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The impact of mood on empathy for pain: Evidence from an EEG study

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Abstract

The current work investigated whether the neural correlates of empathy for pain are altered by mood valence of observers. Following mood induction, participants watched pictures representing painful or nonpainful situations. We used EEG to record neural activity and assessed event-related desynchronization at central sites during pain observation. Greater mu desynchronization was observed during painful relative to nonpainful situations in positive and neutral mood but not in negative mood. We also found that the pain empathy effect, indexed by mu suppression differences between painful and nonpainful conditions, was smaller in negative than in neutral and positive mood, while this effect was similar between neutral and positive mood. The current study demonstrates that observers' mood states influence the motoric component of empathy for pain, and specifically the negative mood suppresses the motoric empathic resonance for others' pain.

KEYWORDS

EEG, empathy for pain, mimicry, mood, mu suppression, personal distress

1 | INTRODUCTION

Empathy is defined as the sharing and inference of emotional or sensory experiences of others (Bernhardt & Singer, 2012). It plays a crucial role in successful social interactions. Traditionally, the investigation of empathy depends on subjective ratings (Batson, Fultz, & Schoenrade, 1987) and self-reported measures (Bryant, 1987; Davis, 1983). Notably, although this method is helpful to understanding subjective emotional reaction to others' feelings, the results of these studies could be influenced by the intention of self-presentation and social desirability (Fan & Han, 2008). Therefore, it is important to investigate the neurophysiological mechanism underlying

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empathy, which provides more objective assessments of empathic processes.

Empathy has been generally assumed from evolutionary (de Waal, Thompson, & Proctor, 2005) and developmental (Eisenberg, Spinrad, & Sadovsky, 2006) perspectives that the initial component preceding the full-blown human empathic ability draws on somatic mimicry (Hatfield, Cacioppo, & Rapson, 1993). This somatic mimicry mechanism, which leads to shared embodied representations of others' experiences or states, stems from the perception–action coupling (supported by the mirror-neuron system), which automatically activates sensorimotor representations and associated automatic responses in the observer (Preston & de Waal, 2002). On the basis of extensive knowledge about the neural mechanisms of the first-hand experience of pain, the perception of pain in others constitutes a valuable and ecologically valid paradigm to investigate the neural underpinning of human empathy (Decety, 2007). Using fMRI, considerable studies demonstrated that overlapping affective and motivational neural networks (e.g., the anterior insula, anterior cingulate cortex, and periaqueductal gray) are activated when pain is experienced and observed (Cheng et al., 2007; Gu & Han, 2007; Jackson, Brunet, Meltzoff, & Decety, 2006; Jackson, Meltzoff, & Decety, 2005; Jackson, Rainville, & Decety, 2006; Lamm, Batson, & Decety, 2007; Singer et al., 2004). As for the perception–action coupling, recent electrophysiological studies pointed out the sensorimotor resonance during empathy for pain. For example, motor evoked potentials, elicited by transcranial magnetic stimulation, were specifically reduced for subjects observing muscles being pricked (Avenanti, Bueti, Galati, & Aglioti, 2005). The observation of needle and Q-tip stimulations delivered to a model differentially modulated the amplitudes of the somatosensory-evoked potentials (P45) (Bufalari, Aprile, Avenanti, Russo, & Aglioti, 2007). In addition, a previous magnetoencephalography (MEG) study observed changes in the activity of primary somatosensory cortex elicited by the perception of pain in others with the use of mu suppression as an index of motor resonance (Cheng, Yang, Lin, Lee, & Decety, 2008).

Mu rhythm, also known as the central Rolandic or sensorimotor rhythm, includes frequencies in the range of 8–13 Hz (Pineda, 2005), and has been consistently observed over the primary sensorimotor cortex (Gastaut, 1952; Pfurtscheller & Andrew, 1999). The suppression of mu activity relates to stronger blood flow in the somatosensory cortex (Ritter, Moosmann, & Villringer, 2009) and occurs when an action is performed, observed, or imagined (Pineda, 2005). The mu suppression has also been reported to be closely associated with the mirror-neuron activity (Cheng, Lee et al., 2008; Cheng, Tzeng, Decety, Imada, & Hsieh, 2006; Gallese & Goldman, 1998; Oberman et al., 2005; Oberman, Pineda, & Ramachandran, 2007; Pineda, 2005). Accordingly, mu suppression can be a reliable indicator of the activity of sensorimotor cortex and mirror neurons, when participants perceive other people in painful situations (Cheng, Yang et al., 2008).

Previous studies have demonstrated that motor mimicry is an early automatic element involved in affective empathy (Iacoboni, 2009; Lamm, Decety, & Singer, 2011; Singer & Lamm, 2009; Sonnby-Borgström, 2002; Sonnby-Borgström, Jönsson, & Svensson, 2003; Varcin, Bailey, & Henry, 2010). Meanwhile, it has been found that observers' mood states, to a large extent, influence their mimicry (Kuhbandner, Pekrun, & Maier, 2010; Likowski et al., 2011). For instance, Kuhbandner et al. (2010) observed that the joint Simonlike effect was strongest after positive affect induction and absent after negative affect induction while participants were performing a Simon task together with another person, suggesting that positive affect increases and negative affect decreases the automatic representation of other individuals' actions. Likowski and colleagues (2011) recorded electromyography of facial muscular reactions when participants observed faces with happy, sad, angry, and neutral expressions after induction of happy and sad mood in use of film clips. Results showed that positive mood induction led to congruent facial reactions to all facial expressions, whereas negative mood induction led to a general reduction of facial muscular reactions, suggesting that, compared to positive mood, negative mood suppresses facial mimicry. Since motor mimicry is underpinned by sensorimotor cortex (Iacoboni, 2009; Lamm et al., 2011) and is modulated by the observers' mood states, it is reasonable to hypothesize that the automatic motoric empathy of pain in others might vary depending on observers' mood states. In line with this, clinical studies demonstrated that patients with mood disorders showed damaged empathic abilities (Thoma, Friedmann, & Suchan, 2013, for review).

However, whether empathy for pain is modulated by moods has not been directly investigated in healthy individuals. Furthermore, given the observation of a significant mood and empathy interaction, how the interaction effects are related to the functioning of the mirror-neuron system needs to be determined. The answer to this question might be provided by monitoring a neural index that validly represents the activity of the mirror-neuron system, such as mu suppression established by prior studies (Cheng et al., 2006; Cheng, Yang et al, 2008; Oberman et al., 2005). Accordingly, in the current study, we aim to investigate the modulation of mu suppression in empathy for pain by mood states of the onlookers. Based on prior studies (Kuhbandner et al., 2010; Likowski et al., 2011; Thoma et al., 2013), we predicted that the motoric component of empathy for pain would be suppressed when the onlookers were in negative mood, but were facilitated when the onlookers were in positive mood.

Specifically, we anticipated that the pain empathy effects indicated by the difference scores of mu suppression between painful and nonpainful conditions were lowered in negative mood relative to neutral, but were increased in positive relative to neutral mood.

2 | METHOD

2.1 | Participants

As paid volunteers, twenty-three students from Southwest University between 18 and 26 years of age (female: $N = 11$, $M = 21.41$, 95% CI = [20.36, 22.55]; male: $N = 12$, $M = 22.50$, 95% CI = [21.54, 23.46]) were included in the study. Post hoc power analysis via G*power (Faul, Erdfelder, Lang, & Buchner, 2007) demonstrated that the final sample size was large enough to obtain a relatively high power (observed power > 0.9) of key results in the current study. All participants were right-handed, with normal or corrected-to-normal vision and hearing, and reported no neurological or psychiatric history. None of them majored in music or had any experience of performing musical instruments. All subjects provided informed consent to the experimental procedure in accordance with the ethical principles of the 1964 Declaration of Helsinki. The study was approved by the Review Board for Human Participant Research, School of Psychology of Southwest University (China).

Before the experiment, dispositional empathy was measured by using the Chinese version of the Interpersonal Reactivity Inventory (IRI; Zhang, Dong, Wang, Zhan, & Xie, 2010). The IRI contains four subscales corresponding to four aspects of empathy: perspective taking, the ability to take the perspective of others and to see things from their perspectives; fantasy scale, the tendency to identify with other persons; empathic concern, the feelings of concern toward others; personal distress, the feelings of unpleasantness when observing others in sufferings.

2.2 | Materials

A musical mood induction procedure was conducted to induce positive and negative mood states (Chen, Yuan, Huang, Chen, & Li, 2008; Yuan et al., 2014). Headphones were used to listen to 32 negative and 32 positive Chinese classical music excerpts, and 32 neutral broadcast excerpts whose efficacy in inducing the intended moods was validated by previous research (Chen et al., 2008; Yuan et al., 2014). The experiment consisted of three blocks corresponding to three mood conditions, whose order was pseudorandomized among subjects. Before the experiment and at the end of each block, subjects were instructed to evaluate the valence and arousal of their current mood on a 9-point scale (valence: 1 = *extremely sad*, 9 = *extremely happy*; arousal: 1 = *not arousing at all*, 9 = *extremely arousing*).

The visual stimuli consisted of 32 digital color pictures that were selected from previous studies (e.g., Fan & Han, 2008), showing one hand or two in painful and nonpainful situations (see Figure 1a). These images were shot from a first-person perspective and represented situations that were common in daily life. Neutral pictures were similar with painful pictures in contexts except for the absence of cues producing pain. Pictures with right or left hand, and with one or two hands were balanced.

2.3 | Procedure

Subjects were seated in a quiet and sound-proofed laboratory, 150 cm away from a computer screen, sustaining horizontal and vertical visual angle both less than 5°. Each trial procedure (see Figure 1b) started with a fixation (+) displaying in the center of the screen lasting for 300 ms. Then, musical/broadcast excerpts were presented via an earphone, whose offsets were followed by a variable blank screen for 400–800 ms. Subsequently, one painful or nonpainful image was presented for 1,000 ms in the center of a gray background of a 21-inch LCD monitor. Subjects were instructed

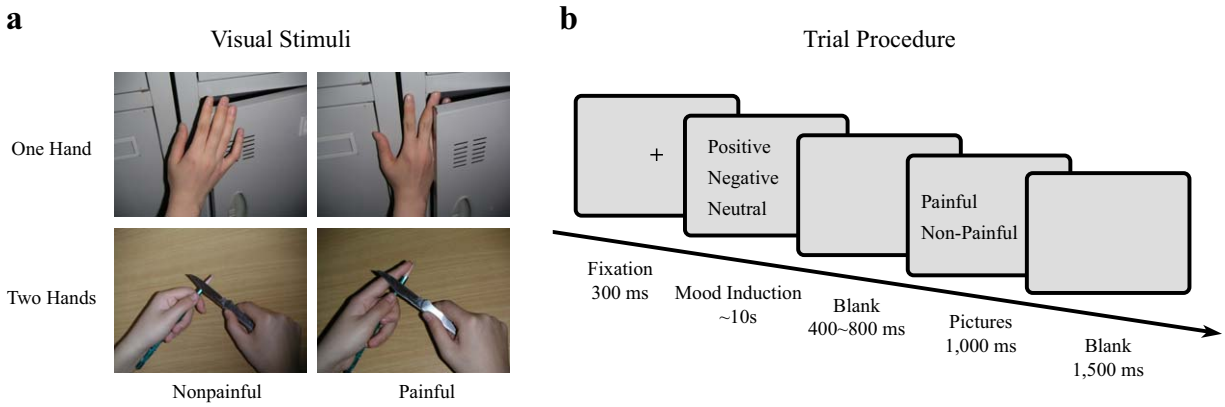

FIGURE 1 (a) Illustration of visual stimuli. (b) Illustration of trial procedure

to press the *F* (or *J*) key if the painful picture appeared, and press the *J* (or *F*) key if the neutral picture appeared. The key press responses were counterbalanced between the left and right hand. The sequences of visual stimuli in each block were randomized. Finally, a blank screen lasting 1,500 ms was presented. At the end of experiment, similar to previous studies (Fan & Han, 2008; Han, Fan, & Mao, 2008), participants were required to evaluate the intensity of pain felt by the model in the stimuli and also the unpleasant feelings by themselves while observing painful and nonpainful stimuli. The evaluations were rated with a 6-point scale (1 = *no pain*, 6 = *very painful*; or 1 = *no unpleasantness*, 6 = *very unpleasant*) with the Face Pain Scale–Revised (FPS-R) adapted from the Faces Pain Scale (Bieri, Reeve, Champion, Addicoat, & Ziegler, 1990), which consists of six photocopied faces showing neutral to extremely painful expressions.

2.4 | EEG recording and analysis

EEG was recorded at 500 Hz using an elastic cap (Brain Products) with 64 sensors according to the extended 10–20 system, with two additional mastoid electrodes and a ground electrode on the medial frontal aspect. The electrode FCz was used as online reference. Electrode impedance was kept at less than 5 kΩ. Raw EEG data were amplified with a dc∼100 Hz band-pass and were filtered with a notch filter at 50 Hz.

Offline EEG processing and analyses were conducted using self-written MATLAB (MathWorks) scripts using functions from the EEGLAB environment (Delorme & Makeig, 2004; v13.5.4b). EEG data were downsampled at 250 Hz and filtered with a high-pass filter at 1 Hz (FIR filter conducted with pop_eegnewfilt of default paramaters, 0.5 Hz cutoff frequency, −6 dB). Nonbrain electrodes were removed and artifactual channels were rejected by clean_ rawdata plugin in EEGLAB, left 55.78 (95% CI = [54.53, 57.04]) clean channels per subject. EEG data were then offline rereferenced to the average. Continuous data were segmented 1,000 ms prior to the onset of image and 2,000 ms poststimulus onset (i.e., each epoch of 3,000 ms). The baseline was corrected by using whole epochs to improve the reliability of independent components (Groppe, Makeig, & Kutas, 2009). Epochs with nonstereotyped artifacts were rejected, and 168 epochs (95% CI = [162.48, 171.87]) per dataset remained for further independent components analysis (ICA). As for the remaining epochs, a significant main effect of pain, $F(1, 22) = 15.82$, $p < .001$, was found with small differences (painful: $N = 27.01$, 95% CI = [26.26, 27.77]; nonpainful: $N = 28.71$, 95% CI = [28.03, 29.39]). There was no significant main effect of mood and interaction effect between pain and mood (all *p*s > .25). Thus, the number of artifact-free, clean epochs was similar across the six conditions.

Epoched data were decomposed into maximally independent components using an extended infomax algorithm implemented by the runica() function with default parameters. After ICA, we also calculated a single-equivalent current dipole model for each IC scalp topography with DIPFIT plugin in EEGLAB (http://sccn.ucsd.edu/wiki/A08:_DIPFIT). For dipole localization, a four-shell spherical model was applied. We excluded independent components with residual variance >15% (low amplitude ICs and noisy scalp maps) and dipoles outside the brain (related to noncortical sources) as well as manually rejecting any remaining artifactual components related to eyeblinks and lateral eye movement. Finally, 13.09 ICs (95% CI = [11.30, 14.87]) per subject, and 301 ICs across all subjects were left.

Clean epoched data were subjected to Morlet wavelet decomposition, which was implemented in EEGLAB newtimef() function. Power was calculated with 50 log-spaced center frequencies ranging from 3 to 50 Hz and 200 linearly spaced time bins across the epoch. Considering the trade-off between frequency and temporal resolution, the wavelets were modified based on the parameter [3, 0.8], specifically 3 cycles at the lowest frequency (3 Hz) and 10 cycles at the highest frequency (50 Hz). The normalization of power used a decibel (dB) transform (dB power = 10*log10 [power/baseline]). The baseline activity was grand-averaged power across conditions at each frequency from −300 to −100 ms prestimulus. Statistical analysis on the baseline power (8–to13 Hz, −300 to −100 ms) showed no significant effect (all *p*s > .15).

In the grand-averaged time-frequency image illustrated in Figure 2, prominent desynchronization of mu oscillations occurred. Time-frequency window of interest (8–13 Hz, 500–1,000 ms) was selected by visual inspection according to the maximal strength of event-related desynchronization (ERD) in the alpha band averaged across all channels,

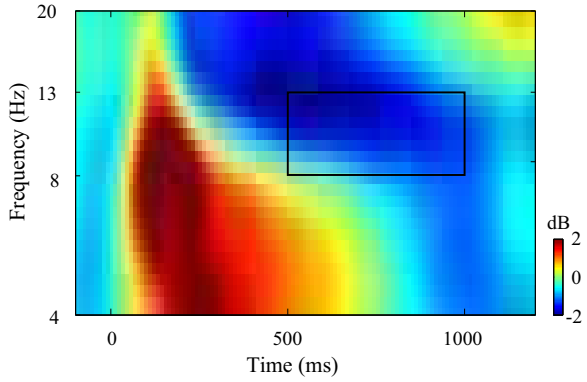

FIGURE 2 Grand-averaged time-frequency image across all channels, subjects, and conditions. The black rectangle represents the time frequency window of interest in the current study

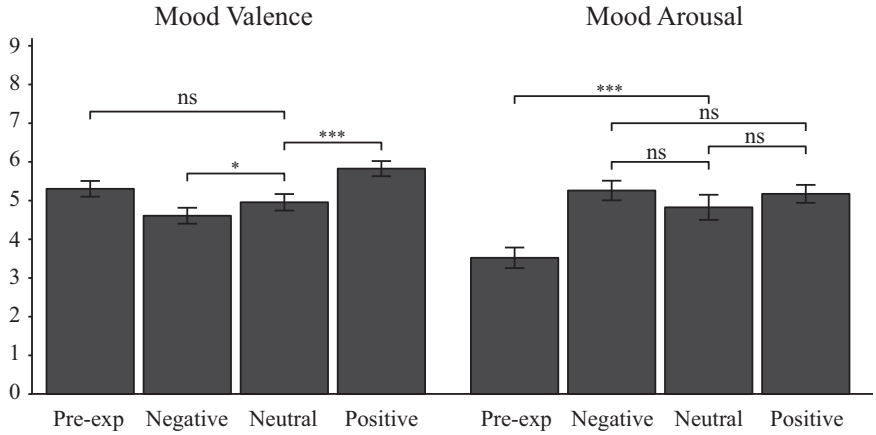

FIGURE 3 Results of the mood valence and arousal ratings for the preexperiment phase, and for neutral, positive, and negative mood induction phases. Error bars indicate standard error of mean. *ns* = not significant; *$p < .05$; ***$p < .001$

subjects, and conditions to avoid potential problems of double dipping (Kriegeskorte, Simmons, Bellgowan, & Baker, 2009). Although data were obtained from all electrodes across the scalp, mu suppression was defined as the mean mu power measured over sensorimotor cortex (C3, Cz, C4; Oberman et al., 2005, 2007). The mu suppression were subjected to a three-way repeated measures analysis of variance (ANOVA), Mood (negative, positive, neutral) × Pain (painful, neutral) × Site (C3, Cz, C4). Based on previous studies (Cheng, Chen, & Decety, 2014; Cheng, Yang et al., 2008; Hoenen, Lübke, & Pause, 2015; Hoenen, Schain, & Pause, 2013; Woodruff, Martin, & Bilyk, 2011), the pain empathy effects were operationally defined as the difference scores of mu suppression between painful and nonpainful conditions. The degree of freedom of the *F* ratio was corrected according to the Greenhouse-Geisser method when sphericity hypothesis was violated. Bonferroni method was used for post hoc pairwise comparisons.

3 | RESULTS

3.1 | Mood manipulation check

Mood valence and arousal ratings were analyzed by a repeated measures ANOVA with four levels of mood induction phase: preexperiment (without induction), neutral, negative, and positive. Valence ratings were significantly different among mood phases, $F(3, 66) = 10.90$, $p < .001$; $\eta_p^2 = .332$. The valence rating scores were significantly higher in the positive mood ($M = 5.83$, 95% CI = [5.42, 6.23]) compared to the neutral mood, $t(22) = 3.90$, $p < .001$, Cohen's $d = .887$, and the preexperimental phases, $t(22) = 2.40$, $p = .01$, Cohen's $d = .546$. Additionally, the valence rating scores were higher in the neutral mood, $t(22) = 1.7$, $p = .05$, Cohen's $d = .346$, and the preexperimental phases, $t(22) = 3.60$, $p < .001$, Cohen's $d = .709$, relative to the negative mood phase ($M = 4.61$, 95% CI = [4.18, 5.04]). The mood valence ratings were not significantly different during neutral mood ($M = 4.96$, 95% CI = [4.51, 5.40]) and the preexperiment ($M = 5.30$, 95% CI = [4.88, 5.73]) phases, $t(22) = 1.90$, $p = .07$, Cohen's $d = .349$. Moreover, we tested the strength of mood valences between negative and positive mood by subtracting out valence scores of neutral valence and found no significant difference, $t(22) = -1.70$, $p = .104$, Cohen's $d = -0.511$.

The mood arousal ratings were significantly different across the four mood induction phases, $F(3, 66) = 13.5$, $p < .001$, $\eta_p^2 = 0.38$. Positive ($M = 5.17$, 95% CI = [4.69, 5.66]), negative ($M = 5.26$, 95% CI = [4.74, 5.79]), and neutral ($M = 4.83$, 95% CI = [4.15, 5.50]) mood phases elicited higher mood arousal than the preexperiment ($M = 3.52$, 95% CI = [2.97, 4.07]) phase, $t(22) = 5.80$, $p < .001$, Cohen's $d = 1.38$; $t(22) = 5.00$, $p < .001$, Cohen's $d = 1.397$; $t(22) = 4.10$, $p < .001$, Cohen's $d = .917$, respectively. There were no significant differences among positive, negative, and neutral mood phases (all $ps \geq .2$). Thus, the results of mood valence and arousal ratings consistently showed that the mood induction procedure used in the present study was effective in inducing corresponding positive, negative, and neutral mood states (see Figure 3).

3.2 | Behavioral performance

The mean reaction times (RTs) and response accuracies in each condition are shown in Table 1. The ANOVAs on RTs showed main effects of pain, $F(1, 22) = .05$, $p > .250$, and mood, $F(2, 44) = 0.59$, $p > .250$. In addition, the interaction effect between pain and mood, $F(2, 44) = 0.03$, $p > .250$, was not significant. The ANOVAs on response accuracies showed a significant main effect of pain, $F(1, 22) = 11.885$, $p = .002$, $\eta_p^2 = .24$, showing that subjects' accuracies were higher to nonpainful than painful pictures. We observed no significant main effect of mood, $F(2, 42) = 1.75$, $p = .185$,

TABLE 1 Mean RTs and response accuracy (standard deviation) in each stimulus condition

	RTs (ms)		Accuracy (%)	
	Painful	Neutral	Painful	Neutral
Negative	655 (59.0)	658 (51.6)	84.66 (8.62)	89.49 (9.67)
Neutral	664 (53.4)	659 (42.7)	86.78 (9.07)	91.94 (6.08)
Positive	665 (57.7)	660 (49.1)	88.07 (9.38)	92.90 (7.23)

and no significant interaction effect of Pain × Mood, $F(2, 44) = .02$, $p > .250$.

3.3 | The FPS-R scores

The mean scores and standard deviation of the subjective reports are shown in Table 2. Two-way ANOVA showed significant main effects of pain in other's pain scores, $F(1, 22) = 262.38$, $p < .001$, $\eta_p^2 = .923$, and in self-unpleasantness scores, $F(1, 22) = 106.76$, $p < .001$, $\eta_p{}^2 = .829$, with the painful stimuli rated more painful and unpleasant than nonpainful stimuli. However, we observed no significant main effect of mood and interaction effects between pain and mood (all ps $> .18$).

3.4 | EEG results

An omnibus three-way repeated measures ANOVA was conducted. There was a significant three-way interaction effect of Pain × Mood × Site, $F(4, 88) = 3.83$, $p = .015$, $\eta_p^2 = .148$, Greenhouse-Geisser epsilon = .721). The breakdown of three-way interaction revealed a significant Pain × Mood interaction effect at C4, $F(2, 44) = 3.50$, $p = .039$, $\eta_p^2 = .137$, but not at Cz, $F(2, 44) = .89$, $p = .393$, and C3, $F(2, 44) = 3.19$, $p = .051$.

The breakdown of the Pain × Mood interaction at C4 showed that painful stimuli elicited stronger mu ERD than nonpainful stimuli in positive, $F(1, 22) = 8.41$, $p = .008$, $\eta_p^2 = .277$, and neutral, $F(1, 22) = 4.83$, $p = .039$, $\eta_p^2 = .18$, mood, but not in negative, $F(1, 22) = .05$, $p > .250$, mood (See Figure 4).

The pain empathy effects (see Figure 4c), as indexed by the mu ERD differences between painful and neutral stimuli, were more pronounced during positive, $F(1, 22) = 6.45$, $p = .019$, $\eta_p^2 = .277$, and neutral, $F(1, 22) = 4.32$, $p = .049$, $\eta_p^2 = .164$, in comparison with negative mood states. There was no significant differences in the empathy for pain in mu suppression between positive and neutral mood, $F(1, 22) = .19$, $p = .667$.

TABLE 2 Mean FPS-R scores (standard deviation)

	Other's pain		Self's unpleasantness	
	Painful	Neutral	Painful	Neutral
Negative	4.87 (0.92)	1.09 (0.29)	3.91 (1.47)	1.30 (0.76)
Neutral	4.52 (1.08)	1.13 (0.34)	3.47 (1.30)	1.17 (0.38)
Positive	4.61 (1.19)	1.30 (0.88)	3.70 (1.26)	1.30 (0.76)

3.5 | Correlation analysis

Correlation analysis demonstrated that significant correlation between pain empathy effect in mu suppression and pain ratings or IRI scores only emerged at the right-central location (C4), where the largest mood modulation of mu suppression was also recorded linked with empathy for pain. Results (see Figure 5) showed that the pain empathy effect following positive mood induction was negatively correlated with the FPS-R difference scores for other's pain, $r(22) = -.626$, $p = .001$, and self-unpleasantness, $r(22) = -.587$, $p = .003$, suggesting that, in positive mood, the more that pain or self-unpleasantness increased, the more pronounced the pain empathy effect was presented. However, this relation was not significant in negative (other's painfulness: $r(22) = -.122$, $p > .250$; self-unpleasantness: $r(22) = -.284$, $p = .200$) and neutral mood (other's painfulness: $r(22) = -.339$, $p = .100$; self-unpleasantness: $r(22) = -.249$, $p > .250$). Additionally, the pain empathy effect in mu suppression was positively correlated with the scores of the personal distress subscale of the IRI in positive, $r(22) = .459$, $p = .030$, and negative mood, $r(22) = .437$, $p = .040$, suggesting that increasing dispositional personal distress during emotional sharing predicts reduced empathic effects for other's pain as indexed by mu suppression (noting that mu suppression is represented by negative values). This association did not exist with all the other IRI subscales. There was also no significant correlation, $r(22) = .133$, $p > .250$, between pain empathy effects in mu suppression and trait personal distress scores in neutral mood.

4 | DISCUSSION

The goal of the present study was to explore whether and how the mood states of observers modulate the neural activity related to empathy for pain by using EEG. Specifically, we focused on the suppression of mu rhythms between 8 and 13 Hz (Pfurtscheller, Stancák, & Neuper, 1996), which presumably reflects the activation of sensorimotor cortex and mirror neuron system implicated in simulating other's actions and feelings (Gallese & Goldman, 1998; Pineda, 2005). Our behavioral data showed that the painful pictures were rated more painful than neutral pictures, regardless of mood, suggesting that the experimental materials used to elicit pain in the present study are valid. The manipulation check of mood

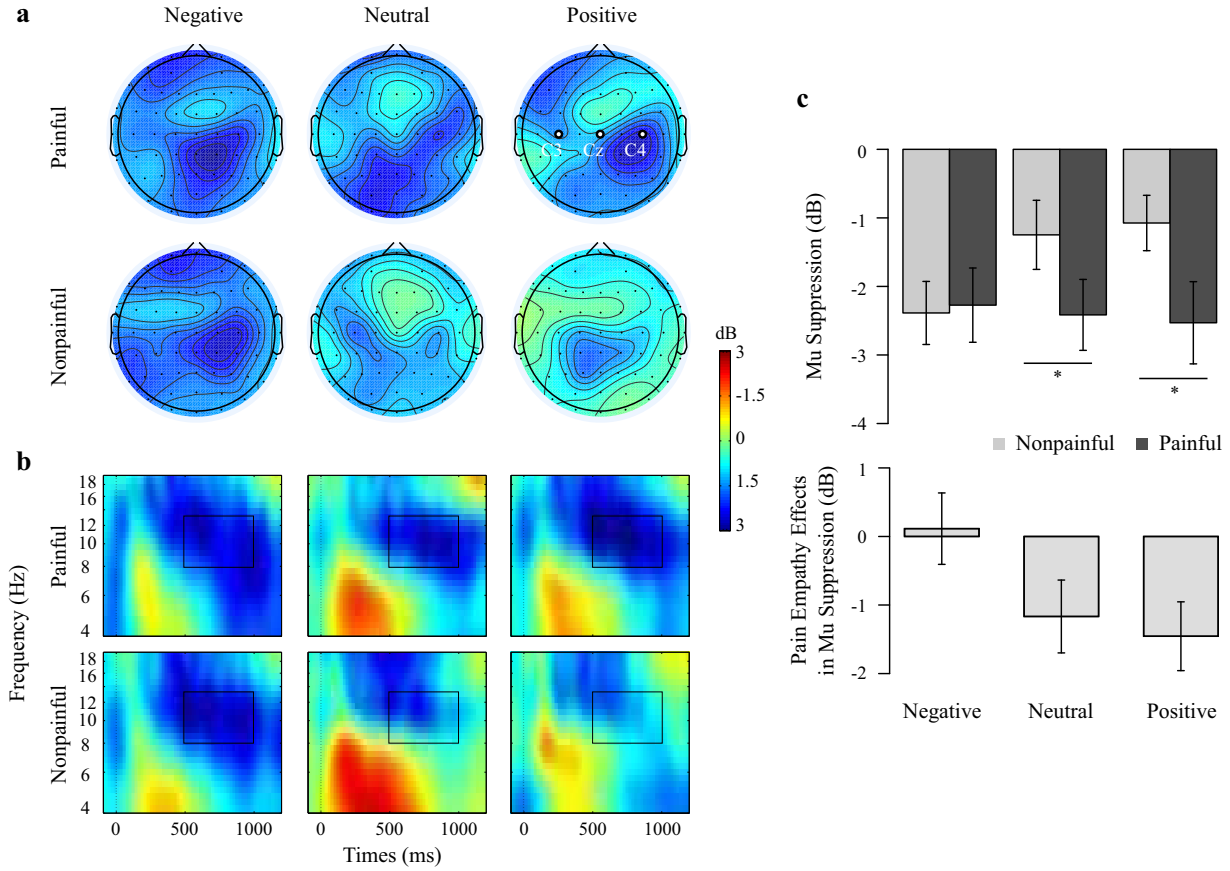

FIGURE 4 (a) Topography of mu suppression (8–13 Hz, 500–1,000 ms). The electrodes of interest were marked. (b) Averaged time frequency image at right-central site (C4). The black rectangle stands for the time-frequency window of interest in the current study. (c) Mean event-related desynchronization (ERD) scores in the mu frequency range (8–13 Hz) by each of the six orthogonally combined conditions (i.e., mu suppression, top) and pain empathy effects of mu suppression (bottom, painful minus nonpainful conditions) at the right-central site (C4). Error bars indicate standard error of the mean

induction procedures demonstrated that the specific moods were successfully induced.

In the present study, we observed that painful stimuli elicited stronger mu suppression than nonpainful stimuli in positive and neutral mood. This is consistent with previous studies reporting that watching painful compared to nonpainful situations suppressed somatosensory neural oscillations to a significantly stronger degree (Cheng, Yang et al., 2008; Yang, Decety, Lee, Chen, & Cheng, 2009). In addition, in both positive and negative mood, we observed a significant positive correlation between the personal distress measure and the empathy-related mu suppression. The personal distress subscale of the IRI accounts for the dispositional negative emotional sharing in response to the distress of others (Davis, 1983). Also, we observed that the mu suppression following positive mood induction increased significantly with FPS-R scores for other's pain and self-unpleasantness, two essential measures of one's situational empathic responses for others' distress (Fan & Han, 2008; Han et al., 2008). Here, considering that mu suppression is a reflection of sensorimotor mimicry during pain empathy (Cheng, Lee et al., 2008), and IRI and FPS-R have been taken as a reliable dispositional (Gazzola, Aziz-Zadeh, & Keysers, 2006) and situational (Bieri et al., 1990) measure of empathy, respectively, the present findings indicate that mu suppression can be a reliable index of motoric empathy for pain in the current study.

More importantly, the pain empathy effects in mu suppression were lower in negative mood than in neutral and positive mood. This suggests that in negative mood the shared representations of states and feelings during observation of other's pain were suppressed to some extent, which is consistent with previous studies demonstrating lowered ability of mirroring other's actions and facial expressions in negative mood (Kuhbandner et al., 2010; Likowski et al., 2011) and impairments of empathic abilities in depression (Thoma et al., 2013). Furthermore, the modulation effect of negative mood on pain empathy and the relationship between empathic neural responses and pain ratings are more pronounced in the right hemisphere. Consistently, an extensive brain lesion and experimental studies showed a dominant role of the right hemisphere in empathic abilities and theory

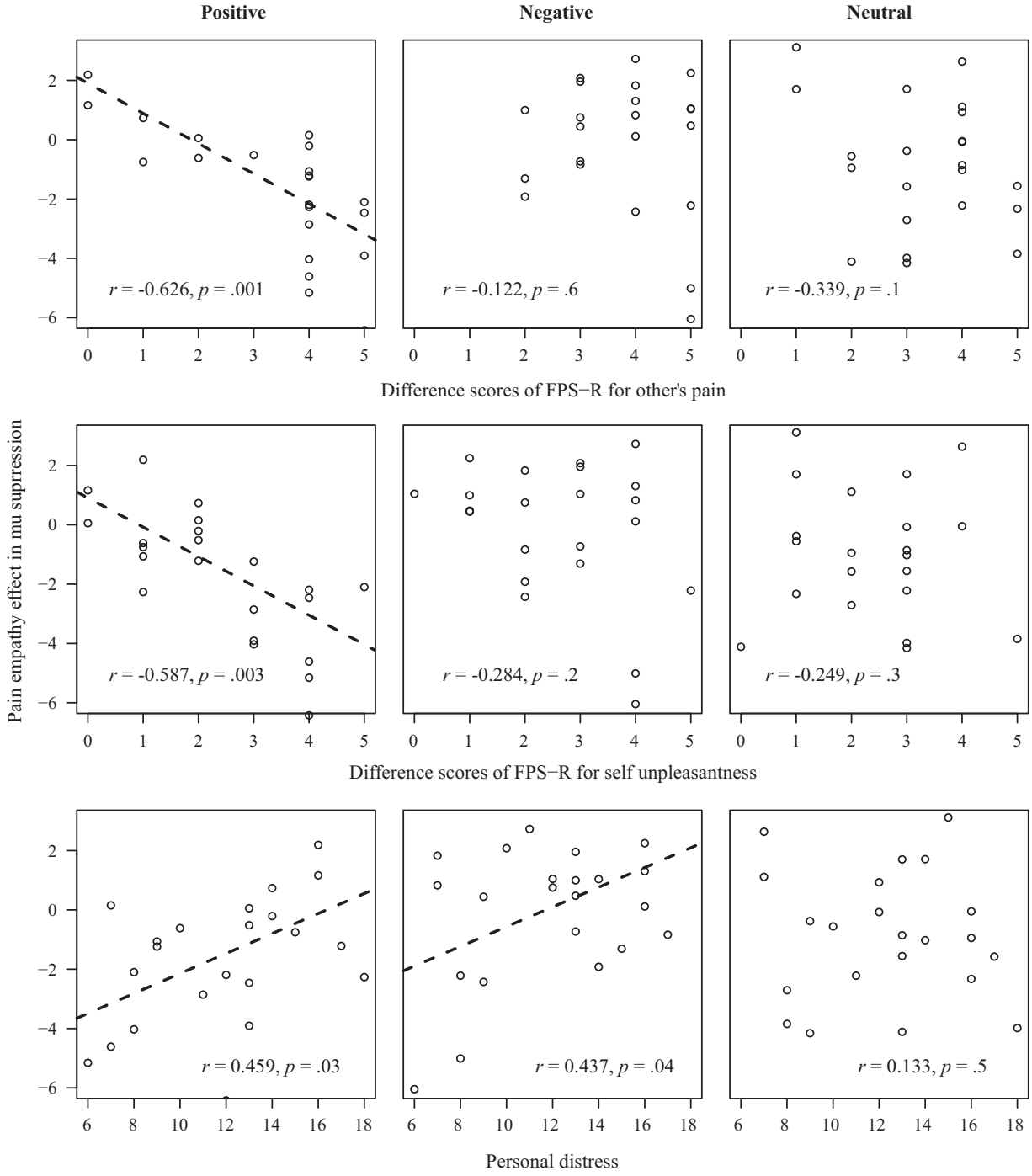

FIGURE 5 Scatter plot (with best-fitting regression line) demonstrating the relation between pain empathy effects in mu suppression at right-central site (C4) and the difference scores of FPS-R, or the scores of trait personal distress in IRI

of mind (Oishi et al., 2015; Rueckert & Naybar, 2008; Shamay-Tsoory, Tomer, Berger, Goldsher, & Aharon-Peretz, 2005; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004; Tullett, Harmon-Jones, & Inzlicht, 2012; Weed, McGregor, Feldbæk Nielsen, Roepstorff, & Frith, 2010). Meanwhile, this finding is consistent with the results that personal distress, an empathic disposition that describes one's strength of personal unpleasantness during sharing others' sufferings (Davis, 1983; Zhang et al., 2010), increased along with reduced pain empathy effects in mu suppression. This is in line with the evidence that personal distress may reduce or even prevent somatomotor mapping of others' pain (Avenanti, Minio-Paluello, Bufalari, & Aglioti, 2009).

By contrast, the current study did not observe the enhancement of pain empathy effect in positive mood compared with neutral mood. It has been established that positive mood is linked with heightened approach motivation and

behavioral tendencies (Albert, López-Martín, Carretié, 2010; Fredrickson, 2004; Wang et al., 2011) and facilitated mobility (Canbeyli, 2010). In this regard, positive mood is likely to increase, rather than decrease, the motoric components of empathy for others' sufferings, consequently leading to enhanced sensorimotor resonance indicated by mu suppression. This explanation is consistent with our findings that only positive mood is associated with a pronounced negative correlation between the pain empathy effect in mu suppression and the two behavioral measures of empathy (self-unpleasantness and other's pain). However, caution should be exercised with this explanation as we did not find a significant enhancement of empathy-related mu suppression during positive relative to neutral mood states.

Of note, the results showed different levels of mu activity to nonpainful stimuli in various mood states, which might cause confusion in understanding the findings in the current study. A handful of evidence has demonstrated that moods or emotional contexts can influence the motor cortical activity and mobility as well (Canbeyli, 2010; Goldstein et al., 2007; Krüger, Seminowicz, Goldapple, Kennedy, & Mayberg, 2003; Ladavas, Nicoletti, Umiltá, & Rizzolatti, 1984; Liotti & Mayberg, 2001; Liotti, Mayberg, McGinnis, Brannan, & Jerabek, 2002; Liotti, Sava, Rizzolatti, & Caffarra, 1991; Mayberg et al., 1999). Specifically, negative mood compared with the rest was associated with increased right premotor cortex activity (consisting of the mirror-neuron system; Krüger et al., 2003; Mayberg et al., 1999). Liotti et al. (2002) found that sad mood induction in healthy subjects and in unipolar depressed subjects was associated with a greater involvement of motor/premotor cortex. It was also indicated that negative mood interferes selectively with right hemisphere processing at the premotor stage (Ladavas et al., 1984; Liotti et al., 1991). This is consistent with our observation that negative relative to positive and neutral mood is linked with enhanced mu suppression for nonpainful/neutral stimuli (see Figure 4).

Although the current study showed the modulation effects of moods on mu suppression, the subjective pain ratings were not altered by various mood states. It is well known that empathy is a multifaceted construct that consists of not only the bottom-up processes that might be powered by emotional contagion and mimicry, but also the top-down processes that are involved in high-level social cognitive processes (e.g., self-other differentiation, emotion regulation) and modulated by contextual appraisal (Singer & Lamm, 2009). It has been empirically shown that sensory-motor processes, including motor mimicry, might have contributed to the neural activity elicited by the picture-based paradigm that was applied in the current study (Lamm et al., 2011). Accordingly, the incongruence between neural and behavioral indexes can only demonstrate that various mood states have an impact on the early automatic motoric but not the late controlled cognitive empathic processes. Future studies using more sophisticated research design and more objective measurements are required to be implemented in order to fully know whether moods can modify the high-level component of empathy.

Another caution that should be taken is the effect of affective priming. Affective priming is the phenomenon that the congruence or incongruence between target stimuli and prime stimuli will facilitate or compromise the processing and responses to the target stimuli, respectively (see Murphy & Zajonc, 1993, for more information). In the current study, we observed the suppression effect of negative mood and the enhancement effect of positive mood on the early motoric component of empathy for pain. However, evidence of the effect of positive mood was weaker. Although there was no interaction effect between mood and pain and also no main effect of mood in reaction time and accuracy, suggesting that behavioral responses were neither facilitated nor impeded, we could not ensure that the effect of affective priming was completely excluded. It is possible that negative mood co-opts brain regions underpinning the resonance with negative emotions (empathy for pain). On the other hand, positive mood might co-opt brain regions underpinning the resonance with positive emotions. Along with this interpretation, the present findings demonstrate that negative emotions might impact empathy toward negative emotions rather than empathy in general.

In summary, the current study showed a mood modulation effect on the early automatic motoric component of pain empathy, but not on the late controlled cognitive component of pain empathy. Specifically, the pain empathy effect of motoric empathy was decreased in negative mood, which was preferentially observed in the right hemisphere. The current findings might be helpful for understanding the damaged empathic abilities in patients of mood disorders (Thoma et al., 2013).

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