

Physical coldness enhances racial in-group bias in empathy: Electrophysiological evidence

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ABSTRACT

Empathy for others' pain plays a key role in prosocial behavior and is influenced by intergroup relationships. Increasing evidence suggests greater empathy for racial in-group than out-group individuals' pain and the racial in-group bias undergoes sociocultural and biological influences. The present study further investigated whether and how physical environments influence racial in-group bias in empathy by testing the hypothesis that sensory experiences of physical coldness versus warmth enhance differential empathic neural responses to racial in-group vs. out-group individuals' suffering. We recorded event-related brain potentials to painful versus neutral expressions of same-race and other-race faces when participants held a cold or warm pack. We found that brain activity in the N2 (200–340 ms) and P3 (400–600 ms) time windows over the frontal/central region was positively shifted by painful (vs. neutral) expressions. Moreover, the N2/P3 empathic neural responses were significantly larger for same-race than other-race faces in the cold but not in the warm condition. Moreover, subjective ratings of different temperatures in the cold vs. warm conditions predicted larger changes of racial in-group bias in empathic neural responses in the N2 time window. Our findings suggest that sensory experiences of physical coldness can strengthen emotional resonance with same-race individuals.

1. Introduction

Racial in-group favoritism in behavioral tendency (e.g., intention to help members of one's own group more than members of other groups) can be observed in real life situations and has been identified in empirical psychological research. For instance, it was found that individuals reported greater altruistic motivation toward same-race compared to other-race individuals during judicial decisions and clinical pain treatments and the racial in-group favoritism in behavioral tendency was associated with better sharing of racial in-group members' emotional states (Drwecki et al., 2011; Johnson et al., 2002).

The behavioral findings led to increasing interests in brain imaging research to reveal whether and how the brain activity underlying emotional understanding and sharing (i.e., empathy) is modulated by racial intergroup relationships between observers and targets. An early functional magnetic resonance imaging (fMRI) study reported brain imaging evidence of racial in-group bias in empathic neural responses by showing that the anterior cingulate cortex responded more strongly to perceived painful stimulations applied to same-race than other-race

individuals (Xu et al., 2009). Subsequent studies also revealed racial in-group bias in empathic neural responses in the sensorimotor cortex (Avenanti et al., 2010), dorsal medial prefrontal cortex (Mathur et al., 2010; Cheon et al., 2011), anterior insula (Azevedo et al., 2013; Sheng et al., 2014) and temporoparietal junction (Cheon et al., 2011). Event-related potentials (ERPs) research also revealed differential neural responses over the frontal/central regions to same-race and other-race individuals' pain as early as 120 ms after stimulus onset (Sheng and Han, 2012; Sheng et al., 2013, 2016; Han et al., 2016; Contreras-Huerta et al., 2014; Sessa et al., 2014). These brain imaging findings demonstrate that racial in-group bias in empathy occurs during the early stage of neural processes of others' suffering and in multiple brain regions involved in social cognition and emotion.

Because of the novel social significance of the brain imaging findings of racial in-group bias in empathy, recent research has focused on sociocultural and biological mechanisms involved in racial in-group bias in empathy. For example, an ERP study found that changing intergroup relationships by including other-race individuals into one's own team for competition reduced the racial in-group bias in empathy

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by enhancing neural responses to other-race individuals' pain (Sheng and Han, 2012). An fMRI study showed that priming a specific cultural trait (e.g., independence) can decrease racial in-group bias in empathic neural responses in the cingulate and insula (Wang et al., 2015). People with long-term life experiences with other-race individuals during development showed comparable empathic neural responses in the cingulate to perceived pain of same-race and other-race individuals (Zuo and Han, 2013). These findings indicate that social relationship and sociocultural experiences contribute to the differential empathic neural responses to same-race and other-race individuals' pain.

Other studies have revealed biological mechanisms underlying racial in-group bias in empathic neural responses. For instance, by examining repetition suppression of ERP amplitudes to painful expressions, Sheng et al. (2016) showed that neural responses to painful expression of a target face at 120–180 ms (P2) over the central/frontal region was decreased by a preceding face with pain expression when the two faces were of the same race but not different races, suggesting that distinct neuronal populations are engaged in coding painful expressions of same-race and other-race faces. Sheng et al. (2013) also found that intranasal administration of oxytocin selectively increased the P2 amplitudes to same-race but not other-race painful expressions, suggesting engagement of different neural transmitters in the processing of same-race and other-race individuals' pain. A recent fMRI study further revealed greater racial in-group bias in empathic neural responses in the anterior cingulate in G compared with A allele carriers of oxytocin receptor gene polymorphism rs53576 (Luo et al., 2015). Together these findings suggest that racial in-group bias in empathic neural responses is possibly mediated by multiple level biological mechanisms.

While the aforementioned brain imaging findings suggest socio-cultural and biological underpinnings of racial in-group bias in empathic neural responses, it remains unclear whether and how physical environments influence the neural correlates of racial in-group bias in empathy. It has been proposed that an inclement environment with scarce resources threatens human survival and demands increased in-group favoritism (Brewer, 1979). In line with this proposal, a cross-culture study of 116 nations that examined inhabitants' cultural adaptations to climate-based demands and wealth-based resources found that inhabitants in lower-income countries reported greater indices of in-group favoritism such as compatriotism, nepotism, and familism when they had to cope with harsher climates (Van de Vliert, 2011). Laboratory studies also reported that physical coldness compared to warmth increased interpersonal distance (Bargh and Shalev, 2012), which was associated with psychological distance (e.g., Wang and Yao, 2016), and diminished prosocial behavior (Williams and Bargh, 2008). Because empathy has been supposed to be a proximate mechanism of prosocial behavior (Batson et al., 1987; De Waal, 2008; Batson, 2011; Decety et al., 2016) and empathic neural responses can predict altruistic behavior (Hein et al., 2010; Ma et al., 2011), one may hypothesize that an inclement environment that demands in-group favoritism for individuals' survival may increase racial in-group bias in empathic neural responses. In consistent with this hypothesis we recently found that reminding one's own death, which may occur in an inclement environment, increased racial in-group bias in empathic neural responses in multiple brain regions (Li et al., 2015). However, this study did not directly test how harsh physical environments influence racial in-group bias in empathic neural responses. It is likely that sensory experiences in a cold environment may enhance racial in-group bias in empathic neural responses relative to sensory experiences in a warm environment.

The present study tested this hypothesis by recording ERPs from Chinese healthy adults while they perceived Asian and Caucasian faces with painful or neutral expressions. Sheng and Han (2012) found that, during judgments of race identity of faces, the amplitude of a frontal positive activity at 128–188 ms (P2) was enlarged by painful compared to neutral expressions and this effect was stronger for same-race than

other-race faces. A following negative activity at 200–300 ms (N2) showed similar racial in-group bias in neural responses to painful expression. However, the racial in-group bias in empathic neural responses in the P2/N2 time windows was eliminated (i.e., participants showed similar empathic neural responses to same-race and other-race

a race version of the Implicit Association Test (Greenwald et al., 1998). They categorized Asian faces/positive words with one key and Caucasian faces/negative words with another key in two blocks and Asian faces/negative words with one key and Caucasian faces/positive words with another key in another two blocks. A D score, calculated based on an established algorithm of response latencies (Greenwald et al., 2003), provided an index of participants' implicit attitudes toward racial in-group and out-group faces. A D score larger than zero indicates that in-group faces are associated with a positive rather than negative attitude compared to out-group faces. Participant also completed the Interpersonal Reactivity Index (IRI, Davis, 1983) to measure their empathy traits.

2.3. EEG recording and analysis

The EEG Recordings were taken from 64 scalp electrodes (based on the 10/20 system) and two electrodes placed on the left and right mastoids. Eye blinks and vertical eye movements were monitored with electrodes located above and below the left eye. The horizontal electrooculogram was recorded from electrodes placed 1.5 cm lateral to the left and right external canthi. The EEG was amplified (band pass 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 continuing for 1200 ms. Trials contaminated by eye movements and muscle potentials exceeding $\pm 50 \mu\text{V}$ at any electrode or response errors were excluded from average. This resulted in rejection of $19.2 \pm 10.5\%$ of the trials. The baseline for the measurements of ERP amplitudes was the mean voltage of a 200 ms pre-stimulus interval, and the time windows for the measures referred to the stimulus onset. The mean amplitudes of each ERP component were calculated at the frontal (Fz, FCz, F3, F4, FC3, and FC4), central and parietal (Cz, C3, C4, CPz, CP3, CP4) and occipitotemporal (P7, P8, PO7, and PO8) electrodes. The analysis of the P2 and N2 components was conducted over the frontal and central electrodes. The central electrodes were included for the analysis of the long latency component, such as the P3, and the parietal and occipital electrodes were included for the analysis of the early posterior ERP components, such as the N170. Preliminary repeated measures analyses of variance (ANOVAs) of behavior and ERP data included treatment order as a between-subject variable. Neither the main effect of treatment order nor its interaction with other variables was significant ($F < 1$). Thus we reported the results of the ANOVAs of reaction times (RTs), response accuracies, and the mean ERP amplitudes with Treatment (cold versus warm), Expression (painful versus neutral), and Race (Same-race (Asian) versus Other-race(Caucasian)) as within-subjects variables. The ANOVAs of the mean ERP amplitudes recorded at the bilateral electrodes included Hemisphere (electrode over the left versus right hemispheres) as a within subjects variable. To further quantify the effects of cold/warm manipulations, we calculated the difference waves by subtracting ERPs in the control (25 °C) condition from those in the cold and warm conditions. The amplitudes of the difference waves were also subject to ANOVAs with Treatment (cold versus warm), Expression (painful versus neutral), and Race (Same-race(Asian) versus Other-race (Caucasian)) as within-subjects variables.

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 reveal locations of the underlying source processes and do not require a priori hypotheses regarding the field distribution of the active sources. We performed the analysis using sLORETA to assess the potential 3D current sources of neural activity that differentiated between ERPs to painful and neutral expressions. 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 painful

and neutral expressions. The log of the F ratio of averages was used and considered with a 0.95 level of significance.

3. Results

3.1. Behavioral results

Behavioral performances are shown in Table 1. Response accuracies were high ($> 90\%$) and did not significantly differ between the cold and warm conditions ($p > 0.05$). ANOVAs of RTs showed a significant main effect of Race in both the cold and warm conditions ($F(1,39) = 15.01$ and 6.40 , $ps < 0.05$, $\eta^2 = 0.44$ and 0.25) as participants responded faster to Asian than to Caucasian faces. The interaction of Race and Expression was significant in the cold condition ($F(1,39) = 12.82$, $p < 0.005$, $\eta^2 = 0.40$) and marginally significant in the warm condition ($F(1,39) = 4.01$, $p = 0.06$, $\eta^2 = 0.17$). Post hoc analyses revealed that RTs were longer to Caucasian than to Asian faces with neutral expressions in both the cold and warm conditions ($F(1,39) = 5.61$ and 3.67 , $ps < 0.005$, $\eta^2 = 0.62$ and 0.42) but did not significantly differ between Caucasian and Asian faces with painful expressions ($ps > 0.5$).

Painful expressions were rated with higher scores of pain intensity and self-unpleasantness relative to neutral expressions ($F(1,39) = 1288.94$ and 82.24 , $ps < 0.001$, $\eta^2 = 0.97$ and 0.68 , Table 2), but these effects did not differ significantly between same-race and other-race faces ($ps > 0.4$). Participants rated the cold pack with a lower temperature than the warm pack (cold: $M = 1.95$, $SD = 1.01$, warm: $M = 8.55$, $SD = 1.16$, $t(1,39) = 26.06$, $p < 0.001$). However, rating scores of unpleasantness related to the cold and warm pads did not differ significantly (cold: $M = 3.94$, $SD = 1.61$, warm: $M = 3.60$, $SD = 1.34$, $t(1,39) = -1.34$, $p > 0.1$). The D score in the Implicit

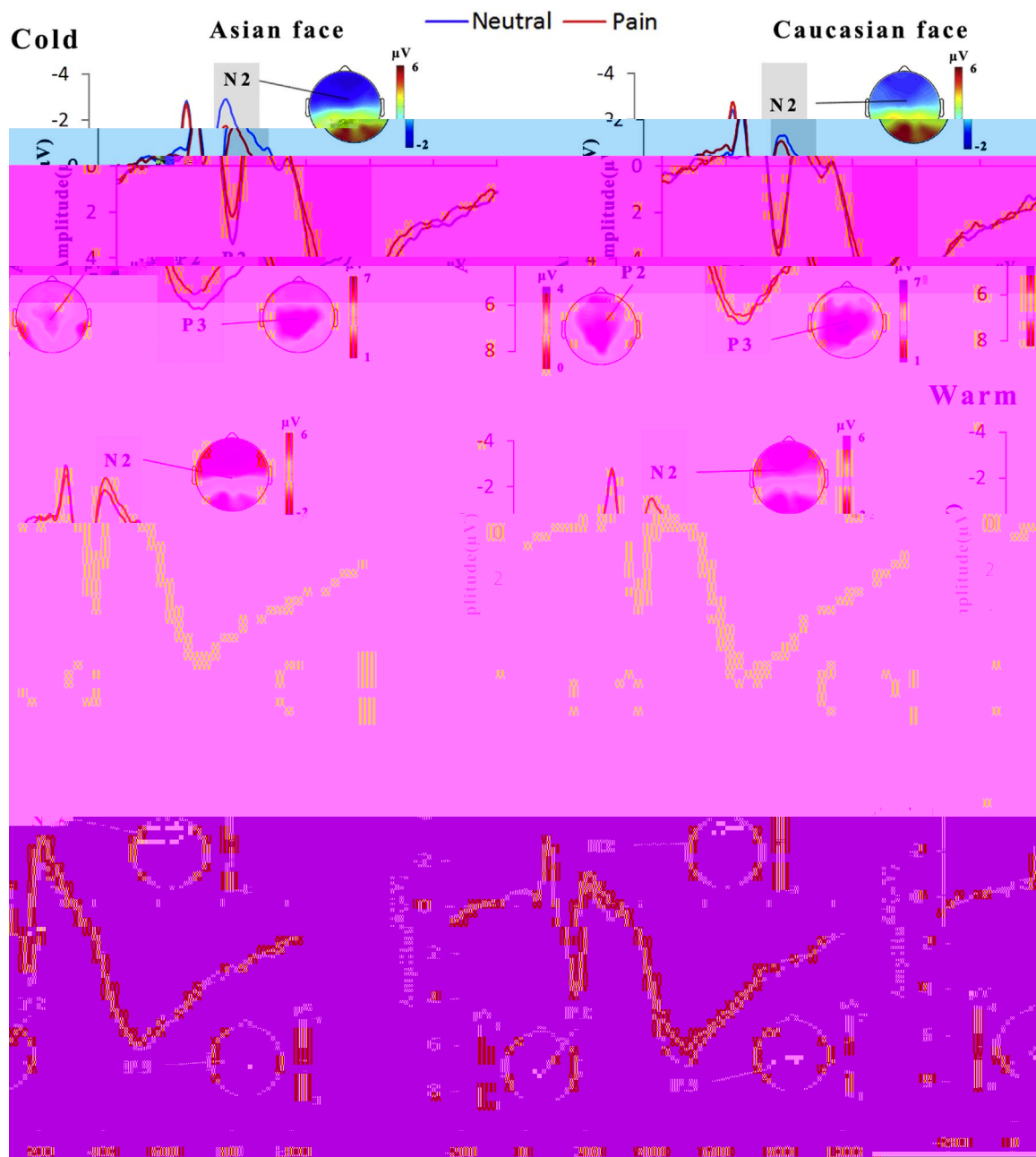


Fig. 1. Illustration of the ERP results in the cold and warm conditions. ERPs recorded at FCz to painful and neutral expressions of Asian and Caucasian faces in the cold, warm, and neutral conditions.

Table 3
Mean amplitudes of FCz for each component (Mean ± SE).

FCZ		Cold condition		Warm condition		Treatment × Race × Expression		
		Asian	Caucasian	Asian	Caucasian	F	p	η ²
P2	Pain	2.57 ± 0.47	3.03 ± 0.52	2.38 ± 0.51	3.10 ± 0.56	3.89	0.06	0.091
	Neutral	1.57 ± 0.47	2.64 ± 0.50	1.77 ± 0.49	2.52 ± 0.50			
N2	Pain	0.51 ± 0.51	0.49 ± 0.51	0.54 ± 0.50	0.49 ± 0.52	6.80	0.01	0.149
	Neutral	-1.52 ± 0.51	-0.55 ± 0.49	-1.31 ± 0.54	-0.70 ± 0.49			
P3	Pain	5.66 ± 0.64	5.99 ± 0.59	5.17 ± 0.58	6.10 ± 0.61	9.35	0.004	0.193
	Neutral	4.69 ± 0.62	5.61 ± 0.58	4.65 ± 0.61	5.20 ± 0.61			

3.2.1. Treatment effects on empathic neural responses

To test our hypothesis of differential racial in-group bias in empathic neural responses, we conduct ANOVAs of the amplitudes of each ERP component in the cold and warm conditions with Treatment

(cold versus warm), Expression (painful versus neutral), and Race (Same-race(Asian) versus Other-race(Caucasian)) as independent within-subjects variables. The ANOVAs of the P2 amplitudes at 140–188 ms over the frontal/central electrodes showed significant main effects of

Race (Fz: $F(1,39)=32.72$, $p < 0.001$, $\eta^2=0.46$; FCz: $F(1,39)=35.55$, $p < 0.001$, $\eta^2=0.48$; Cz: $F(1,39)=39.68$, $p < 0.001$, $\eta^2=0.50$; F3-F4: $F(1,39)=27.68$, $p < 0.001$, $\eta^2=0.42$; FC3-FC4: $F(1,39)=33.69$, $p < 0.001$, $\eta^2=0.46$; C3-C4: $F(1,39)=49.12$, $p < 0.001$, $\eta^2=0.56$) and Expression (Fz: $F(1,39)=32.92$, $p < 0.001$, $\eta^2=0.46$; FCz: $F(1,39)=33.76$, $p < 0.001$, $\eta^2=0.46$; Cz: $F(1,39)=36.81$, $p < 0.001$, $\eta^2=0.49$; F3-F4: $F(1,39)=31.88$, $p < 0.001$, $\eta^2=0.45$; FC3-FC4: $F(1,39)=32.42$, $p < 0.001$, $\eta^2=0.45$; C3-C4: $F(1,39)=28.58$, $p < 0.001$, $\eta^2=0.42$). The P2 amplitudes were enlarged by other-race faces and by painful vs. neutral expressions (see Fig. 1 and Table 3). These replicate the previous ERP findings (e.g., Ito and Bartholow, 2009; Sheng and Han, 2012) and suggest that the frontal/central P2 was involved in coding of both racial identity and emotional state (i.e., pain). The interactions of Expression \times Race on the P2 amplitude were not significant (Fz: $F(1,39)=1.41$, $p=0.24$, $\eta^2=0.04$; FCz: $F(1,39)=3.42$, $p=0.07$, $\eta^2=0.08$; Cz: $F(1,39)=1.98$, $p=0.17$, $\eta^2=0.05$; F3-F4: $F(1,39)=1.39$, $p=0.25$, $\eta^2=0.03$; FC3-FC4: $F(1,39)=1.65$, $p=0.21$, $\eta^2=0.04$; C3-C4: $F(1,39)=1.64$, $p=0.21$, $\eta^2=0.04$), indicating similar effects of painful expression on the P2 amplitude to same-race and other-race faces. These results replicate the previous ERP results that the task demand of pain judgments lead to similar neural responses to painful (vs. neutral) expression of racial in-group and out-group individuals (Sheng and Han, 2012). However, neither the main effect of Treatment nor its interaction with Race and Expression was significant on the P2 amplitude ($ps > 0.05$). Thus physical warm/cold treatments failed to modulate the early empathic neural responses to painful (vs. neutral) expression with the current sample size.

The ANOVAs of the N2 amplitudes at 200–340 ms showed significant main effects of Race (Fz: $F(1,39)=63.34$, $p < 0.001$, $\eta^2=0.62$; FCz: $F(1,39)=61.22$, $p < 0.001$, $\eta^2=0.61$; Cz: $F(1,39)=53.06$, $p < 0.001$, $\eta^2=0.58$; F3-F4: $F(1,39)=45.06$, $p < 0.001$, $\eta^2=0.54$; FC3-FC4: $F(1,39)=49.52$, $p < 0.001$, $\eta^2=0.56$; C3-C4: $F(1,39)=58.90$, $p < 0.001$, $\eta^2=0.60$) and Expression (Fz: $F(1,39)=21.59$, $p < 0.001$, $\eta^2=0.36$; FCz: $F(1,39)=22.27$, $p < 0.001$, $\eta^2=0.36$; Cz: $F(1,39)=23.01$, $p < 0.001$, $\eta^2=0.37$; F3-F4: $F(1,39)=19.11$, $p < 0.001$, $\eta^2=0.33$; FC3-FC4: $F(1,39)=18.79$, $p < 0.001$, $\eta^2=0.33$; C3-C4: $F(1,39)=17.23$, $p < 0.001$, $\eta^2=0.31$). The N2 was of larger amplitude to same-race than other-race faces and was of smaller amplitude to painful compared to neutral expressions (Figs. 1 and 2A). Interestingly, these effects were quantified by significant interactions of Treatment \times Race \times Expression (Fz: $F(1,39)=5.79$, $p < 0.05$, $\eta^2=0.13$; FCz: $F(1,39)=6.80$, $p < 0.05$, $\eta^2=0.15$; Cz: $F(1,39)=7.36$, $p < 0.05$, $\eta^2=0.16$; F3-F4: $F(1,39)=7.32$, $p < 0.05$, $\eta^2=0.16$; FC3-FC4: $F(1,39)=7.42$, $p < 0.05$, $\eta^2=0.16$; C3-C4: $F(1,39)=3.44$, $p=0.07$, $\eta^2=0.08$). simple effect analyses further revealed significant main effects of Expression on the N2 amplitude in the warm condition (Fz: $F(1,39)=11.89$, $p=0.001$, $\eta^2=0.23$; FCz: $F(1,39)=11.83$, $p=0.001$, $\eta^2=0.23$; Cz: $F(1,39)=12.11$, $p=0.001$, $\eta^2=0.24$; F3-F4: $F(1,39)=12.11$, $p=0.001$, $\eta^2=0.24$; FC3-FC4: $F(1,39)=12.11$, $p=0.001$, $\eta^2=0.24$; C3-C4: $F(1,39)=12.11$, $p=0.001$, $\eta^2=0.24$). However, the interaction of Race \times Expression on the N2 amplitude was not significant ($ps > 0.05$), suggesting comparable empathic neural responses to same-race

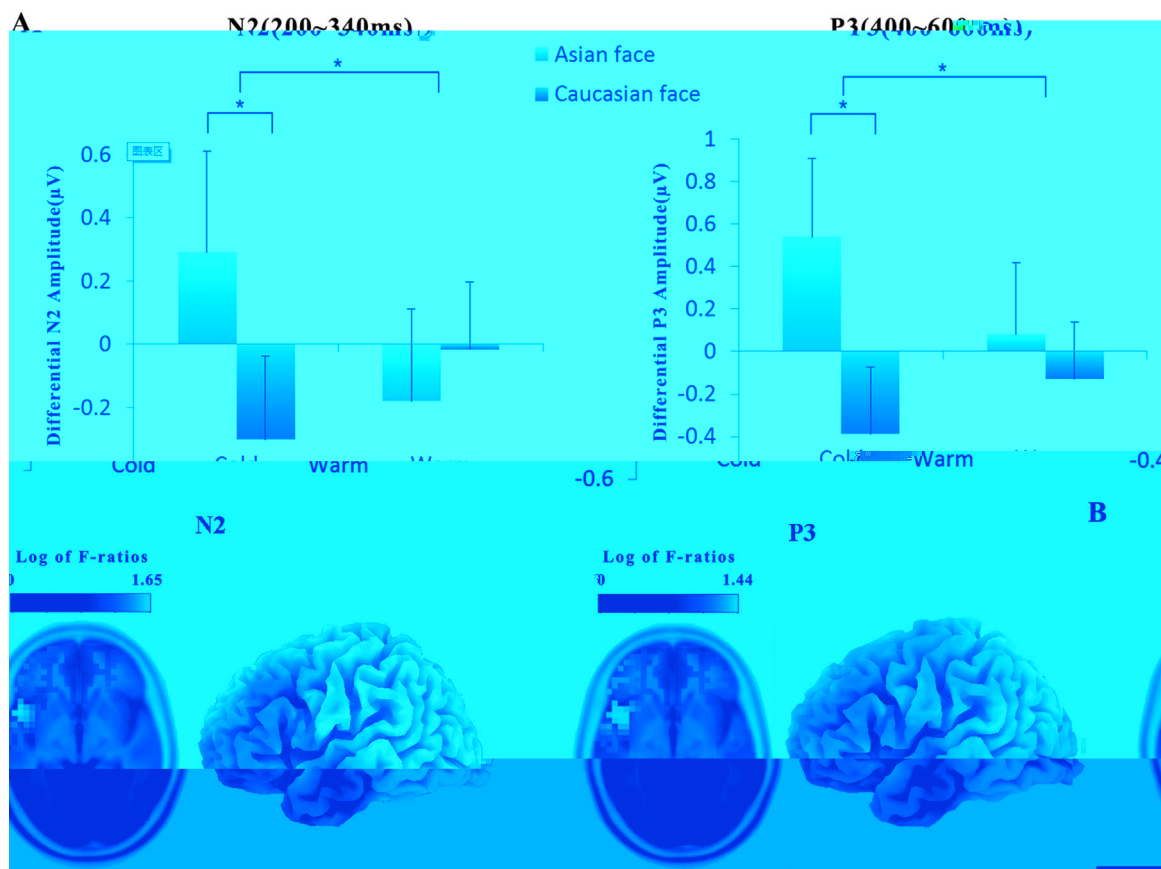


Fig. 2. (A) The mean amplitudes of the diff

$= 20.56$, $p < 0.001$, $\eta^2 = 0.35$; FC3-FC4: $F(1,39) = 23.45$, $p < 0.001$, $\eta^2 = 0.38$; C3-C4: $F(1,39) = 21.70$, $p < 0.001$, $\eta^2 = 0.36$; CP3-CP4: $F(1,39) = 14.88$, $p < 0.001$, $\eta^2 = 0.28$). Source estimation using sLORETA suggested that the neural activity in the P3 time window that differentiated between painful and neutral expressions of Asian faces also had potential sources in left anterior insular and inferior frontal cortex (peak MNI coordinates: $-45, 10, -5$, Fig. 2B).

Finally, we assessed whether treatment, facial expression and race identity influence the amplitude of N170 related to perceptual processing of faces. The ANOVAs of the N170 amplitudes showed significant main effects of Race (P7-P8: $F(1,39) = 44.93$, $p < 0.001$, $\eta^2 = 0.54$; P07-P08: $F(1,39) = 49.71$, $p < 0.001$, $\eta^2 = 0.56$) and Expression (P7-P8: $F(1,39) = 6.54$, $p < 0.05$, $\eta^2 = 0.14$), suggesting larger N170 amplitudes in response to same-race than other-race faces and to neutral than painful expressions. However, neither the main effect of treatment nor its interaction with race and expression was significant on the N170 amplitude ($ps > 0.05$). There was no significant correlation between the N170 amplitudes and the D score ($ps > 0.1$).

3.2.2. Relationships between subjective ratings, implicit racial attitudes and treatment effects

To test whether differential subjective ratings of sensory feelings in the cold vs. warm conditions could predict the differences in racial in-group bias in the empathic neural response between the two conditions, we calculated the correlations between the differential rating scores and the differential racial in-group bias in empathic neural responses in the N2 or P3 time windows in the cold and warm condition. These analyses revealed significant correlations in the N2 time window (F4: $r(40) = 0.36$, $p < 0.05$; FCz: $r(40) = 0.40$, $p < 0.01$; FC4: $r(40) = 0.37$, $p < 0.05$; Cz: $r(40) = 0.38$, $p < 0.05$; C4: $r(40) = 0.47$, $p < 0.05$) and in the P3 time window (Fz: $r(40) = 0.36$, $p < 0.05$; FCz: $r(40) = 0.39$,

$p = 0.01$; Cz: $r(40) = 0.40$, $p < 0.01$; C4: $r(40) = 0.34$, $p < 0.05$, Fig. 3A), suggesting that larger differences in subjective ratings of sensory feelings between cold and warm treatments positively predicted larger changes of racial in-group bias in N2/P3 empathic neural responses in these two conditions.

Next we assessed whether the differential racial in-group bias in the empathic neural responses between the cold and warm conditions was associated with the racial in-group bias in the implicit attitude indexed by the D score in the Implicit Association Test. We found that the D score was positively correlated with the differential racial in-group bias in the empathic neural response between the cold and warm conditions in the N2 time window (C4: $r(40) = 0.35$, $p < 0.05$; CPz: $r(40) = 0.36$, $p < 0.05$; CP4: $r(40) = 0.36$, $p < 0.05$; Pz: $r(40) = 0.40$, $p < 0.01$) and in the P3 time window (CPz: $r(40) = 0.35$, $p < 0.05$; Pz: $r(40) = 0.38$, $p < 0.05$, Fig. 3B), suggesting that racial in-group bias in empathic neural response in participants with greater negative implicit attitudes toward other-race faces were more sensitive to the cold/warm manipulations. We also examined whether individuals' empathy traits were associated with the effect of treatment on the racial in-group bias in the N2 and P3 amplitudes but failed to find significant correlations between the IRI score and the effect of treatment on the racial in-group bias in the empathic neural response in the N2 and P3 time windows ($ps > 0.05$).

4. Discussion

The present study examined the influences of sensory (cold vs. warm) experiences on racial in-group bias in empathic neural responses. We predicted that sensory experiences in a cold environment enhance racial in-group bias in empathic neural responses relative to sensory experiences in a warm environment. Our ERP results support

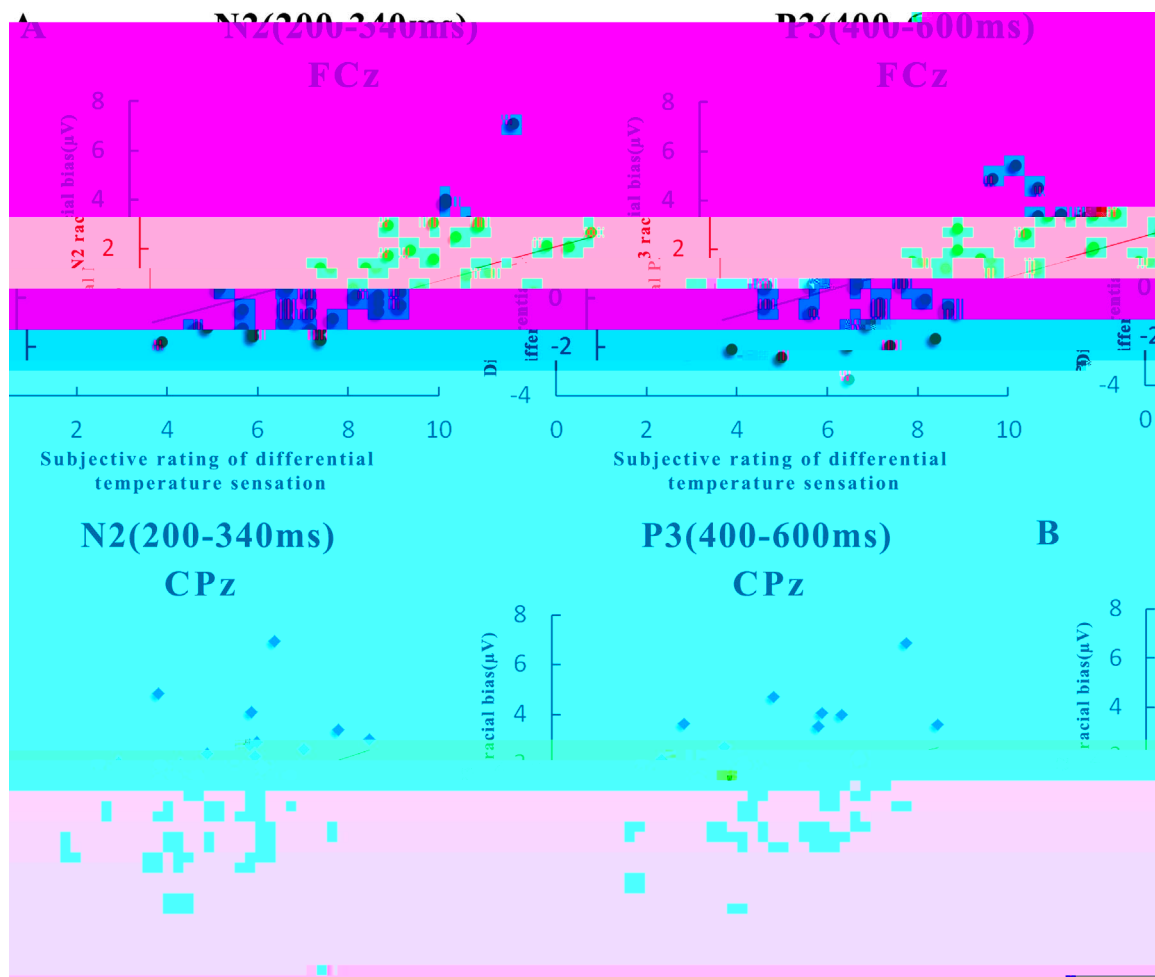


Fig. 3. (A) The correlation between the differential racial bias in the N2 and P3 amplitudes and differential subjective rating scores of temperature sensation to cold vs. warm treatment. (B) The correlation between the racial bias in the implicit racial attitudes and the differential racial bias in the N2 and P3 amplitudes between cold and warm conditions.

this hypothesis by showing evidence that cold experiences during holding a cold pack in hand, which simulated an inclement environment, enhanced racial in-group bias in empathic neural responses to others' pain relative to warm experiences during holding a warm pack in hand.

The ERP results supporting our hypothesis came from the analyses of the N2 and P3 amplitudes to same-race and other-race faces which were positively shifted by painful than neutral expressions. The modulations of the N2/P3 amplitudes by painful vs. neutral expressions are consistent with the previous ERP findings (Sheng et al., 2012, 2013; Han et al., 2016; Contreras-Huerta et al., 2014; Sessa et al., 2014). Moreover, we showed evidence that racial in-group bias in empathic responses in the N2 and P3 time windows was increased in the cold vs. warm conditions. Specifically, we found that, after taking into account of the N2/P3 amplitudes in the control condition, the differential N2/P3 amplitudes to painful (vs. neutral) expressions did not differ significantly between same-race and other-face faces (Fig. 2A) in the warm condition. However, when holding a cold pack, empathic neural responses in the N2/P3 time windows were significantly greater to same-race than other-race faces. Consistent with the effect of cold/warm manipulations on racial in-group bias in empathic neural responses, we found that individuals' subjective ratings of cold vs. warm feelings positively predicted the differential racial bias in the empathic neural response in the N2/P3 time window between cold and warm conditions. These results together suggest a causal link between cold experiences and increased racial in-group bias in empathic neural responses.

In addition, we found that the effect of physical coldness on racial in-group bias in empathic neural responses varied depending on participants' negative implicit attitudes toward other-race faces. The racial in-group bias in empathic neural responses in both the N2 and P3 time windows were more easily influenced by the cold/warm manipulations in those with greater negative attitudes toward other-race faces. It has been shown that experiencing warm or cold significantly affected attitudes (e.g., social trust) toward others (Williams and Bargh, 2008; Kang et al., 2011). Our study employed a within-subject design and the IAT was conducted after both warm and cold treatments. Thus it is unlikely that participants' implicit attitudes toward same-race and other-race faces were changed specifically by warm or cold treatments. Our results suggest possible interactions between sensory experiences and attitudes toward same-race and other-race individuals on the racial in-group bias in empathic neural responses and leave an open question for future research to clarify, i.e., where and how such interactions occur in the brain.

The early P2 amplitude that was sensitive to painful and neutral expressions did not show racial in-group bias in the task that required pain judgments. This is consistent with our previous findings that a task demand of focusing on each individual's painful feeling prompted empathic neural responses to both racial in-group and out-group members' suffering (Sheng and Han, 2012; Sheng et al., 2014, 2016). However, the P2 empathic responses did not vary in accordance with the cold vs. warm manipulations. Thus sensory experiences were unable to differentially modulate the empathic neural responses to same-race and other-race faces in the early P2 time window in the current sample.

size.

The previous ERP research revealed that the early empathic neural responses in the P2 and N2 time windows to hands and same-race faces were associated with emotional sharing (Fan and Han, 2008; Sheng and Han, 2012) and, to a certain degree, occurred independently of task demands that emphasized top-down attention to emotional cues in visual stimuli. The P3 amplitude to others' pain, however, was increased by top-down attention to others' emotional states (Fan and Han, 2008). There was also evidence that the positive activity over the frontal/parietal region in a similar time window was sensitive to cognitive evaluation of others' mental states (e.g., Sabbagh and Taylor, 2000; Wang et al., 2010). The current ERP results suggest that cold experiences seemed to mainly affect the late differential neural processes to same-race and other-race individuals' pain. Our source estimation suggested that the empathic neural responses in the N2/P3 time windows possibly originated from the left anterior insular and inferior frontal cortex—a brain region that is engaged in coding cold vs. warm temperatures (Kang et al., 2011) and in which empathic neural responses show racial in-group bias (Azevedo et al., 2013; Sheng et al., 2014). A meta-analysis suggested that the left insula is involved in both the cognitive–evaluative and affective

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