

Racial Bias Reduces Empathic Sensorimotor Resonance with Other-Race Pain

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Summary

Although social psychology studies suggest that racism often manifests itself as a lack of empathy [1, 2], i.e., the ability to share and comprehend others' feelings and intentions [3–7], evidence for differential empathic reactivity to the pain of same- or different-race individuals is meager [8, 9]. Using transcranial magnetic stimulation, we explored sensorimotor empathic brain responses [10–15] in black and white individuals who exhibited implicit but not explicit ingroup preference and race-specific autonomic reactivity [16–20]. We found that observing the pain of ingroup models inhibited the onlookers' corticospinal system as if they were feeling the pain [10–15, 21, 22]. Both black and white individuals exhibited empathic reactivity also when viewing the pain of stranger, very unfamiliar, violet-hand models. By contrast, no vicarious mapping of the pain of individuals culturally marked as outgroup members on the basis of their skin color was found. Importantly, group-specific lack of empathic reactivity was higher in the onlookers who exhibited stronger implicit racial bias. These results indicate that human beings react empathically to the pain of stranger individuals [3–7]. However, racial bias and stereotypes may change this reactivity into a group-specific lack of sensorimotor resonance [1–3, 9, 23, 24].

Results and Discussion

Although pain has long been considered an inherently private experience, neuroimaging and neurophysiological studies indicate that when people see or imagine the pain of another person, they map the others' pain onto the network activated during firsthand experience of pain as if they were vicariously experiencing the observed pain [25–34]. This empathic reactivity to others' pain is modulated by the observer's personality [14, 15, 25], previous experience in the same situation [27], and the appraisal of it [28]. Moreover, the perceived model's fairness [25] and the observer model's state or trait similarity strengthen empathic pain resonance [3, 29]. Tellingly, racial cues may be fundamental in modulating the perceived

similarity between two individuals [2]. Despite the notion that racial bias and preference are inherently linked to a lack of empathy [1, 2], information on empathic brain reactivity to the pain of same- or different-race individuals is very scanty. Neural regions associated with self (e.g., the medial prefrontal cortex) may underpin extraordinary empathy for the pain of one's own social group members [8]. Moreover, reduced neural activity in emotional brain areas during observation of painful stimuli applied to the face of different- versus same-race individuals has been reported [9]. Although this finding may hint at lower empathy for the pain of racial outgroup members [9], it is not clear whether the effect is due to racial bias or merely reflects lower visual familiarity or higher perceived dissimilarity between observer and model.

Here we sought to determine whether neurophysiological and autonomic indices of reactivity to others' pain are modulated by racial membership and racial bias. Based on the notion that racial stereotypes and prejudices are more readily observed at implicit rather than explicit levels, we focused on a very basic form of interpersonal reactivity called sensorimotor contagion [10–15], which is indexed by an automatic reduction of the corticospinal excitability of onlookers who observe painful stimuli delivered to a stranger model. Using transcranial magnetic stimulation (TMS), we explored changes in excitability of corticospinal body representations in white-Caucasian (Italian) and black-African (born in Africa and living in Italy) participants who were asked to watch and pay attention to clips depicting (1) needles penetrating the right first dorsal interosseus (FDI) or (2) a Q-tip gently touching the very same hand muscle of stranger black or white models. Motor-evoked potentials (MEPs) to single-pulse TMS of the left motor cortex were recorded from the observers' right FDI (target) and abductor digiti minimi (ADM, control) hand muscles (see [Supplemental Experimental Procedures](#) available online).

Watching painful stimuli administered to the ingroup but not to the outgroup models brought about a reduction of MEP amplitude that was specific for the muscle that the participants observed being stimulated. A preliminary onlooker group (white, black) × muscle (FDI, ADM) × model (ingroup, outgroup) mixed-model analysis of variance (ANOVA) performed on MEP difference (pain – touch) revealed no main effect or interaction involving the factor onlooker group ($F_s < 0.32$, $P_s > 0.59$; [Figure S1A](#)), indicating that blacks and whites showed the same MEP modulation. Hence, data were collapsed across this factor. The muscle × model repeated-measure ANOVA on MEP difference disclosed a significant main effect of model ($F_{1,34} = 8.21$, $p = 0.007$) and, most importantly, a model × muscle interaction ($F_{1,34} = 4.50$, $p = 0.041$; [Figure 1](#)); this was entirely accounted for by the greater inhibition recorded from the FDI muscle during observation of ingroup rather than outgroup models ($p = 0.002$). No difference between ingroup and outgroup models was found for the ADM muscle that was not involved in the painful stimulation ($p = 0.2$).

These findings indicate that seeing pain in members of the same racial group induced a reduction of corticospinal excitability that was specific to the muscle that participants

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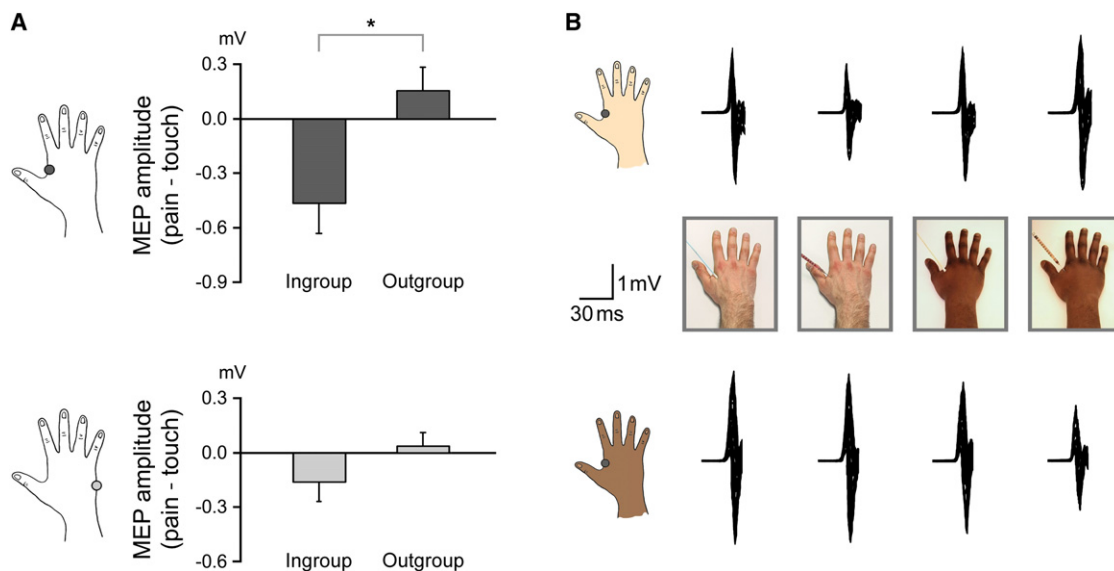


Figure 1. Neurophysiological Evidence of Sensorimotor Contagion

(A) Mean motor-evoked potential (MEP) difference (pain – touch) recorded from the first dorsal interosseus (FDI) (dark gray) and the abductor digiti minimi (ADM) (light gray) muscles during the observation of stimuli applied to the ingroup and the outgroup models. The asterisk denotes significant post hoc comparison. Bars indicate standard error of the mean (SEM).

Observing ingroup but not outgroup models' pain led to resonant inhibition of the FDI muscle that was stimulated in the models: one-sample t tests confirmed that MEP contrasts (pain – touch) for the FDI (target) muscle were significantly different from 0 for the ingroup ($t_{34} = -2.8, p = 0.007$) model, but not for the outgroup ($p = 0.3$) model. For the ADM (control) muscle, MEP contrasts were not different from 0 for either model ($P_s > 0.2$; see also Table S1).

(B) Raw MEPs recorded from the FDI muscle in a white (top) and a black (bottom) representative subject.

observed being penetrated. Thus, similar to real pain [21, 22], watching stimuli supposedly painful to others induces a specific corticospinal inhibition, hinting at the presence of a resonant activation of pain representations in the onlooker's sensorimotor system [10–15, 30–34] (see Supplemental Discussion). Notably, although both whites and blacks provided similar ratings of the level of pain ascribed to the two models (Table S2), sensorimotor contagion, as indexed by corticospinal inhibition, was selective for the ingroup models' pain and absent for the outgroup models' pain.

After subjective evaluation of models' pain, participants completed the Interpersonal Reactivity Index (IRI) [35, 36], a questionnaire assessing empathy-related dispositions. Blacks and whites scored similarly on both cognitive and emotional empathy subscales of the IRI (Table S3). Participants who scored high on the fantasy scale (a cognitive empathy subscale assessing the tendency to imaginatively transpose oneself into others' feelings and actions) showed greater sensorimotor contagion for the ingroup model (see Supplemental Results), in keeping with the notion that cognitive empathy may shape sensorimotor resonance [14, 15, 37].

Crucially, the ingroup-specific pain embodiment paralleled the implicit preference for ingroup members, as revealed by a race version of the Implicit Association Test (IAT) [16, 17]. The race IAT measured the relative ease with which participants made associations between Italian and African ethnic groups and the concepts of good and bad. Studies indicate that racial bias is more readily observed at implicit rather than explicit levels, possibly because explicit measures offer greater opportunities to regulate the expression of bias [16–20]. In keeping with this, only one participant showed explicit racial bias during a postexperimental ad hoc interview [38, 39] (see Supplemental Experimental Procedures); despite

the lack of explicit racial bias in our sample, the race IAT revealed a clear preference for ingroup relative to outgroup members in both white and black subjects. The IAT D index [16, 17] was significantly different from zero in white (Italian) participants (mean D \pm standard error of the mean: 0.65 ± 0.07 ; $t_{17} = 9.77, p < 0.0001$), indicating that they were quicker to associate concepts of good with the term "Italian" rather than with the term "African" and concepts of bad with the term "African" rather than with the term "Italian." A significant racial bias effect was found also in black (African) participants (0.10 ± 0.07 ; $t_{17} = 2.19, p = 0.043$), indicating that they more quickly associated concepts of good with the term "African" rather than "Italian" and concepts of bad with the term "Italian" rather than "African."

Implicit preferences emerge early in life and are largely impervious to cortical control [18]. Studies indicate that implicit ingroup preferences are sensitive to the sociocultural advantages of a given social group being weaker in numerical minorities (e.g., black people in the USA), possibly because of the cultural dominance of the majority [18]. In keeping with this, the race IAT in this study disclosed a stronger bias in white than in black participants ($t_{34} = 6.71, p < 0.001$). However, the neurophysiological marker of reduced sensorimotor empathy for the outgroup models' pain was completely symmetrical in the white and black participants. This suggests that sensorimotor contagion may index the tendency to develop group preferences at levels of processing even more basic and implicit than those indexed by the IAT. Importantly, participants with higher implicit ingroup preference presented greater differences in the corticospinal reactivity to ingroup and outgroup models' pain ($r = -0.46, p = 0.005$). This effect was similarly present in both white ($r = -0.50, p = 0.033$) and black ($r = -0.71, p = 0.002$; Figure 2) participants.

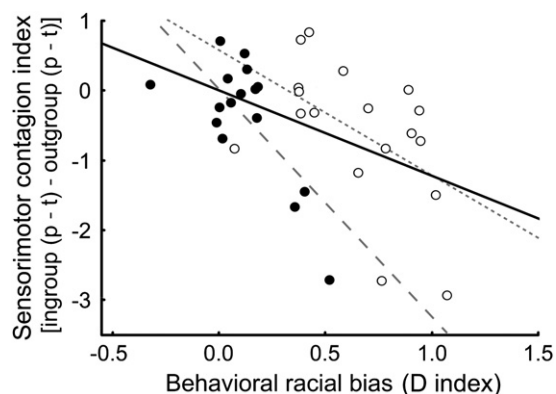


Figure 2. Correlation Analysis between Behavioral and Neurophysiological Markers of Racial Bias

The regression lines indicate the correlation of the entire sample (thick line, $r = -0.47$, $p = 0.005$), the black subjects (stippled line, $r = -0.71$, $p = 0.002$), and the white subjects (dotted line, $r = -0.50$, $p = 0.033$), respectively. Negative correlations indicate greater sensorimotor response to ingroup relative to outgroup models' pain in those subjects who scored high on the race Implicit Association Test. White and black dots indicate whites and blacks.

Our findings suggest a tight link between sensorimotor contagion and implicit race-related preferences. This link discloses social sensitivity in the human sensorimotor system and indicates that markers of social categorization can be found at basic sensorimotor levels of brain processing. This may be particularly novel because most of the existing studies on race perception have reported that racial bias modulates brain regions recruited during affective or cognitive control processing [19, 20].

Although sensorimotor response to others' pain was modulated by racial membership, a comparable increase in skin conductance responses (SCRs) was found when participants observed ingroup and outgroup members' pain (Figure S2A). This suggests that, regardless of the race of the model, in both groups similar emotional responses were evoked by seeing others' pain. Moreover, emotional and cognitive dispositional empathy predicted emotional response to the pain of the two models, further suggesting that ingroup and outgroup members' pain elicited a similar emotional empathic response (Supplemental Results). It is relevant that, although the magnitude of changes in SCRs was comparable for both models, the latency of SCRs was lower for ingroup than for outgroup models (Figure S2B). Thus, although comparable in size, emotional reactivity to outgroup models' stimulations (touch and pain) emerged later than that of ingroup models' stimulations, suggesting that empathic emotional reactions are more immediate for same-race members.

In a similar vein, watching both models experiencing pain brought about a small but significant reduction of heart rate (HR), indicating an orienting response to pain stimuli regardless of the race of the models (Figure S2A). However, HR was lower when watching ingroup rather than outgroup models in an early time window (Figure S2B). This indicates that watching ingroup members being stimulated (both with needles and Q-tips) was associated with an immediate greater orienting and attentional response than seeing outgroup members receiving the same painful and innocuous stimulations. This early differential orienting response to same- versus other-race members was greater in those participants who scored

high on the race IAT (Supplemental Results), suggesting that people with high racial bias process stimulations occurring to the body of ingroup members as more salient events than stimulations occurring to outgroup members.

Notably, earlier emotional reactivity (as indexed by SCR latencies) and greater orienting responses in the early time window (as indexed by changes in HR) were found for ingroup models regardless of the stimulation conditions (touch or pain), suggesting a general temporal advantage in autonomic response to physical events occurring to ingroup relative to outgroup members (see Supplemental Discussion). Therefore, emotional reactivity may imply compassion for the pain of outgroup individuals without impinging on sensorimotor resources. By contrast, sensorimotor resonance may imply that what is observed in others is mapped onto the onlookers according to body-specific, fine-grained coordinates. This process may allow onlookers to intuitively grasp what it feels like to sense similar pain on their own body [5–7, 10–15, 34]. Taken together, the results support the notion that perceiving bodily stimulations on ingroup members leads to an immediate resonance with affective and sensorimotor components of the observed feelings. In contrast, responses to outgroup members' somatic stimulations are less embodied and automatic and likely rely more on slower controlled processing [19, 20]. This view is also in keeping with a recent fMRI study showing in Chinese and Caucasian individuals that neural activity in the anterior cingulate cortex, which is part of the affective division of the pain matrix, was lower when viewing painful stimuli applied to the face of outgroup than of ingroup members [9]; by contrast, lateral frontal regions, more classically involved in controlled processing, showed no differential response to the pain of ingroup or outgroup models [9].

Our study significantly expands previous knowledge by demonstrating that the differential pain-specific empathic brain responses to ingroup and outgroup pain are linked to implicit racial bias. Studies suggest that sensorimotor regions may map physical [40] and cultural [41] similarity. Thus, the selective embodiment of ingroup members' pain may simply be due to higher model-observer somatic similarity or familiarity of ingroup individuals [9, 19] rather than to any ethnicity-related bias.

To further explore this fundamental issue, we tested a subgroup of participants with TMS in two additional conditions in which pain or tactile stimuli were delivered to a violet-colored hand (Figure 3A), which defined no racial group. Note that the violet model was judged as the most unfamiliar and dissimilar by both white and black onlookers (Figure 3B), as revealed by visual analog scales (VAS) collected at the end of the experiment (Supplemental Results).

Although the violet model was perceived as the least familiar and physically similar to the self, the MEPs analysis suggests an empathic sensorimotor response to this model. The onlooker group \times muscle \times model ANOVA on MEP differences (pain – touch) revealed no significant main effect or interactions involving the factor onlooker group ($F_s < 4.29$, $P_s > 0.08$; Figure S1B). Hence, the data were collapsed across groups. The muscle \times model ANOVA on MEP differences revealed a main effect of model ($F_{1,14} = 7.05$, $p = 0.008$) and a marginally significant model \times muscle interaction ($F_{1,14} = 3.33$, $p = 0.065$; Figure 3C). The inhibition found in the FDI muscle for the ingroup models' pain was greater than the inhibition found for the outgroup ($p = 0.004$) and the violet ($p = 0.02$) models' pain; moreover, the inhibition was greater for the violet

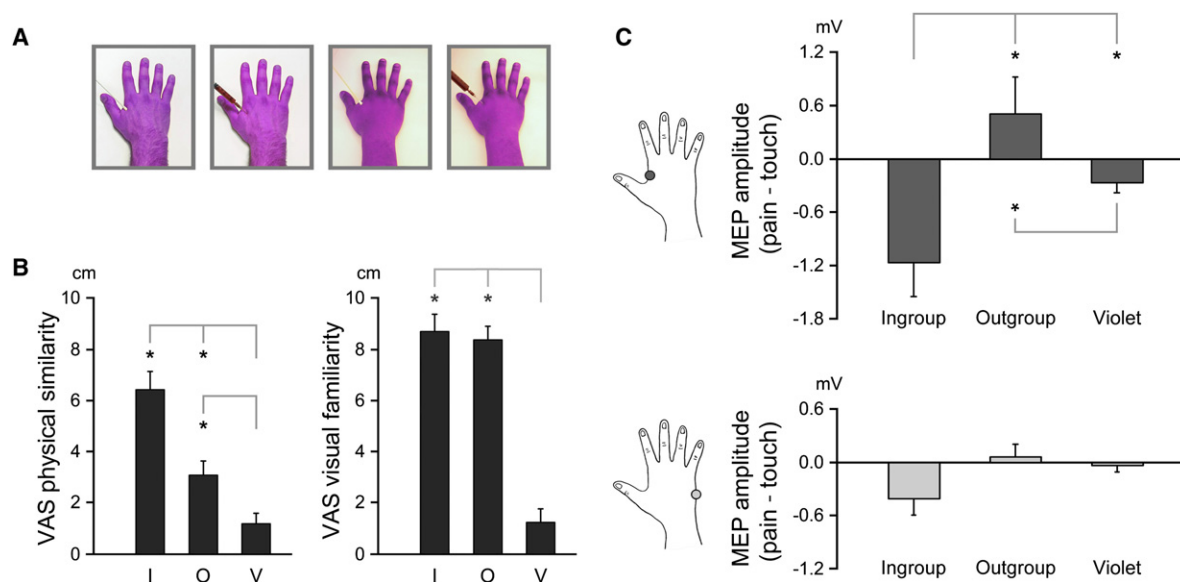


Figure 3. Sensorimotor Contagion of Ingroup, Outgroup, and Unfamiliar Violet Models' Pain

(A) Examples of touch and needle in violet models, to which racial prejudices and stereotypes did not apply.

(B) Subjective ratings (visual analog scale, VAS) of visual familiarity and of physical similarity of the observed hand with respect to the self hand. Subjective ratings indicate that the violet model (V) was judged as more unfamiliar and dissimilar than the ingroup (I) and the outgroup (O) models. Asterisks denote significant post hoc comparisons. See Supplemental Results for details concerning the statistical analysis of VAS data. Bars indicate SEM.

(C) MEP difference (pain – touch) in the subgroup of onlookers tested during observation of ingroup, outgroup, and extremely unfamiliar violet models. Ingroup and outgroup data presented in this figure are a subset of the data from Figure 1. Asterisks denote significant post hoc comparisons. Bars indicate SEM.

Sensorimotor contagion was found for both ingroup and violet models, but not for the outgroup model: one-sample *t* tests on MEP contrast (pain – touch) recorded in the target FDI muscle were significantly different from 0 for the ingroup ($t_7 = -3.1, p = 0.02$) and the violet ($t_7 = -2.3, p = 0.05$) models, but not for the outgroup model ($t_7 = 1.2, p = 0.3$). For the ADM (control) muscle, MEP contrasts were not different from 0 for any of the models (all one-sample *t* tests: $P_s > 0.1$).

than for the outgroup model's pain ($p = 0.05$). No significant modulation was found in the ADM muscle ($P_s > 0.2$).

The outgroup model was perceived as more familiar and similar to the self than the violet model, which represented no racial group. However, a clear sensorimotor contagion was found for the latter but not for the former model. Therefore, the absence of pain resonant mapping cannot be explained by a reduction of observers' familiarity or by somatic similarity with outgroup members. Rather, in keeping with the results of the correlational analysis (Figure 2), this lack of embodied resonance is likely due to racial stereotype and prejudice effects. It should also be noted that the hand of the violet model presented to black and white observers was obtained by coloring the hand of white and black (outgroup) models, respectively. This may indicate that the color of the skin is more important than hand morphology in defining racial groups and modulating basic interpersonal reactivity. Overall, a clear sensorimotor contagion was found not only in response to the pain of stranger individuals belonging to the same racial group but also in response to the pain of stranger, very unfamiliar but not culturally grouped, individuals (violet models). By contrast, no sensorimotor contagion was found in response to the pain of individuals culturally marked as outgroup on the basis of the color of the skin of a nonfacial body part that did not express any specific emotion. The reported lack of empathic brain response to the pain of outgroup members seems to provide a neural foundation for the notion that race-related prejudices can shape social categorization and lead to dehumanized perception of different others

[23, 24]. Remarkably, however, finding sensorimotor contagion with the violet model suggests that lack of empathic reactivity to strangers is not an ineluctable necessity. Moreover, that the differential reactivity to ingroup and outgroup was predicted by racial bias suggests that cultural conditioning (e.g., racial stereotyping), rather than biological or structural factors (e.g., somatic similarity), may shape embodied resonance with others. Thus, the basic reactivity of human beings implies empathy with the pain of stranger individuals [3–7]. This reactivity may be maximal when the perceived similarity with the model is high (ingroup model) but is also present for very unfamiliar others if no stereotype can be applied to them (violet-hand model). Crucially, race bias and stereotypes (outgroup model) may change interpersonal reactivity into a group-specific lack of sensorimotor resonance [1, 2, 9, 23, 24].

Supplemental Information

Supplemental Information includes Supplemental Results, Supplemental Discussion, Supplemental Experimental Procedures, three figures, and three tables and can be found with this article online at [doi:10.1016/j.cub.2010.03.071](https://doi.org/10.1016/j.cub.2010.03.071).

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