS_e = 0.31, p < .001$] indicated that this interaction effect was more pronounced for the personal distress index [mean values (SD): personal distress, similar/pain = 3.53 (1.48), similar/no pain = 0.35 (0.58), dissimilar/pain = 3.30 (1.52), dissimilar/no pain = 1.73 (1.08); empathic concern, similar/pain = 3.12 (1.38), similar/no pain = 0.97 (0.73), dissimilar/pain = 3.25 (1.18), dissimilar/no pain = 1.49 (0.96); see Figures 4A and 4B]. A separate analysis of experienced sadness revealed significant main effects of similarity and pain [$F(1,28) = 16.167, MS_e = 0.635, p < .001$; $F(1,28) = 36.467, MS_e = 1.438, p < .001$] and a nonsignificant similarity \times pain interaction [$F(1,28) < 1$]. This pattern reflects the fact that painful situations and watching dissimilar others triggered higher sadness [mean values (SD): sadness, similar/pain = 1.86 (1.37), similar/no pain = 0.41 (0.60), dissimilar/pain = 2.35 (1.60), dissimilar/no pain = 1.11 (0.90); see Figure 4C].

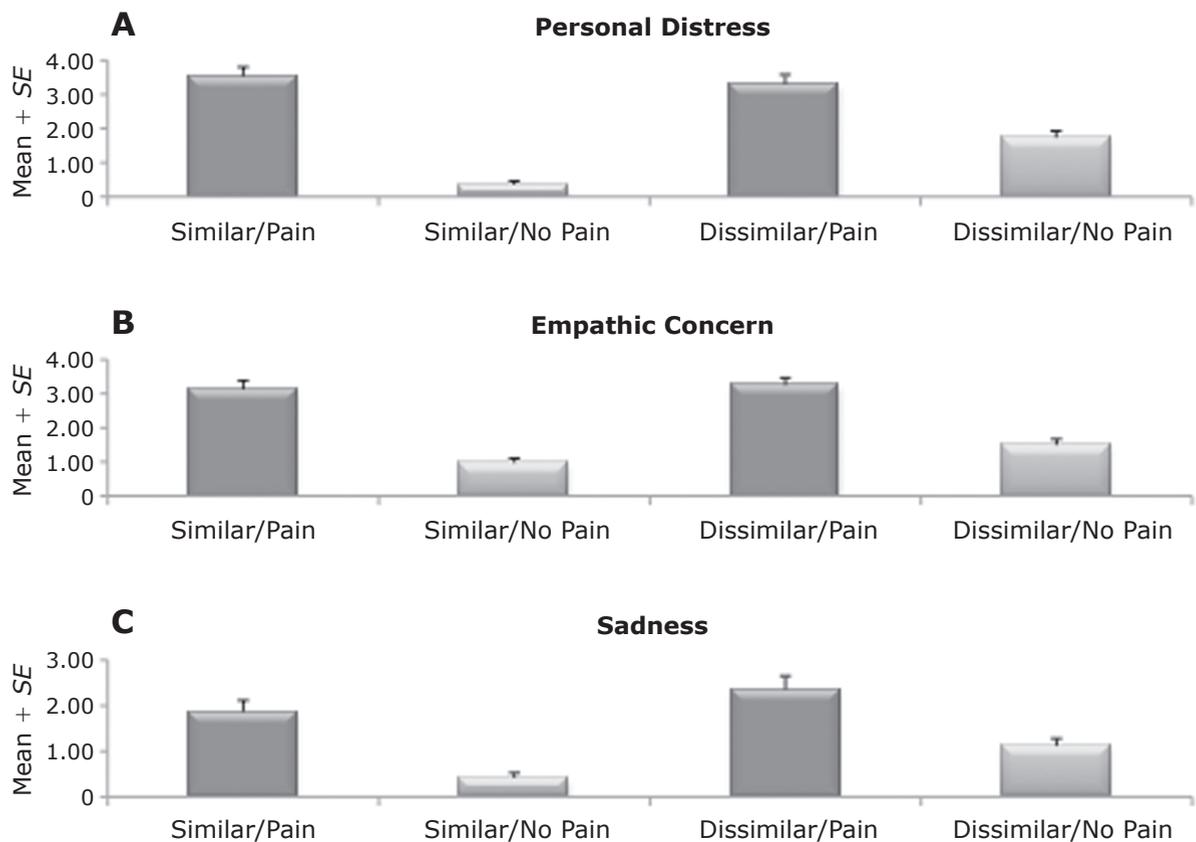


Figure 4. Results of the personal distress, empathic concern, and sadness measures. Participants correctly inferred the target's affective states, since they showed higher empathic concern and personal distress for painful stimulations than for nonpainful ones. However, these matching feeling states, as well as the sadness rating, were modulated considerably by the similarity of the target, since watching nonpainful needle pricks of dissimilar others resulted in higher empathic concern, personal distress, and sadness than did watching nonpainful stimulation of similar others.

Lastly, although qualitative in nature, half of the participants (14) reported having some doubts as to whether these neurological conditions exist, but all reported still feeling that they were able to imagine the pain or no pain the targets were supposedly feeling. Nevertheless, in order to verify whether these doubts influenced the neurophysiological reaction to the stimuli, we formally compared the EEG suppression elicited in believers and doubters.

EEG: 8- to 12-Hz Suppression

The suppression index was first analyzed using an omnibus mixed-model ANOVA. The between-subjects factor was belief (believers, doubters), and the within-subjects factors were site (frontal, central, parietal, occipital), hemisphere (left, right), target similarity (similar, dissimilar), and pain (pain, no pain). When factors included more than two levels, the degrees of freedom were corrected using the Greenhouse–Geisser epsilon values (G–GE), which are provided whenever relevant.

There was no main effect of belief [$F(1,27) = 2.28$, $MS_e = 6.37$, $p = .14$] and no interaction between this factor and any of the within-subjects factors. A significant main effect of site showed that suppression was signifi-

cantly different between the four sites [mean frontal = -0.34 , mean central = -0.31 , mean parietal = -0.38 , mean occipital = -0.52 ; $F(1,27) = 5.5$, $MS_e = 0.514$, $p < .01$; G–GE = 0.65]. Pairwise comparisons revealed that suppression was significantly greater at the occipital than at all other sites ($p < .005$, $p < .01$, and $p = .065$, for occipital versus frontal, parietal, and central, respectively; all Bonferroni corrected). All other pairwise comparisons did not reveal significant differences. A main effect of similarity showed that suppression was slightly greater for dissimilar-other conditions ($M = -0.40$) than for similar-other conditions ($M = -0.38$) [$F(1,27) = 4.7$, $MS_e = 0.024$, $p < .05$]. A main effect for pain showed that suppression was greater for pain conditions ($M = -0.41$) than for no-pain conditions ($M = -0.37$) [$F(1,27) = 5.6$, $MS_e = 0.065$, $p < .05$]. There was no main effect of hemisphere [$F(1,27) = 1.2$, $MS_e = 0.177$, $p = .284$].

The site \times pain interaction was not significant [$F(1,27) = 2.4$, $MS_e = 0.015$, $p = .087$]. Nevertheless, supporting the observed trend, separate planned t test comparisons for pain versus no pain within each region revealed that although suppression was greater for pain conditions all over the scalp (Figure 5), this effect was

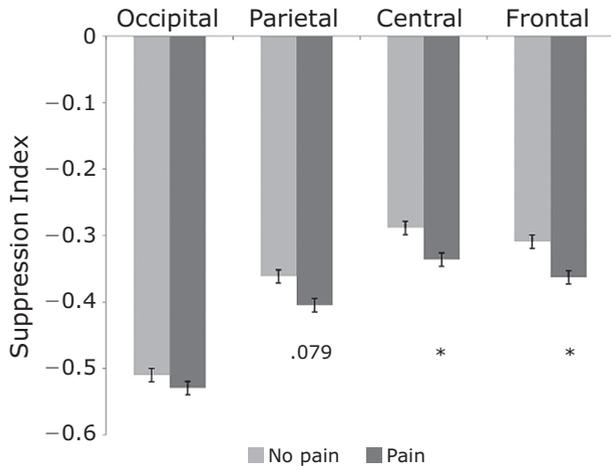


Figure 5. The main effect of pain on mu/alpha suppression as observed at the different anterior–posterior distribution levels. The asterisks denote a significant difference. Note the frontocentral distribution of these effects.

significant in the frontal (−0.36 and −0.31) and central (−0.34, −0.29) regions [$t(27) = 2.865, p < .01$; $t(26) = 3.209, p < .005$, respectively], but not in the parietal (−0.40, −0.36) and occipital (−0.53, −0.51) regions [$t(26) = 1.8319, p = .079$; $t(26) = 1.019, p = .317$]. It is particularly interesting to note that although suppression itself was greatest in the occipital sites, the effect of pain was actually not expressed in these sites, but in the more central and frontal ones.

On the basis of a priori predictions and planned comparisons (initiated by previous fMRI results; Lamm et al., 2010), we further investigated two other interactions (although they were not significant). These were the interactions between pain and similarity [$F(1,27) = 2.5, MS_e = 0.099, p = .126$] and between site and similarity [$F(1,27) = 2.0, MS_e = 0.005, p = .145$].

Planned t tests comparing, across sites, the pain and the no-pain conditions separately for similar and dissimilar other revealed that in the similar-other condition, the effect of pain was significant, such that pain conditions elicited greater suppression than did no-pain conditions [$M = -0.41$ and -0.35 , respectively; $t(26) = 2.64, p < .05$]; in contrast, in the dissimilar-other condition, there was no difference between pain and no-pain conditions [$M = -0.41$ and -0.39 , respectively; $t(26) = -0.963, p = .345$], both of which showed suppression values that were as big as those for pain in the similar-other condition (Figure 6).

Lastly, separate t tests for similar-other versus dissimilar-other conditions in each region (across hemispheres) revealed that although suppression was greater for dissimilar-other conditions across the scalp (Figure 7), this effect was significant in the central regions [dissimilar = −0.33, similar = −0.29; $t(27) = 3.250, p < .005$], but not in any of the other regions [frontal regions, dissimilar = −0.35, similar = −0.32, $t(27) = 1.824, p = .08$; parietal regions, dissimilar = −0.39, similar = −0.37, $t(27) = 1.789, p = .085$; occipital regions, dissimilar = −0.52, similar = −0.51, $t(27) = 0.708, p = .485$]. Note that, again, although suppression itself was greatest in the

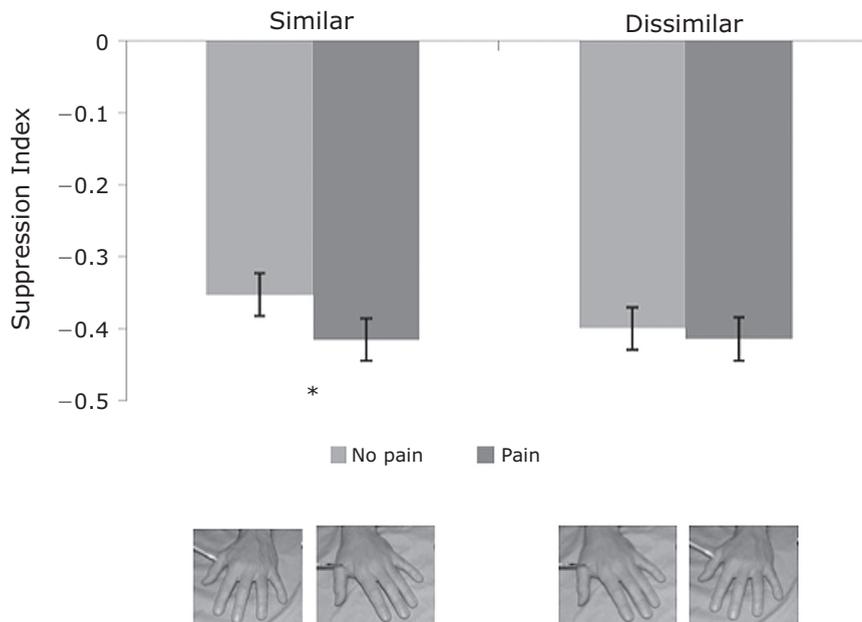


Figure 6. t tests comparing mu/alpha suppression in the pain and no-pain conditions separately for the similar- and dissimilar-other conditions. Note that there were no significant effects in the dissimilar-other condition, where the “nonpainful” stimulus was actually painful for the observer. The asterisks denote a significant difference.

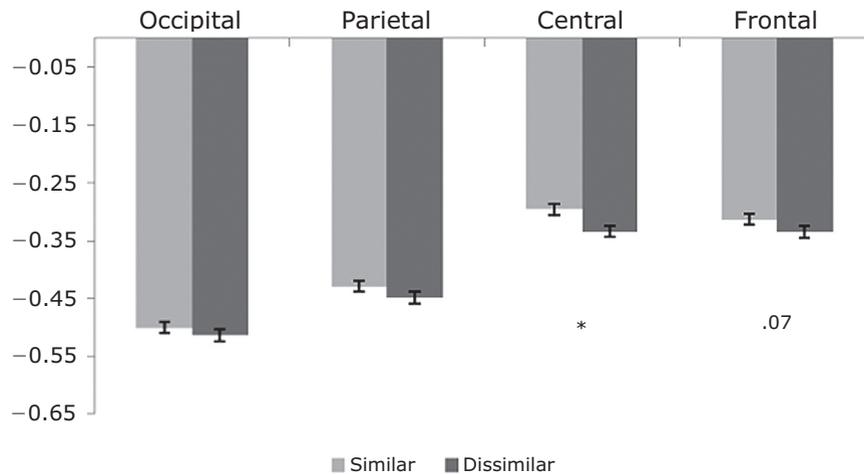


Figure 7. Mu/alpha suppression for similar- versus dissimilar-other conditions at different sites. Note again the frontocentral distribution of these effects. The asterisk denotes a significant difference.

occipital regions, the effect of similarity was actually not expressed at these sites but in central regions.

DISCUSSION

The goal of this study was to explore how empathy elicited by observing pain-inducing actions would be reflected in the observer's brain, as manifested by EEG modulations, and how the observer's ability to identify with the target would affect this process. Specifically, we focused on the suppression of idling EEG rhythms between 8 and 12 Hz (Pfurtscheller et al., 1996), which, when recorded over the sensory-motor cortex, presumably reflect the activation of motor attention or mirroring systems implicated in simulating the other's actions and feelings (for a review, see Pineda, 2005). To achieve these goals, we manipulated the presence of pain enacted in the scene and the extent to which this pain could potentially be shared by the observer. In order to maximize potential empathy, the participants were instructed to imagine the feelings of the targets and to evaluate their affective states (Batson et al., 1997; Lamm, Batson, & Decety, 2007). Hence, the present paradigm captured two possible (empathic) components generated by the involvement of the participants with the presented scenes. One component is an automatic reaction, which is putatively elicited in the observer seeing a painful situation with which he or she can identify. The second is a controlled reaction mediated by cognitive mechanisms and is based on the understanding of the other's sensations and feelings, even if they cannot be directly shared (see Lamm et al., 2010).

The forced choice performance and the pain ratings confirmed the effectiveness of the experimental manipulations. Feelings of empathic concern and personal distress were clearly in line with our expectations, as well as with the findings of Lamm et al. (2010). Participants correctly inferred the target's affective states, since they showed higher empathic concern and personal distress

for painful stimulations than for nonpainful ones. However, these matching feeling states were modulated considerably by the similarity of the target, since watching "nonpainful" needle pricks of dissimilar others resulted in both higher empathic concern and higher personal distress than did nonpainful (Q-tip) stimulation of similar others. The higher feelings of distress might result from an automatically evoked and insufficiently reappraised aversive egocentric response to the needle pricks, which would be painful for the participants. The heightened empathic concern, on the other hand, might result from feeling higher concern for the neurological patient's general condition. A similar mechanism might explain the results for the sadness data, where participants generally reported more sadness when watching pictures of the neurological-dissimilar patients, irrespective of whether they received painful or nonpainful stimulation.

Turning to EEG results, we found significant EEG suppression in the 8- to 12-Hz frequency range in all the experimental conditions, relative to a baseline during which the participants focused on a fixation cross. Notwithstanding the posterior distribution, the experimental effects on the magnitude of EEG suppression were significant only at anterior sites, mostly central and frontal. The distribution of these effects is consistent with the findings of previous studies on mu rhythm suppression recorded over the sensory-motor cortex (Pineda, 2005). At these sites, the EEG suppression induced by observing painful situations was larger than that for nonpainful situations, regardless of whether the pain could be shared by the observer or not. In addition, there was a main effect of similarity, such that dissimilar others (neurological patients) elicited greater suppression than did similar others (patients with "normal" pain perception). Although the pain \times similarity interaction was not significant, planned pairwise comparisons showed that the pain effect might have been modulated by similarity. In the similar-other condition—that is, when the observer could easily iden-

tify with the pain inflicted in the other—suppression was larger in the painful condition (the needle prick) than in the painless condition (the Q-tip touch). In contrast, in the dissimilar-other condition, suppression was equal in the task-defined painful condition (the Q-tip) and the task-defined painless condition (needle prick) and was as large as in the similar-pain condition (see Figure 6). In other words, the EEG measures seemed to be modulated by pain both when the observer could identify, in pain perception, with the target, in line with the suggested link between mu suppression and a simulation mechanism (Buccino et al., 2004), and in the absence of painful-looking stimulation, by means of top-down mechanisms, when the observer was told that a patient experiences Q-tip stimulation as painful (see Decety & Lamm, 2006; Lamm et al., 2010).

This study serves as another step linking mu suppression to higher social-cognitive processing related to the hand region, in addition to motor stimulation. Our results are consistent with those in Cheng, Yang, et al.'s (2008) study, demonstrating that the perception of pain in others modulates neural activity in the primary somatosensory cortex and supporting the idea that the perception of pain in others elicits subtle somatosensory activity. Moreover, this pattern suggests that whereas participants could imagine the pain experienced by the neurological patients, they could not suppress their own reaction to a needle prick, even though it had been defined as painless for the patient. The results thus unveil an automatic response to potentially painful scenes that is unrelated to empathy, probably revealing a sensory-motor reaction to threat. The latter hypothesis is in line with the participants' rating performance, as well as with a previous TMS study demonstrating an automatic elevation in the cortico-spinal motor fibers' sensitivity when the participants watched a movie showing a needle being pushed into someone's hand (Avenanti et al., 2005). However, given the extended distribution of the suppression in the present study, and the low spatial resolution of EEG in general, we cannot unequivocally relate the recorded ERD exclusively with motor activity.

Regardless of whether the present effects are associated primarily with a motor-mirror/simulation mechanism, with an affective network, or with both, the automatic reaction to a needle prick supports a distinction between automatic and controlled sources of empathic reactions to pain (Lamm et al., 2010). According to this view, sharing another's absence of feelings in situations that are distressing or harmful for the self is much more challenging than sharing another's distress from a situation that is innocuous for the self. Indeed, the extra effort needed to identify with dissimilar others, as well as the need to inhibit representations that have been activated via sensory channels and, perhaps, mechanisms associated with perception-action coupling (Singer & Lamm, 2009), may all play a role in the unexpected higher suppression for dissimilar-other than for similar-other conditions over the central regions, and also in the overall higher suppression observed across the experimental conditions over the posterior cortex. This result was also surprising considering a

recent fMRI study demonstrating that the empathic neural response in the ACC decreased significantly when participants viewed faces of out-group members in pain, as compared with faces of in-group members (Xu, Zuo, Wang, & Han, 2009). It is possible that, like alpha suppression (e.g., Klimesch, 1999), mu suppression phenomena are augmented when the cognitive load imposed by the observed stimuli is higher. Situated over the sensory-motor cortex, this may be a more motor-focused attention or a body-focused one. Moreover, it is possible that the modulation of alpha rhythms over the posterior regions was elicited by visual features (e.g., a hand pricked by a needle) that attracted the observer's attention, in addition to their top-down modulation by experimental constraints.

In conclusion, the present results add electrophysiological evidence to models of empathy for pain consisting of both emotional sharing and cognitive evaluation (e.g., Decety & Jackson, 2004; Preston & de Waal, 2002). The data unveil an automatic response to potentially painful scenes, probably revealing a sensory-motor reaction to threat, along with an empathic response, that is most likely driven by cognitive evaluation. As may be expected on the basis of performance in the behavioral tasks and measures, identifying with another who is different from oneself requires more cognitive resources, particularly when the interpretation of the other's feelings contradicts those of the self.

AUTHOR NOTE

The study was supported by an NSF (BCS-0718480) award to J.D. Correspondence concerning this article should be addressed to J. Decety, Departments of Psychology and Psychiatry, University of Chicago, 5848 S. University Ave., Chicago, IL 60637 (e-mail: decety@uchicago.edu).

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(Manuscript received March 29, 2010;
revision accepted for publication August 9, 2010.)