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Embodied neural responses to others' suffering

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To investigate whether and how facial mimicry in observers affects their empathic neural responses to others' pain expressions, we recorded event-related potentials (ERPs) from Chinese adults while viewing pain and neutral expressions of Asian and Caucasian faces. Facial mimicry was manipulated by allowing participants to freely move their facial muscles (the relaxed condition) or asking them to hold a pen horizontally using both teeth and lips to prevent facial muscle movement and facial mimicry (the blocked condition). We found that the frontal N1 at 100–120 ms was enlarged by pain vs. neutral expressions. The N1 modulation by facial expressions was significantly reduced in the blocked compared to relaxed conditions and this effect was observed for Asian but not Caucasian faces. The findings suggest that facial mimicry plays a causal role in the early empathic neural response and the embodied empathic neural responses are constrained by the racial intergroup relationship.

Keywords: Facial mimicry; Pain expression; ERP; Race.

Empathy is the ability to understand and share others' emotions. Because of the key role of empathy in altruistic motivation (Batson, 2011) and prosocial behaviors (Preston & De Waal, 2002; de Waal, 2008), the underlying neural mechanisms have been studied extensively using neuroimaging during the last decade. One line of research focused on the neural correlates of empathy for others' painful feelings. Functional magnetic resonance imaging (fMRI) studies have shown that, relative to viewing non-painful stimuli applied to others or neutral expression, perceived painful stimuli applied to others or pain expression elicited prominent activations in the typical pain matrix consisting of the anterior insula (AI), anterior cingulate cortex (ACC) and supplementary motor area (SMA), and somatosensory cortex (Botvinick et al., 2005; Gu & Han, 2007; Gu, Liu, Dam, Hof, & Fan, 2013; Gu et al., 2010; Han et al., 2009; Jackson, Meltzoff, &

Decety, 2005; Saarela et al., 2007; Singer et al., 2004; see Fan, Duncan, Greck, & Northoff, 2011; Lamm, Decety, & Singer, 2011; for meta-analysis). Event-related potential (ERP) studies have shown that the perception of painful compared with non-painful stimuli applied to others modulates the amplitudes of early frontocentral (the frontal N1 and P2), mid-late frontocentral (N2 and N3), and late centroparietal (P3) ERP components (Contreras-Huerta, Hielscher, Sherwell, Rens, & Cunnington, 2014; Decety, Yang, & Cheng, 2010; Fan & Han, 2008; Han, Fan, & Mao, 2008; Li & Han, 2010). Perception of pain versus neutral expression also enlarged the amplitude of the frontocentral P2 component (Huang & Han, 2014; Sheng & Han, 2012; Sheng, Liu, Zhou, Zhou, & Han, 2013).

Interestingly, increasing evidence suggests that empathic neural responses to others' suffering are strongly modulated by multiple factors, such as

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attentional demand (Fan & Han, 2008; Gu & Han, 2007; Sheng & Han, 2012), affective link (Singer et al., 2006), personal experiences (Cheng et al., 2007), and intergroup relationships (Hein, Silani, Preuschhoff, Batson, & Singer, 2010; Huang & Han, 2014; Riečanský, Paul, Kölbl, Stieger, & Lamm, *in press*; Sessa, Meconi, Castelli, & Dell'Acqua, 2014; Sheng & Han, 2012; Xu, Zuo, Wang, & Han, 2009). For example, enhanced attention to painful cues in stimuli or others' painful emotional states significantly increased empathic neural responses (Fan & Han, 2008; Gu & Han, 2007; Sheng & Han, 2012). Ingroup membership determined by shared ethnicity or shared belief also augmented the neural activity to perceived pain in others (Azevedo et al., 2013; Huang & Han, 2014; Mathur, Harada, Lipke, & Chiao, 2010; Sheng & Han, 2012; Sheng, Liu, Li, Fang, & Han, 2014; Xu et al., 2009). While these findings indicate that empathy for others' pain endures cognitive, affective, and social influences, to date, it is unknown whether and how an onlooker's bodily states affect his/her empathy for others' suffering. It is unclear to what degree facial mimicry can improve empathic neural responses to perceived pain expression. Addressing this issue helps to clarify the fundamental mechanisms of empathy related to the theories of embodying emotion.

The basic idea of embodying emotion is that the process of others' emotional states involves perceptual, somatovisceral, and motoric reexperiencing (or embodiment) of one's own relevant emotion (Niedenthal, 2007). The embodiment of emotion influences how emotional information is processed and congruence between an onlooker's bodily expression of emotion and others' emotional states facilitates the processing of others' emotion. In contrast, incongruence between an onlooker's bodily state and perceived emotion impairs comprehension of others' emotion. In support of the theory of embodying emotion, it has been shown that people automatically mimic smiles expressed by virtual characters in dynamic animations (Mojzisch et al., 2006) or in an interactive live setting (Bourgeois & Hess, 2008). Electromyographic (EMG) research also revealed that, when viewing a smile, one's own zygomaticus major muscle contracts within 500 ms after stimulus onset (Dimberg & Thunberg, 1998). Other studies provided evidence for a causal relationship between facial mimicry and the processing of emotion. For example, relative to keeping participants from moving their shoulders, asking participants to avoid facial movements and to clench their teeth slowed responses to categorize positive or negative expressions (Stel & van

Knippenberg, 2008). In addition, blocking the movement of expression-relevant facial muscles by asking participants to hold a pen using teeth and lips significantly impaired the detection of facial expressions (Niedenthal, Brauer, Halberstadt, & Innes-Ker, 2001; Oberman, Winkielman, & Ramachandran, 2007). An ERP study (Wiswede, Münte, Krämer, & Rüsseler, 2009) reported that manipulation of facial muscles related to smile by asking participants to bite a pen modulated the amplitude of error related negativity (ERN) that is increased in participants scoring high versus low on scales for anxiety and worry (Hajcak, McDonald, & Simons, 2003, 2004). Finally, it has been shown that, during imitation of angry faces, blocking facial mimicry using botulinum toxin attenuated the activation of the left amygdala and its functional coupling with brainstem regions implicated in autonomic manifestations of emotional states (Hennenlotter et al., 2009). Taken together, these findings indicate that facial mimicry plays a critical role in recognition of others' emotional states.

An EMG study has shown evidence for an association between empathy ability and facial mimicry (Sonnyby-Borgström, 2002). It was found that when exposed to angry and happy faces, the high-empathy subjects exhibited a higher degree of mimicking behavior than the low-empathy subjects, and this difference emerged at short exposure times (17–40 ms) that represented automatic reactions. In addition, the high-empathy group showed a significantly higher correspondence between facial expressions and self-reported feelings. These findings suggest a link between facial mimicry and empathy. However, there remain several questions regarding the relationship between facial mimicry and empathy. First, does facial mimicry play a causal role in empathic neural responses to others' pain? If facial mimicry is important in constructing embodied simulations for others' pain, then blocking facial muscles engaged in facial mimicry should weaken neural responses to perceived pain expression. Second, the previous ERP research uncovered both early automatic empathic neural responses within 380 ms after stimulus onset that were not influenced by task demands and late empathic neural responses after 380 ms that were modulated by top-down task demands (Fan & Han, 2008). Thus, it is interesting to examine whether facial mimicry affects either early automatic component or late task-dependent component of empathic neural responses, or both. Because high- and low-empathy subjects showed difference in early automatic facial reactions to facial expression

(Sonnby-Borgström, 2002), it is possible that facial mimicry produces greater effects on the early automatic than the late empathic neural responses. Third, as people showed enhanced empathic neural activity to perceived pain in ingroup compared to outgroup members (Azevedo et al., 2013; Huang & Han, 2014; Mathur et al., 2010; Sheng & Han, 2012; Sheng et al., 2014; Xu et al., 2009), it would be interesting to investigate whether the effect of facial mimicry on empathic neural responses depends on the intergroup relationship between observers and targets. As EMG recordings revealed that participants showed increased frowns in reaction to ingroup compared to outgroup members' angry faces (Bourgeois & Hess, 2008), facial mimicry may produce stronger effects on empathic neural responses to pain expressions of ingroup compared to outgroup members.

The present study tested these hypotheses by recording ERPs from healthy Chinese adults during perception of pain versus neutral expressions of racial ingroup (i.e., Asian) and outgroup (i.e., Caucasian) faces. The stimuli and paradigm were similar to those in our previous work (Sheng & Han, 2012; Sheng et al., 2013). However, during the electroencephalography (EEG) recordings, facial mimicry was manipulated by allowing participants to freely move their facial muscles or to hold a pen horizontally using both teeth and lips. This manipulation prevents facial muscle movement and facial mimicry (Niedenthal et al., 2001; Niedenthal, Winkielman, Mondillon, & Vermeulen, 2009; Oberman et al., 2007). Empathic neural responses were denoted as differential ERPs to pain versus neutral expressions. We were particularly interested in whether facial mimicry manipulations differentially modulate empathic neural responses to pain expression of racial ingroup and outgroup members. Given that people are more familiar with emotional expression of racial ingroup than outgroup members (Elfenbein & Ambady, 2002), facial mimicry may play a more important role in empathic neural responses to pain expression of racial ingroup members. Thus, we would expect a greater effect of facial mimicry manipulations on empathic neural responses to pain expression of racial ingroup than outgroup members.

There has been ample evidence that imitation and mimicry are automatic and facilitate empathy (Iacoboni, 2009; Keysers, Kaas, & Gazzola, 2010) and that imitation and mimicry are underlain by the mirror neuron system consisting of the inferior parietal lobule, the posterior inferior frontal gyrus, and adjacent ventral premotor cortex in humans (Iacoboni & Dapretto, 2006). Thus, one may expect that the effect of facial mimicry manipulations on

empathy for others' suffering occurs in the early state of neural responses to pain expression because early empathic neural responses are automatic and less affected by top-down attention compared to late empathic neural responses (Fan & Han, 2008). In addition, as both fMRI (e.g., Gu & Han, 2007; Saarela et al., 2007) and ERP (Sessa et al., 2014) studies have revealed, neural responses to others' pain in the inferior frontal and inferior parietal cortices—brain regions overlapping with the mirror neuron system—suggest that the effect of facial mimicry manipulations on empathic neural responses would be observed in these brain regions. High time resolution of ERPs and related source estimation allowed us to test these predictions.

METHODS

Participants

Twenty-four Chinese college students were enrolled in the present study as paid volunteers (Mean \pm SD = 21.3 \pm 2.1 years, 11 males). All were right-handed, had normal or corrected-to-normal vision, and reported no neurological history. Informed consent was obtained prior to experiment. This study was approved by the ethics committee at the Psychology Department, Peking University.

Mimicry manipulations

Similar to the previous research (Niedenthal et al., 2009), we blocked participants' facial mimicry by asking them to hold a clean pen sideways lightly by teeth and close the lips (the Blocked condition). In a control condition, participants performed the same task with the same stimuli and nothing in their mouth (the Relaxed condition).

Stimuli and procedure

Stimuli consisted of photographs of faces from 16 Caucasian and 16 Asian models (half males in each racial group) adopted from Sheng and Han (2012), as illustrated in Figure 1. Each model contributed a picture of neutral expression and a picture of pain expression, resulting in 64 photographs. Asian and Caucasian faces were matched in perceptual features (e.g., luminance), emotional intensity, and social features (e.g., attractiveness) (Sheng & Han, 2012). Luminance levels of the face stimuli, shown in

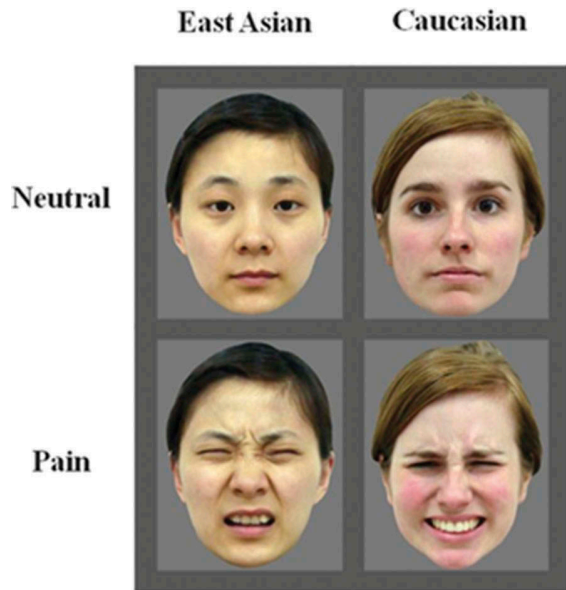


Figure 1. Illustration of the face stimuli used in the current experiment.

Table 1, were subjected to a repeated measure analysis (ANOVA) with Gender (male vs. female faces), Race (Asian vs. Caucasian faces), and Expression (pain vs. neutral faces) as independent variables. This analysis did not show any significant effect ($F_s < 1$), indicating comparable luminance levels of the face stimuli in different conditions.

The current study adopted a within-subject design with Blocking (Blocked vs. Relaxed), Racial Group (Asian vs. Caucasian), and Expression (pain vs. neutral) as independent variables. During EEG recordings, each photograph was presented in the center of a gray background on a 21-inch color monitor, subtending a visual angle of $3.8^\circ \times 4.7^\circ$ (width \times height: 7.94×9.92 cm) at a viewing distance of 120 cm. Each trial consisted of a face stimulus with a duration of 200 ms, which was followed by a fixation cross with a duration varying randomly between 800 ms and 1400 ms. Participants performed pain judgments (pain vs. neutral

expressions) on each stimulus with a left or right button press using the left or right index finger. There were eight blocks of 128 trials. Participants were asked to hold a pen in their mouth in four blocks of trials; there was nothing in their mouth in the other four blocks of trials. The order of Blocked and Relaxed conditions was counterbalanced across participants.

After the EEG session, participants were asked to rate the intensity of pain portrayed by each face and their own subjective feelings of unpleasantness induced by each face on a nine-point Likert scale (1 = *not at all painful or unpleasant*, 9 = *extremely painful or unpleasant*). To assess explicit subjective attitudes toward Asian and Caucasian faces, participants were also asked to rate the likability of each face on a nine-point Likert scale (1 = *not at all*, 9 = *extremely strong*). To assess implicit attitudes toward same-race and other-race faces, participants were asked to complete a race version of the Implicit Association Test (IAT; Greenwald, McGhee, & Schwartz, 1998). The Interpersonal Reactivity Index (IRI; Davis, 1983) was completed to measure participants' empathy ability.

EEG recording and analysis

The EEG was continuously recorded from 62 scalp electrodes that were mounted on an elastic cap in accordance with the extended 10–20 system and were referenced to the average of the left and right mastoid electrodes. The electrode impedance was maintained at less than 5 k Ω . Eye blinks and vertical eye movements were monitored with electrodes located above and below the left eye. The horizontal electro-oculogram was recorded from electrodes placed 1.5 cm lateral to the left and right external canthi. The EEG was amplified (bandpass 0.1–100 Hz) and digitized at a sampling rate of 250 HZ. The ERPs in each condition were averaged separately off-line with an epoch beginning 200 ms before stimulus onset and

TABLE 1
Luminance levels (*mean* \pm *SD*) of the face stimuli (cd/m^2)

	<i>Neutral</i>		<i>Pain</i>	
	<i>Female</i>	<i>Male</i>	<i>Female</i>	<i>Male</i>
Caucasian	31.50 \pm 6.09	31.50 \pm 2.67	32.13 \pm 5.44	30.00 \pm 3.74
Chinese	31.00 \pm 5.73	30.63 \pm 5.42	31.63 \pm 4.69	30.63 \pm 5.73

continuing for 1200 ms. Trials contaminated by eye blinks, eye movements, muscle potentials exceeding $\pm 50 \mu\text{V}$ at any electrode, or response errors were excluded from the average. This resulted in rejection of 20.1% and 19.6% trials in Blocked and Relaxed conditions, respectively. The baseline for ERP measurements was the mean voltage of the 200-ms pre-stimulus interval and the latency was measured relative to the stimulus onset. The mean amplitudes of each ERP component were calculated at electrodes selected from frontal (Fz, F3, F4, F5, F6, FCz, FC3, FC4, FC5, FC6), central (Cz, C1, C2, CPz, CP3, CP4), parietal (Pz, P3, P4), and occipito-temporal (PO7, PO8, P7, P8) regions. Behavioral performances and the mean amplitudes of ERP components were subjected to ANOVAs with Blocking (Blocked vs. Relaxed), Racial Group (Asian vs. Caucasian), and Expression (pain vs. neutral) as independent variables. We reported a range of F - and p -values based on separate analyses for different electrode groups in the main text, but give these values in supplementary tables.

Both voltage topography and the standardized Low Resolution Brain Electromagnetic Tomography (sLORETA; Pascual-Marqui, 2002) were used to estimate potential sources of empathic neural responses. sLORETA is a linear method of computing statistical maps from EEG data that reveals locations of the underlying source processes and does not require a priori hypotheses regarding the field distribution of the active sources. We performed the analysis using sLORETA to assess the 3D current source of neural activity that differentiated between ERPs to pain and neutral expressions of same-race faces in the Relaxed condition. A boundary element model was first created with about 5000 nodes from a realistic head model. Statistical nonparametric mapping was calculated in a specific time window to estimate the source that differentiated ERPs to pain and neutral expressions. The log of the F ratio of

averages was used and considered with a 0.95 level of significance.

EMG recording

To monitor participants' facial muscle movement in the Blocked and Relaxed conditions, during EEG recording, two electrodes were placed over the right masseter and right risorius muscles of each participant to record EMG activity related to the action of biting and upper lip raising (Prkachin, 1992, 2009). Four participants failed during EMG recording due to technique problems and thus 20 participants were used for EMG data analysis. The mean power of EMG activity in the band of 60 ~ 90 Hz—the typical EMG frequency (Merletti & Di Torino, 1999) recorded at our sampling rate—calculated and subjected to ANOVAs with Blocking (Blocked vs. Relaxed) and Sequence (Block 1, 2, 3, and 4 in each condition) as independent variables.

RESULTS

Behavioral results

The results of behavioral performances during EEG recording are shown in Table 2. Response accuracies of judgments of pain versus neutral expressions were high ($> 89\%$). ANOVAs of response accuracies showed a significant main effect of Expression ($F(1, 23) = 15.88, p < .005$) and a significant interaction of Racial Group \times Expression ($F(1, 23) = 12.44, p < .005$). Participants responded more accurately to neutral compared with pain expressions for Asian faces but not for Caucasian faces. ANOVAs of RTs showed a significant interaction of Racial Group \times Expression ($F(1, 23) = 13.57, p < .005$), as participants

TABLE 2
Results of behavioral performances during EEG recording (*mean* \pm *SD*)

		<i>Blocked</i>		<i>Relaxed</i>	
		<i>Neutral</i>	<i>Pain</i>	<i>Neutral</i>	<i>Pain</i>
Reaction Time (ms)	Asian	549 \pm 62	555 \pm 58	540 \pm 65	549 \pm 60
	Caucasian	560 \pm 53	550 \pm 59	551 \pm 63	543 \pm 65
Accuracy (%)	Asian	93.3 \pm 3.55	90.6 \pm 4.14	94.0 \pm 4.21	89.5 \pm 5.10
	Caucasian	91.7 \pm 5.26	91.6 \pm 3.29	92.4 \pm 4.75	92.6 \pm 4.82

TABLE 3
Rating scores related to same-race and other-race faces (*mean*±*SD*)

	<i>Pain intensity</i>		<i>Unpleasantness</i>		<i>Likability</i>	
	<i>Neutral</i>	<i>Pain</i>	<i>Neutral</i>	<i>Pain</i>	<i>Neutral</i>	<i>Pain</i>
Asian face	1.49 ± 0.76	6.84 ± 0.57	2.52 ± 1.33	4.87 ± 1.68	4.64 ± 0.84	3.88 ± 0.93
Caucasian face	1.52 ± 0.76	6.88 ± 0.72	2.44 ± 1.45	4.80 ± 1.57	4.97 ± 1.04	4.21 ± 0.95

responded slower to pain compared to neutral expressions of Asian faces but faster to pain compared to neutral expressions of Caucasian faces. Neither the main effect of Blocking nor its interaction with other factors was significant ($ps > .05$).

Rating scores of pain intensity, self-unpleasantness, likability, and IAT scores were collected from 22 participants. Rating scores of pain intensity and self-unpleasantness related to face stimuli were higher for pain than for neutral expressions ($F(1, 21) = 657.98$ and 41.91 , $ps < .001$); however, these rating scores did not differ significantly between Asian and Caucasian faces ($F(1, 21) = 0.49$ and 0.49 , $ps > .1$). Likability ratings were higher for neutral than pain expressions ($F(1, 21) = 18.99$, $p < .001$) and were higher for Caucasian than Asian faces ($F(1, 21) = 10.68$, $p < .005$), suggesting a more positive explicit attitudes toward neutral than pain expressions and toward Caucasian than Asian faces (see Table 3). The D score of IAT was calculated according to the established algorithm of the latencies (Greenwald, Nosek, & Banaji, 2003) and was significantly larger than zero (Mean ± *SD* = 0.25 ± 0.41 , $t(21) = 2.85$, $p < .05$), suggesting a more positive implicit attitudes toward Asian than Caucasian faces.

ERP results

ERPs to faces in the current experiment were characterized by a negative wave at 100–120 ms (N1) and a positive deflection at 148–188 ms (P2) over the frontocentral area, as illustrated in Figure 2. These were followed by a negative wave at 200–320 ms (N2) over the frontocentral region and a long-latency positivity at 400–680 ms (P3) over the central/parietal area. Face stimuli also elicited a negativity at 140–200 ms over the lateral occipital region (N170, Figure 3). These were similar to those observed in our previous research (Huang & Han, 2014; Sheng & Han, 2012; Sheng et al., 2013).

ANOVAs of the N1 amplitude at 100–120 ms showed a marginally significant main effect of Expression over the frontocentral electrodes (FC5: $F(1, 23) = 4.31$, $p = .049$; F5: $F(1, 23) = 4.01$, $p = .057$) as the N1 amplitude tended to be larger to pain compared to neutral expressions. There was also a significant main effect of Blocking ($F(1, 23) = 4.26$ – 5.57 , $ps < .05$; see Table S1 for statistical details) as the N1 amplitude was larger in the Relaxed compared to Blocked conditions. Interestingly, ANOVAs of the N1 amplitude revealed significant triple interactions of Blocking × Racial Group × Expression over the frontal electrodes ($F(1, 23) = 4.26$ – 5.37 , $ps < .05$, Figure 2). Separate analyses showed significant interactions of Blocking and Expression on the N1 amplitude to Asian faces ($F(1, 23) = 4.54$ – 11.59 , $ps < .05$) but not to Caucasian faces ($ps > .05$). Post hoc analyses further confirmed that the N1 amplitude was enlarged to pain compared to neutral expressions of Asian faces in the Relaxed condition ($F(1, 23) = 4.27$ – 8.55 , $ps \leq .05$) but not in the Blocked condition ($ps > .05$). The voltage topographies of the difference waves to pain (vs. neutral) expressions are illustrated in Figure 4. The sLORETA analysis suggested two potential sources of the N1 activity at 100–120 ms that differentiated between pain and neutral expressions. One was localized to the ACC and the orbital frontal cortex at 100–110 ms (peak MNI coordinates: $-5, 55, -15$) and one to the right inferior parietal cortex at 110–120 ms (peak MNI coordinates: $50, -60, 40$, Figure 2D).

ANOVAs of the P2 amplitude at 148–188 ms showed significant main effects of Racial Group ($F(1, 23) = 17.46$ – 24.72 , $ps < .001$) and Expression ($F(1, 23) = 23.58$ – 36.89 , $ps < .001$), and significant interactions of Expression and Racial Group ($F(1, 23) = 4.44$ – 6.32 , $ps < .05$), due to that pain versus neutral expressions increased the P2 amplitude and this effect was greater to Asian than Caucasian faces. ANOVAs of the N2 amplitude at 200–328 ms showed significant main effect of Racial Group

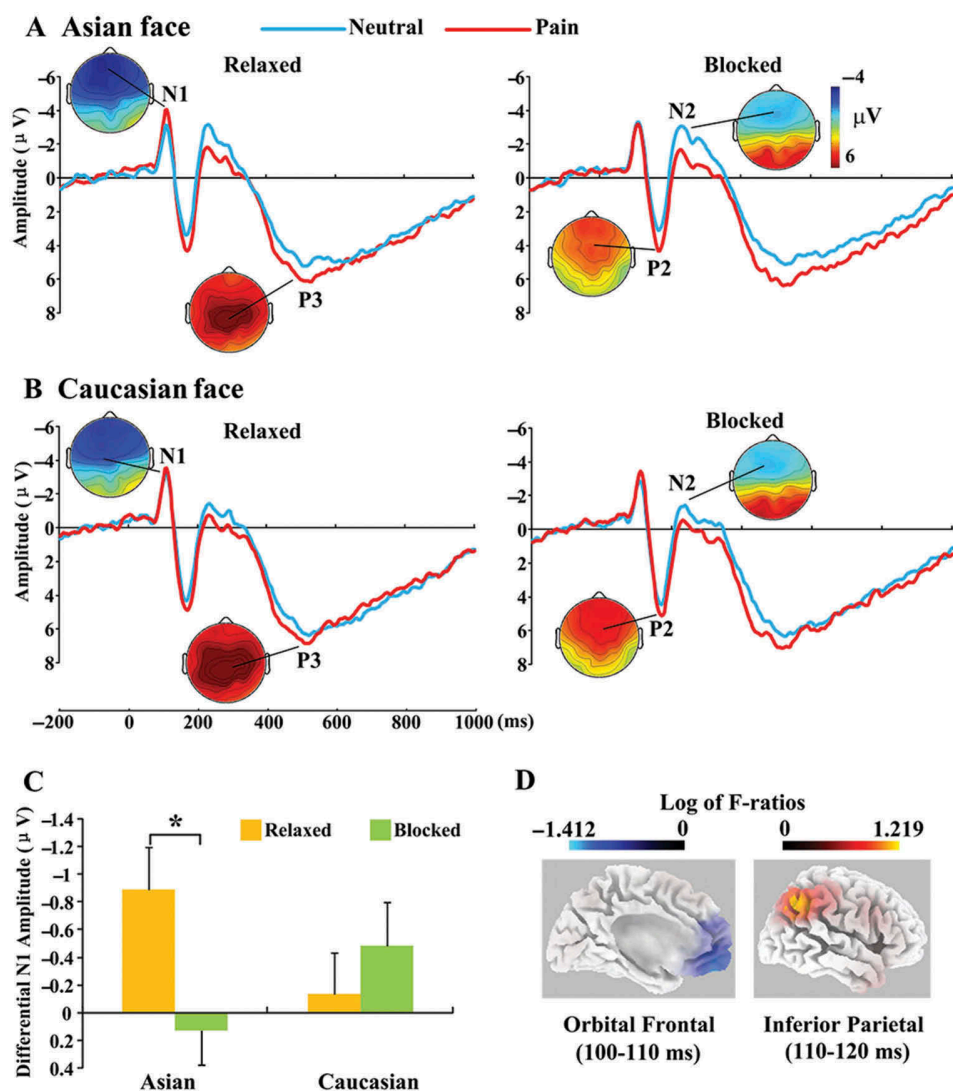


Figure 2. (A) Grand-averaged ERPs to Asian faces recorded at the electrode F5. Voltage topographies illustrate the scalp distribution of the maximum amplitude of each ERP component. (B) Grand-averaged ERPs to Caucasian faces recorded at the electrode F5. (C) The differential N1 amplitudes at 100–120 ms to pain vs. neutral expressions at F5. Error bars indicate standard errors. (D) Source estimation of the neural activity that differentiated between pain and neutral expressions of Asian faces at 100–110 ms and 110–120 ms.

($F(1, 23) = 31.50\text{--}52.60$, $ps < .001$) and Expression ($F(1, 23) = 15.58\text{--}42.89$, $ps < .005$), being larger to Asian than Caucasian faces and smaller to pain than neutral expressions. Similarly, ANOVAs of the P3 amplitudes at 400–680 ms showed significant main effect of Racial Group ($F(1, 23) = 28.60\text{--}45.24$, $ps < .001$) and Expression ($F(1, 23) = 4.44\text{--}8.51$, $ps < .05$), being larger to Caucasian than Asian faces and to pain than neutral expressions.

Recent studies have shown inconsistent results regarding how emotion expressions modulate the N170 (Rellecke, Sommer, & Schacht, 2013). Our previous research suggested a trend of modulations

of the N170 by pain expression (Sheng & Han, 2012), but this effect did not reach significance possibly due to the sample size. The ANOVAs of the N170 amplitude at 140–200 ms in the current work showed significant main effects of Racial Group ($F(1, 23) = 24.04\text{--}32.96$, $ps < .001$) and Expression ($F(1, 23) = 6.11\text{--}13.21$, $ps < .05$), and significant interactions of Expression and Racial Group ($F(1, 23) = 5.62\text{--}6.14$, $ps < .05$), suggesting that pain versus neutral expressions decreased the N170 amplitude and this effect was greater to Asian than Caucasian faces. However, neither the main effect of Blocking nor its interaction with other factors on the

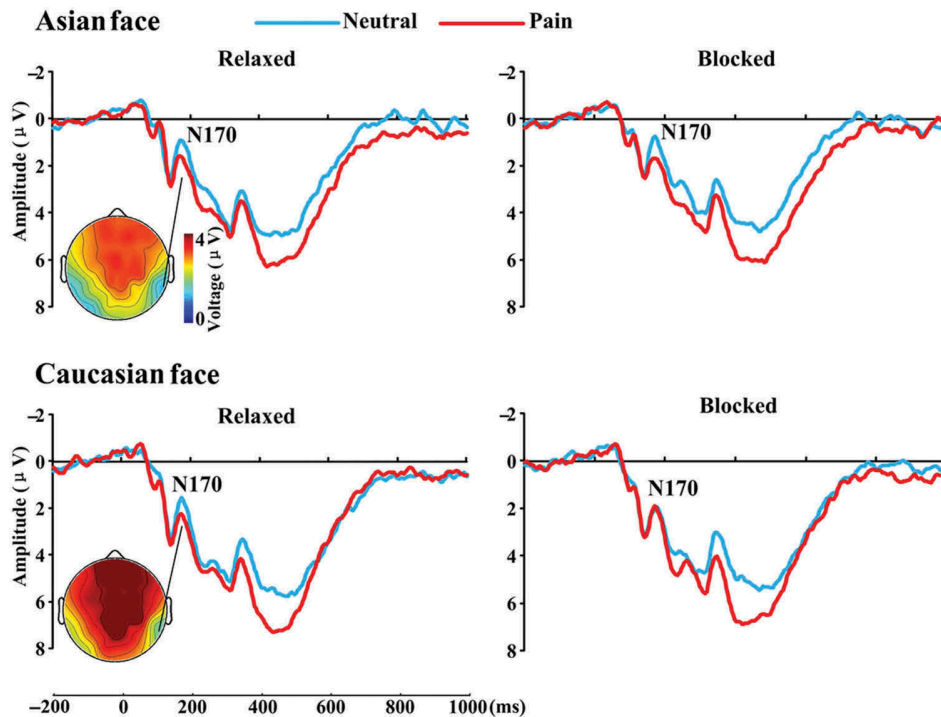


Figure 3. Grand-averaged ERPs to Asian and Caucasian faces recorded at electrode P8.

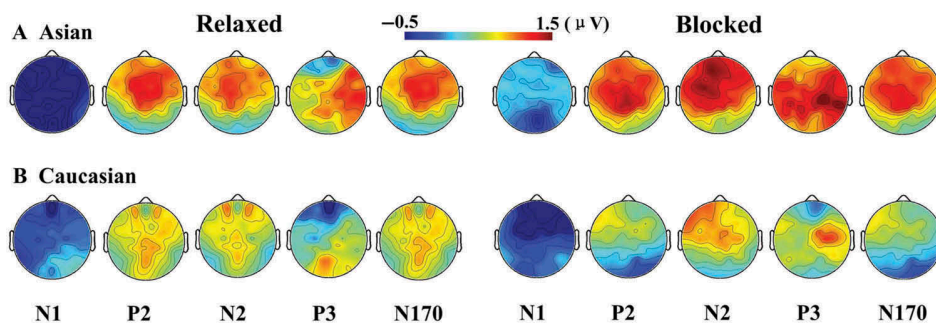


Figure 4. Voltage topographies of the difference waves to pain (vs. neutral) expression in the time windows corresponding to each ERP component.

P2, N2, P3, and N170 amplitude was significant ($ps > .05$).

To examine whether empathic neural responses were associated with subjective feelings of perceived pain and participants' empathy traits, we calculated correlations between the differential ERP amplitudes to pain versus neutral expressions and differential rating scores of pain intensity and self-unpleasantness. The empathic neural responses to Asian faces in the N1 time window were negatively correlated with subjective rating of pain intensity

($r = -.55$ – $-.44$, $ps < .05$, see Figure 5A) and self-unpleasantness ($r = -.57$ – $-.46$, $ps < .05$, see Figure 5B) in the Relaxed condition. The larger the N1 amplitude increased by pain versus neutral expressions of Asian faces in the Relaxed condition, the stronger feelings of others' pain and one's own unpleasantness. To further assess whether the mimicry effect on empathic neural responses was related to participants' empathy traits, we calculated the mimicry effect by subtracting empathic neural responses of Asian faces (i.e., N1 amplitude to pain

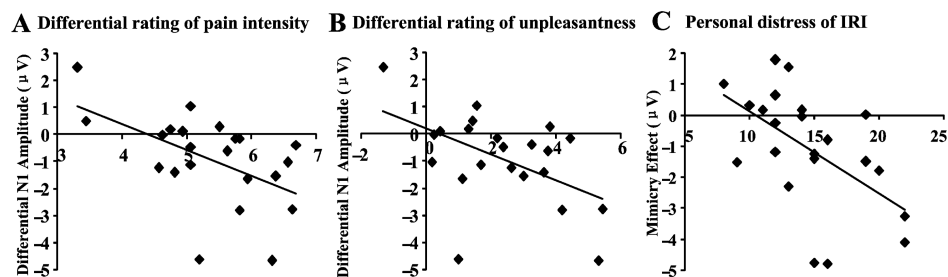


Figure 5. (A) Correlation between subjective ratings of pain intensity and differential N1 amplitudes to pain vs. neutral expressions of Asian faces in the Relaxed condition. (B) Correlation between subjective ratings of self-unpleasantness and differential N1 amplitudes to pain vs. neutral expressions of Asian faces in the Relaxed condition. (C) Correlation between subjective ratings of personal distress and the mimicry effect on the differential N1 amplitudes to pain vs. neutral expressions of Asian faces.

vs. neutral expressions) in the Blocked condition from those in the Relaxed condition. We then calculated the correlation between IRI scores and the mimicry effect on the empathic neural response in the N1 time window. This revealed a significantly negative correlation between the N1 mimicry effect and the subscale of personal distress in IRI ($r = -.56 \sim -.41$, $ps < 0.05$, see Figure 5C), the larger the personal distress score, the greater mimicry effect on the N1 amplitude.

EMG results

ANOVAs of the EMG activity revealed a significant main effect of Block (masseter: $F(1, 19) = 30.28$, $p < .001$; risorius: $F(1, 19) = 24.85$, $p < .001$) and Sequence (masseter: $F(3, 57) = 8.78$, $p < .001$; risorius: $F(3, 57) = 5.11$, $p < .005$). The interaction of Block and Sequence did not reach significance ($ps > .05$). These results suggest that EMG activity was stronger in the Blocked than Relaxed conditions and tended to increase as EEG recording preceded (Figure 6).

DISCUSSION

The current work examined the role of facial mimicry in empathic neural responses to perceived pain expressions. Our EMG results confirmed stronger muscle tension when participants held a pen using both teeth and lips than when they were able to move their facial muscles freely, suggesting that the mimicry manipulation prevented facial mimicry during perceiving pain expressions. Our ERP results first showed that pain compared to neutral expressions significantly augmented the amplitude of the frontocentral N1 and P2 and of the parietal P3 components. Pain versus neutral expressions also induced positive shift of N2 amplitudes over the frontocentral region. These findings replicate the previous ERP findings of empathy for pain (Contreras-Huerta et al., 2014; Decety et al., 2010; Fan & Han, 2008; Han et al., 2008; Huang & Han, 2014; Li & Han, 2010; Sheng & Han, 2012; Sheng et al., 2013). Our ERP results also showed that the increased P2 amplitude to pain versus neutral expressions was more salient when

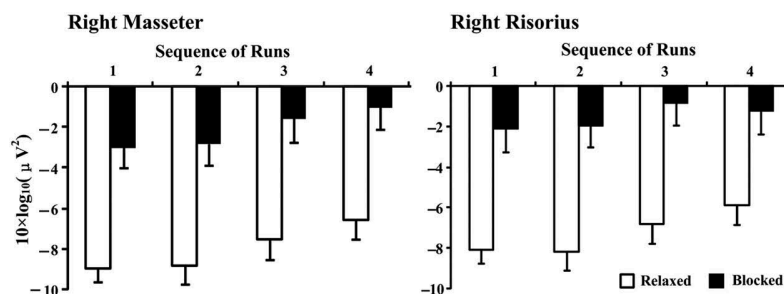


Figure 6. Illustrations of EMG activity recorded at the electrodes over the right masseter and risorius. The EMG activity was transformed using $10 \times \log_{10}$ and was display separately for the relaxed and blocked conditions.

an observer and a target were of the same race than when they were of different races, replicating the racial ingroup bias in empathic neural responses (Sheng & Han, 2012; Sheng et al., 2013).

Most importantly, we showed evidence that facial mimicry significantly modulated the empathic neural responses. Specifically, we found that the N1 amplitude in the Relaxed condition was enlarged by pain versus neutral expression of faces that were of the same race as participants. Our previous research using the same stimuli and task showed similar N1 modulation by pain expression, though this effect did not reach significance, possibly due to a small sample size (Sheng & Han, 2012). Other ERP studies also reported reliable modulations of the N1 amplitude by perceived pain in others (Contreras-Huerta et al., 2014). It has been suggested that the increased N1 amplitude in response to fearful facial expression may reflect early coding of threatening messages (Bar-Haim, Lamy, & Glickman, 2005; Luo, Feng, He, Wang, & Luo, 2010). Pain expression may also convey threatening messages and can be perceived as more arousing and more unpleasant (Simon, Craig, Gosselin, Belin, & Rainville, 2008) and thus led to the increased N1 amplitude compared to neutral expression. Consistent with the previous findings, our source estimation of the neural activity in the N1 time window suggested the engagement of the ACC, the orbital frontal cortex, and the right parietal cortex.

Our results further confirmed that the effect of pain expression on the N1 amplitude was substantially reduced when facial mimicry was blocked by asking participants to hold a pen horizontally using both teeth and lips. The differential N1 amplitude to pain versus neutral expressions in the Relaxed condition was significantly associated with subjective feelings of others' pain and one's own unpleasantness and thus related to empathy. The effect of the mimicry manipulation on the N1 amplitude to pain expression provides the first ERP evidence that facial mimicry is engaged during the early empathic neural response. The previous EMG studies reported evidence for the association between facial mimicry and emotion recognition (Dimberg & Thunberg, 1998; Niedenthal et al., 2001; Oberman et al., 2007) but did not disclose whether and how the neural correlates of emotion recognition vary as a consequence of facial mimicry. Our ERP results indicate that the early empathic neural activity to pain expression—a neural correlate of subjective

feelings with a possible origin in the ACC, the orbital frontal cortex, and the inferior parietal cortex—depends on an onlooker's bodily ability to express the same expression. In addition, we found that participants who reported greater personal distress showed increased mimicry effects on the N1 amplitude to perceived pain expression. Such an association did not demonstrate a causal relationship between personal distress and the mimicry effects on empathic neural responses. However, this result allowed us to speculate that individuals with greater personal feelings of anxiety and discomfort might more strongly depend on mimicry for early sharing of others' painful emotional states. Once facial muscles are blocked and mimicry is dampened, the early empathic neural responses are decreased and other cognitive/affective strategies may be used for understanding others' feelings.

Interestingly, ERP components in later time windows such as the P2, N2, and P3 were not modulated by the mimicry manipulation, though the amplitudes of these components were also sensitive to pain expressions. However, the empathic neural responses in the P2/N2/P3 time windows undergo perceptual and cognitive influences. For example, the P2 empathic neural response was enhanced by a task demand that required focused attention on others' pain (Sheng & Han, 2012). The P3 empathic neural response was significantly reduced when stimulus reality was impaired (Fan & Han, 2008). Thus, it appears that an onlooker's bodily state does not influence the early and late empathic neural responses in the same vein. The empathic neural responses may be dissociated into two stages in the sense that an onlooker's bodily state modulates early empathic neural responses, whereas cognitive strategy such as attention affects the engagement of the late empathic neural activity.

Other ERP research demonstrates that the N1 amplitude is modulated by other facial expressions, such as fear (Eimer & Holmes, 2002; Holmes, Vuilleumier, & Eimer, 2003) and the late ERP components respond to affective pictures with different levels of arousal (see Olofsson, Nordin, Sequeira, & Polich, 2008 for review). It has been suggested that the earlier ERP components (< 300 ms) are linked to attention orientation for unpleasant pictures and the late ERP components are associated with enhanced stimulus processing during memory encoding for arousing pictures (Olofsson et al., 2008). If, according to the theory of embodying emotion, processing others' emotional states engages re-experiencing of one's own relevant

emotion (Niedenthal, 2007), we can assume a similar relationship between bodily states of emotion and encoding/representation of others' emotional states regardless of what emotion (e.g., pain, fear) is processed. This may then allow us to predict that blocking facial mimicry should also modulate the neural activity underlying the processing other types of emotion. This can be tested in future research.

Our ERP results also suggest a new mechanism underlying the racial ingroup bias in empathy. Previous ERP and fMRI studies unveiled multiple factors that contribute to the racial ingroup bias in empathic neural responses. For example, the lack of individuated process and perspective-taking characterizes the perception of perceived pain in racial outgroup members (Drwecki, Moore, Ward, & Prkachin, 2011; Sheng & Han, 2012; Sheng et al., 2014). Perception of pain expressions of racial ingroup and outgroup individuals recruits distinct neuronal populations at a specific stage of the processing stream (Sheng, Han, & Han, 2015). Oxytocin, a neuropeptide that functions as both hormone and neurotransmitter and plays a key role in social attachment and affiliation, facilitates empathic neural responses in the P2 time window to racial ingroup but not outgroup members' suffering (De Dreu et al., 2010, 2011; Domes, Heinrichs, Michel, Berger, & Herpertz, 2007; Sheng et al., 2013). The racial ingroup bias in empathy for pain is also associated with the oxytocin receptor gene (OXTR) because G/G compared to A/A homozygous of OXTR rs53576 showed stronger ACC/SMA activity in response to racial ingroup members' pain (Luo et al., 2015). Life experiences also influence racial ingroup bias in empathic neural responses as Chinese adults who were brought up in Western countries, where Caucasians consist of the majority of population, did not show racial ingroup favoritism in the ACC and insular activity in response to perceived pain in others (Zuo & Han, 2013). The current findings complement the previous work by illustrating that, besides the distinct cognitive strategy and biological function of hormone/neurotransmitter associated with perceived pain in racial ingroup and outgroup members, an observer's bodily state may also contribute to the racial ingroup bias in empathy due to the greater sensitivity of facial muscles to perceived pain expression in racial ingroup than outgroup members. This may be attributed to more social experiences and greater similarity in physical appearance with racial ingroup versus outgroup members, which may lead to a stronger sense of familiarity with racial ingroup members. Together with the finding of increased frowns in

response to ingroup member's compared to the outgroup member's angry faces (Bourgeois & Hess, 2008), our ERP results support the proposition that embodied emotion recognition and embodied empathy are dependent on the intergroup relationship between an onlooker and a target. The automatic and unconscious facial mimicry may play a causal role in understanding and sharing the emotional states of ingroup members.

Early ERP findings have related the N170 to structural encoding of faces in temporo-occipital processing areas (Eimer, 2000; Itier, Latinus, & Taylor, 2006; Itier & Taylor, 2002). Recent research has shown evidence that the N170 amplitude is also modulated by facial expression, being enhanced by positive or negative emotional faces (e.g., Calvo & Beltrán, 2013; Bublatzky, Gerdes, White, Riemer, & Alpers, 2014; see Rellecke et al., 2013, for a recent review of emotion effect on the N170). In addition, the modulation of the N170 amplitude by facial expression was influenced by the racial relationship between an onlooker and perceived faces (Tortosa, Lupiáñez, & Ruz, 2013). White participants showed an enlarged N170 to angry/happy than neutral expressions of black faces and to angry compared to happy expressions of white faces. Our ERP results suggest that pain expressions modulated the N170 amplitude in a way different from those observed in the previous research. Relative to neutral expressions, pain expressions decreased rather than increased the N170 amplitude, and this effect was greater for racial ingroup compared to outgroup faces. These results suggest that the racial relationship between an onlooker and perceived faces also influences the structural encoding of emotional faces, that is, relative to those of racial outgroup faces, the structural encoding was decreased whereas the emotional processing was enhanced for racial ingroup faces. However, the modulation of the N170 amplitude by pain versus neutral expressions was not influenced by the manipulation of facial mimicry. Thus, although pain versus neutral expressions modulated the neural activity in multiple time windows, the effect of facial mimicry on empathic neural responses occurred only in the early time window over the frontal region, indicating the specificity of the effect of facial mimicry on empathy in terms of both temporal and spatial characters of the brain activity.

In conclusion, the current study provided the first ERP evidence that blocking facial mimicry reduced the early empathic neural responses to others' suffering. Our results cast new light on the pivotal role of facial mimicry in empathy for pain. In addition, our results showed brain imaging evidence

that the effect of facial mimicry on the early empathy neural responses was constrained by the social intergroup relationship between an onlooker and a target. Empathy for pain of racial ingroup members may be enhanced by facial mimicry, which, however, may not facilitate empathy for pain of racial outgroup members in a similar vein.

Supplementary material

Supplementary (Table S1) is available via the "Supplementary" tab on the article's online page (<http://dx.doi.org/10.1080/17588928.2015.1053440>).

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