Trends in Cognitive Sciences

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Feature Review

Neurocognitive Basis of Racial Ingroup Bias in Empathy

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Racial discrimination in social behavior, although disapproved of by many contemporary cultures, has been widely reported. Because empathy plays a key functional role in social behavior, brain imaging researchers have extensively investigated the neurocognitive underpinnings of racial ingroup bias in empathy. This research has revealed consistent evidence for increased neural responses to the perceived pain of same-race compared with other-race individuals in multiple brain regions and across multiple time-windows. Researchers have also examined neurocognitive, sociocultural, and environmental influences on racial ingroup bias in empathic neural responses, as well as explored possible interventions to reduce racial ingroup bias in empathic brain activity. These findings have important implications for understanding racial ingroup favoritism in social behavior and for improving interracial communication.

Highlights

Racial ingroup bias in empathy for individuals in pain is characterized by increased neural responses to perceived pain of same-race relative to other-race individuals across multiple brain regions and multiple time-windows, and is evident in multiple ethnic groups.

Racial ingroup bias in empathic brain activity is mediated by distinct neurobiological mechanisms related to same-race and other-race pain and is affected by sociocultural and physical environments.

Although ingroup bias in empathic brain activity has been widely documented, both laboratory manipulations and real-life interracial experiences can reduce racial ingroup bias in empathy by increasing empathic neural responses to other-race pain.

Racial Bias in Social Behavior and Empathy

Race is a socially constructed concept that categorizes people into ethnic groups according to perceived physical and behavioral characteristics, and associates same-race/other-race individuals with different values, social status, and distinct altruistic motivations [1,2]. Racial discrimination in social behavior, although disapproved of by many contemporary cultures, is ubiquitous. Racial bias in social decision making and behavior has been widely reported in news media, and research literatures have documented racial bias in social behavior, such as in medical care and criminal justice systems (Box 1). Racial bias in social behavior can be understood from a historical perspective [3] or interpreted in terms of sociopolitical factors (e.g., socioeconomic status) [4,5]. Nevertheless, as mental activity provides the proximate mechanism for social behavior, it is essential to understand the psychological and neural processes underlying racial bias in social decision making and behavior.

Over the past two decades, there has been much interest in exploring **empathy** (see Glossary) as a potential mechanism for racial bias in social behaviors. Empathy refers to the affective and cognitive processes involved in understanding and sharing the emotional states of others, which produces motivation for helping others in need [6–10]. Behavioral experiments based on self-reported social decision making and empathy have shown evidence for greater empathy for same-race than other-race individuals, and moreover, the racial ingroup bias in empathy (RIBE) is associated with racial bias in social behavior (Box 1). While the self-reported results are suggestive, it is difficult for solely self-reported studies to examine the broad existence of RIBE across different racial groups and different countries because self-reported results depend upon how local cultures tolerate blatant racial discrimination. Therefore, researchers require objective measures of RIBE to reduce the influences of dominant antiracial-bias cultures on the investigation of RIBE and its underlying mechanisms.

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 https://doi.org/10.1016/j.tics.2018.02.013

Box 1. Racial Discrimination in Social Decision Making and Behavior

Despite cultural values and social norms that counteract racial discrimination, research literatures have documented widespread racial discrimination in contemporary societies. Early studies revealed undertreatment of African-Americans and other racial/ethnic minorities in the US, in prescriptions and medication for various types of clinical (e.g., chronic and cancer) pain [110–118]. Recent studies further reported racial bias in pain treatment in the emergency department, such that black patients (both adults and children) were less likely to receive pain medication than white patients [118–121].

Racial bias in social decision making and behavior in the criminal justice system has also been documented. The analysis of early capital cases revealed that the presence of more white males on the jury dramatically increased the likelihood of a death sentence in 'black kills white' cases [3]. Examination of a more recent data set of felony trials in Florida between 2000 and 2010 revealed that juries formed from all-white jury pools convicted black defendants significantly more often than white defendants [122]. Literature reviews have further strengthened the conclusion that jurors often make harsher judgments of defendants from other racial groups and are more likely to give death sentences in cases involving black or Latino defendants and white victims [4,123].

What are the psychological underpinnings of racial discrimination in social decision making and behavior? A survey reported that physicians underestimate pain in black patients compared with other ethnicities [80]. Laboratory experiments based on self-reports suggest an association between racial bias in social behavior and racial ingroup bias in empathy (RIBE). It was discovered that, after watching videos of black and white patients' genuine facial expressions of pain, white undergraduates and nursing professionals reported both pro-white pain treatment decisions and pro-white feelings of empathy, and, moreover, the pro-white pain treatment bias was correlated with the pro-white empathy bias [81,82]. Similarly, after reading a passage involving a black or a white defendant in a criminal case, white university students reported greater empathy and assigned more lenient punishments towards the white than the black defendant [83]. These findings suggest that distinct empathy for same-race and other-race pain may contribute to racial discrimination of social behavior as a proximate psychological underpinning.

Fortunately, brain imaging research has contributed greatly to our understanding of the neural underpinnings of empathy and their associations with social behavior [11,12]. The methods and findings of the brain imaging approach to RIBE allow researchers to investigate how interracial relationships between a target and an onlooker modulate empathic neural responses when viewing same-race and other-race individuals' suffering. Social, cognitive, and affective neuroscience studies of RIBE have opened a new avenue toward the understanding of neurobiological underpinnings of racial bias in social behavior and shed new light on possible interventions aimed at reducing racial bias in social decision making and behavior.

First, this review gives a brief introduction to neuroimaging studies that have investigated brain activity related to empathy for individuals in pain. Second, brain imaging findings over the past decade that have revealed distinct patterns of empathic neural responses to same-race and other-race individuals' pain are reviewed. Third, brain imaging findings that uncovered cognitive/neurobiological mechanisms of RIBE and sociocultural/environmental influences on RIBE for pain are presented. Examples of brain imaging studies that have examined possible interventions to reduce racial ingroup bias in empathic brain activity are also presented. Finally, potential contributions of the findings to our understanding of social problems pertaining to race and future directions of brain imaging research on RIBE are discussed.

Brain Activity Engaged in Empathy

To examine neural correlates of understanding and sharing the emotional states of others, brain imaging studies of empathy for individuals in pain have focused on a few critical issues. These include whether and how brain activation differentiates between others' emotional states (e.g., pain versus neutral), whether responses to the pain of others and one's own pain share neural substrates, and whether and how neural responses to the pain of others are associated with the self-reports of one's own feelings and prosocial behavior. Both **fMRI** with high spatial resolution and **electroencephalography (EEG)/event-related potentials (ERPs)** with high temporal resolution have been used to identify empathic neural responses to the pain of others. A typical

Glossary

Anterior cingulate cortex (ACC): a frontal part along the middle line of the brain that surrounds the frontal part of the corpus callosum and is involved in various types of mental processes, such as error detection, conflict monitoring, first-hand and vicarious pain experiences.
Anterior insula (AI): a cerebral cortical region folded deep within the lateral frontal part of the brain that is engaged in multiple cognitive and affective processes, such as self-awareness, interpersonal experience, stress, and pain.
Blood oxygen level-dependent (BOLD) responses: a change of the relative levels of oxyhemoglobin and deoxyhemoglobin that can be detected using fMRI and is supposed to be associated with functional activity of neuronal populations underlying various mental processes.
Electroencephalography (EEG)/event-related potential (ERP): synchronous activities of neuronal populations engaged in specific psychological processing, which can be time-locked to stimulus events, can be recorded from electrodes over the scalp, and have high temporal resolution.
Empathy: the mental processes that mediate understanding and sharing other individuals' emotional states. Empathy has been observed in humans and other mammals, such as chimpanzees and rats, and is believed to mediate altruistic behavior.
fMRI: a noninvasive method for recording blood oxygenation level-dependent signals that have high spatial resolution and are used to examine brain responses associated with specific stimuli or tasks.
Ingroup favoritism: a pattern of behavior or mental (cognitive or affective) process that favors members of one's ingroup over members of an outgroup and is associated with intergroup conflict and prejudice.
Magnetoencephalography (MEG): a noninvasive method for recording magnetic fields with high temporal resolution that are produced by electrical currents occurring in the brain, using arrays of sensitive magnetometers such as SQUIDs

paradigm used in brain imaging studies of empathy for individuals in physical pain is to compare **blood oxygen level-dependent (BOLD) responses** or ERPs with video clips or photos of others' body parts when receiving painful or non-painful stimulation [13–18], video clips or photos of faces with painful or neutral expressions [19–21], or symbolic cues indicating others receiving painful or non-painful stimulation [22,23].

Consistent fMRI evidence has demonstrated increased neural responses to the physical pain of others in the **anterior cingulate cortex (ACC)**, supplementary motor area (SMA), **anterior insula (AI)**, second somatosensory cortex (SII), inferior parietal cortex, and amygdala [24,25]. Affective sharing and empathic neural responses to the pain of others have been observed very early during development [26,27]. EEG/ERP studies provide evidence that the amplitudes of both phase-locked and non-phase-locked electrophysiological responses are modulated by perceived physical pain in others and that such responses can take place as early as 150 ms after stimulus onset and be sustained in several later time-windows (Box 2).

Importantly, the neural circuit underlying empathy for individuals in physical pain partially overlaps with the neural circuit engaged in first-hand pain experience in the ACC and AI [23,25]. Similarly, imagining the pain of others and imagining one's own pain also show overlapping activity in the ACC and AI [28]. Neural responses to the pain of others and one's own pain can be reduced by placebo analgesia and these effects can be similarly blocked by the administration of the opioid antagonist naltrexone [29].

These findings suggest there are shared neural underpinnings of empathy for others in physical pain and of first-hand pain experiences, although patterns of functional connectivity between the key brain regions of the neural circuit might be different between first-hand pain experience and empathy for others in pain [30]. For example, viewing the social pain of others (e.g., observing others being excluded from a game [31–33], in the midst of a natural disaster

(superconducting quantum interference devices).

Medial prefrontal cortex (mPFC): the medial region of the prefrontal cortex, which is involved in social cognition; the dorsal part is engaged in mental state reasoning and the ventral part is engaged in self-reflection.

Oxytocin: a neuropeptide of nine amino acids produced in parvocellular neurons of the hypothalamus. Oxytocin is an evolutionarily ancient and conserved hormone and also functions as a neurotransmitter. Oxytocin has been implicated in important reproductive and adaptive functions in animal models, including sexual behavior and pair-bonding, and in social cognition and emotion in humans.

Transcranial magnetic stimulation: a method that produces a magnetic field via a coil to stimulate small regions of the brain, which has been widely used to investigate brain function.

Box 2. Time Course of Empathic Neural Responses

EEG/ERP studies investigated empathic neural responses by recording EEG and analyzing amplitudes of phase-locked ERPs and the power of non-phased-locked neural oscillations elicited by viewing other individuals' pain. ERPs in response to perceived painful simulations versus non-painful stimulations to others' body parts are characterized by increased amplitudes of a positive ERP component at 140–200 ms over the central area and of a long-latency positive component at 300–800 ms over the parietal region [18]. Viewing painful versus non-painful stimulations to other individuals' faces increases the amplitude of an earlier negative ERP component at 80–140 ms over the frontal lobe [50] and induces positive shift of ERP amplitudes at 280–340 ms [49]. ERPs in response to painful versus neutral facial expressions are similarly characterized by enlarged amplitudes of a frontal negative ERP component at 90–120 ms and of a frontal/central positive component at 120–180 ms and decreased amplitude of a central negative component at 200–300 ms [38,44–47]. ERP findings also reveal overlapping neural responses to first-hand experience of pain and empathy for the pain of others in the same time course (e.g., 200–300 ms) [124]. Children from 2 to 9 years of age exhibit decreased amplitude of a negative ERP component at 200–300 ms and increased amplitude of a positive component at 500–700 ms in response to the pain of others [125]. The mean ERP amplitude in the same time-window (e.g., 140–180 ms) in response to perceived pain can predict self-reported measures of both other's pain and one's own feeling of unpleasantness [18]. The ERP amplitudes to other individuals' pain are associated with onlookers' empathy traits [38] and are modulated by task demands [19,38], onlookers' social cultural experience [126], placebo analgesia [29], and intergroup relationships between targets and onlookers [40]. Non-phase-locked activity in response to perceived painful versus non-painful stimulation of others is characterized by increased theta band (3–8 Hz) event-related synchronization at 200–500 ms and decreased alpha band (9–14 Hz) event-related desynchronization at 200–400 ms [37,127]. Moreover, self-reported measures of both other's pain and one's own feeling of unpleasantness are positively correlated with theta band event-related synchronization but negatively correlated with alpha band event-related desynchronization in response to perceived pain. An EEG study also revealed similar activation patterns of alpha oscillations when participants were feeling sad and when they observed same-race (but not other-race) individuals feeling sad [128]. In conclusion, empathy for pain is supported by neural responses in multiple time-windows that predict subjective feelings and correlate with individuals' empathy traits.

[34], or in a stressful social situation [35]), not only activated brain regions mediating affective/sensory processing (e.g., AI, ACC, SII), but also brain regions underlying processes of mentalizing or theory-of-mind [e.g., the **medial prefrontal cortex (mPFC)**, posterior cingulate cortex].

To what extent do neural responses to the pain of others underlie our understanding and sharing of other individuals' painful feelings? Previous brain imaging findings demonstrate that activations in brain regions such as the ACC and AI can differentiate between painful or non-painful states of individuals. The overlapping activities underlying one's own pain and viewing the pain of others in these brain regions provide a neural basis of emotional sharing in onlookers and targets. Most importantly, it has been shown that neural responses to the pain of others can predict onlookers' feelings about this pain and they are associated with onlookers' empathy traits. fMRI studies have shown that ACC and AI activity in response to the pain of others are positively correlated with onlookers' evaluation of this pain [15,36], and onlookers' empathy traits predict the magnitude of ACC and AI activity to other individuals' pain [20,22,31]. EEG/ERP studies have also reported that the amplitudes of electrophysiological responses to the pain of others are correlated with the self-reports of other people's pain and one's own feelings of unpleasantness, and with onlookers' empathy traits [18,37,38], indicating a strong link between brain responses to the pain of others and the self-reporting of empathy. There is also increasing evidence for the coupling between empathic neural responses and onlookers' prosocial behaviors (Box 3); this is consistent with the psychological account of the functional role of empathy in prosocial behavior [6–10].

Taken together, although empathic neural responses are modulated by multiple factors such as attention and attitude (Box 4), previous brain imaging findings have demonstrated a strong coupling between empathic neural responses and shared feelings in onlookers and targets,

Box 3. Empathic Neural Responses and Behavior

As behavioral studies have suggested a key functional role of empathy in altruism [6,7], one would expect that empathic neural responses should predict altruistic behavior. This is indeed observed in a number of brain imaging studies. One fMRI study identified participants' empathy-related activity in the AI and mPFC when viewing others being excluded from a game and asked them to email the excluded victims [31]. Prosocial behaviors toward the victims were estimated by examining how much participants tried to support, comfort, and help the victims through their emails. It was found that the AI/mPFC activity correlated with prosocial behavior and the mPFC activity mediated the link between trait empathy and prosocial behavior. Another fMRI study recorded brain activity in response to the pain of others and then invited participants to make anonymous monetary donations to a charitable organization and found that stronger neural responses to perceived pain in the SII and inferior frontal cortex predicted greater monetary donations in participants who reported high subjective socioeconomic status [129]. It was also found that septal activity during empathy for other individuals' pain or anxiety predicted daily helping, such as giving directions and lending/giving money to others [130].

The link between empathic neural responses and altruistic behavior is modulated by intergroup relationships between onlookers and targets. When soccer fans witnessed an ingroup member or an outgroup member experience pain, their AI activity to ingroup (versus outgroup) members' pain correlated with their decisions of helping ingroup (versus outgroup) members [39]. The mPFC activity in response to same-race suffering predicted altruistic motivation for one's own racial ingroup [34]. These brain imaging findings support the proposition that empathy provides a proximate mechanism of prosocial behavior [6,7,11].

Interestingly, a recent study showed evidence for a link between empathy and onlookers' simple actions, even when those actions did not produce any altruistic effect [88]. It was found that participants pressed a button with greater response force when watching others receiving painful (versus non-painful) stimulations. Moreover, the action of button press without any altruistic effect significantly reduced empathic neural responses in the ACC/SMA and SII. Together, the findings indicate that neural responses to other individuals' pain drive actions with or without altruistic effects, which may possibly in turn lead to relief of one's own distress induced by viewing other individuals' suffering.

Box 4. Modulations of Empathic Neural Responses

Apart from the effects of interracial relationships on empathic neural responses reviewed in the main text, other factors also significantly modulate brain activity underlying empathy for individuals in pain. For example, both fMRI and ERP studies showed that, relative to attention to pain-unrelated cues in stimuli, enhanced attention to other individuals' emotional states increased ACC activity and the amplitude of a long-latency frontal positive activity in response to painful stimulations [14,18]. To imagine oneself in a patient's situation also enhanced neural activities in the insula, ACC, and premotor areas when watching video clips of patients experiencing painful auditory stimulation due to medical treatment [131]. By contrast, increasing cognitive load by asking participants to memorize numbers diminished empathic neural responses to other individuals' happiness, sadness, and anxiety in several regions related to empathy and social cognition (e.g., mPFC, TPJ, and amygdala) [130]. Soccer fans showed greater insular activity in response to an ingroup than an outgroup member's pain [39], suggesting that empathic brain activity is modulated by intergroup relationships between perceivers and targets [132]. Personal closeness alters brain activity related to empathy and mentalizing, such that observing a friend being excluded from a game activated the ACC and AI, whereas observing a stranger being excluded activated the mPFC, precuneus, and temporal pole [32]. Empathic neural responses are also modulated by attitudes toward others. For example, after witnessing a partner behave either fairly or unfairly, individuals showed decreased AI activity to perceived pain in those who played unfairly compared with those who played fairly because they did not like those who played unfairly [23]. Individuals also showed implicit negative attitudes toward people with AIDS and exhibited less ACC activity in response to their physical pain as compared with perceived pain in healthy controls [133]. Finally, professional experiences play a modulatory role in neural responses to other individuals' pain. Naive participants (but not physicians who practice acupuncture) showed empathic activity in the AI, ACC, and somatosensory cortex when observing animated visual stimuli depicting needles being inserted into different body parts, whereas physicians showed activations in the mPFC and TPJ involved in emotion regulation and theory of mind [36]. Taken together, the findings indicate that the human brain has evolved and developed empathic neural responses that are flexible to adapt to variations of cognitive, affective, and motivational changes that underpin complex social interactions. The flexible empathic brain activity provides a neural basis for social decision making and behavior toward different individuals and social groups.

and revealed neural underpinnings of empathy-induced helpful behavior. The studies provide methods for objective measures of empathy for same-race and other-race individuals in pain and for investigation of the neurocognitive basis of RIBE.

Racial Ingroup Bias in Empathic Brain Activity

Although human empathy drives prosocial behavior and social cooperation, people do not empathize with everyone's suffering equally. For instance, empathy is modulated by intergroup relationships between a target and an onlooker, such that people show dampened and disrupted empathic neural responses to soccer fans of an opposing team [39] or individuals with different religious beliefs [40]. Interracial relationships have established coalitions and alliances during evolution [41], thereby producing strong influences on multiple facets of human lives. Researchers have investigated the neural correlates of RIBE, extensively using fMRI and EEG/ERP. Owing to the lack of a 'neutral' racial group that can be used as a control condition, most of the previous neuroimaging studies defined RIBE for pain as increased empathic responses to perceived pain of same-race rather than other-race individuals. In this subsection, brain imaging findings obtained from different laboratories, that characterize the patterns of brain activity in response to perceived suffering of same-race and other-race individuals, are summed up. The relationship between implicit empathic neural responses and explicit self-reported evaluation of empathy in relation to same-race and other-race pain is also discussed.

fMRI Evidence for RIBE

The first brain imaging study of RIBE scanned Chinese and white college students in China, using fMRI, while participants viewed video clips showing faces of six Asian and six Caucasian models [16]. Each 3-s video clip depicted a face with a neutral facial expression, receiving either painful (needle penetration) or non-painful (touched with a cotton swab) stimulation applied to the left or right cheek of the models. Participants had to judge whether or not the model in each

clip was feeling pain by using a button press. To examine whether participants showed explicit racial bias in empathy, after scanning, participants viewed the video clips again and had to rate each model's pain intensity and their own feelings of unpleasantness induced by each video clip. The analysis of BOLD signals first revealed that watching painful versus non-painful stimulations being applied to the models significantly activated the ACC/SMA and the inferior frontal(IF)/AI cortex in both ethnic groups. However, the ACC/SMA activation was significantly decreased in response to painful stimulations applied to other-race than same-race models, and this effect was similarly observed in both Chinese and white students (Figure 1A), indicating RIBE in both ethnic groups. Interestingly, both subject groups gave higher ratings of pain intensity and feelings of unpleasantness for painful (versus non-painful) stimulations and self-reported measures did not differ significantly between same-race and other-race models. Thus, racial ingroup bias in empathic brain activity was evident regardless of the absence of self-reported estimation of RIBE.

Racial ingroup bias in empathic neural responses was further corroborated in a number of fMRI studies using various types of stimuli for different ethnic groups from different countries. For example, passive viewing of video clips of painful (versus non-painful) stimulation applied to Asian and Caucasian faces elicited greater activity in the ACC, AI, and somatosensory cortex for same-race, compared with other-race, models in white university students in Australia [42]. Similarly, passive viewing of video clips of painful (versus non-painful) stimulations to blacks' and whites' hands activated the left AI more strongly for same-race than other-race models in both blacks and whites in Italy [17] (Figure 1B). Performing race judgments on pictures of Asian and Caucasian faces with painful (versus neutral) expressions induced stronger ACC activity for same-race than other-race models in Chinese participants in China [19]. Viewing video clips of dynamic physical or social suffering of black and white models resulted in greater activity in response to same-race than other-race pain in the amygdala, precuneus, and temporoparietal junction (TPJ) in blacks and whites in South Africa [43]. Moreover, viewing photos showing naturalistic visual scenes depicting either blacks or whites in a painful (e.g., in the midst of a natural disaster) or neutral (e.g., attending an outdoor picnic) situation led to stronger activity in mPFC for same-race than other-race models in blacks in the US [34]. In addition, this activation pattern predicted a greater altruistic motivation for one's own racial ingroup. These fMRI findings demonstrate racial ingroup bias in empathic neural responses in multiple nodes of the empathy network and in multiple ethnic groups.

ERP Evidence for RIBE

Early EEG/ERP studies found that perceiving painful (versus non-painful) stimulations applied to other individuals' hands/feet resulted in neural responses as early as 150 ms after stimulus onset, and these effects occurred in multiple time-windows of the empathic neural responses (Box 2), indicating dynamic variations of empathy for the pain of others across time. To examine the time course of racial ingroup bias in empathic brain activity, EEG was recorded from Chinese students in China while they performed judgments of racial identity on each Asian or white face with painful or neutral expressions [38]. The ERP results first revealed that painful (compared with neutral) expressions increased the amplitude of a positive component at 128–188 ms (P2) after stimulus onset over the frontal/central regions (Figure 1C). The difference in the P2 amplitudes to painful versus neutral expressions was positively correlated with self-reports of feelings of unpleasantness induced by perceived painful expressions and dispositional traits of empathic concern. Moreover, the P2 amplitude was enlarged by painful versus neutral expressions of Asian (but not white) faces. The amplitude of a following negative component at 200–300 ms (N2) was decreased (or also positively shifted) by painful versus neutral expressions of Asian (but not white) faces. Racial ingroup bias in empathic neural

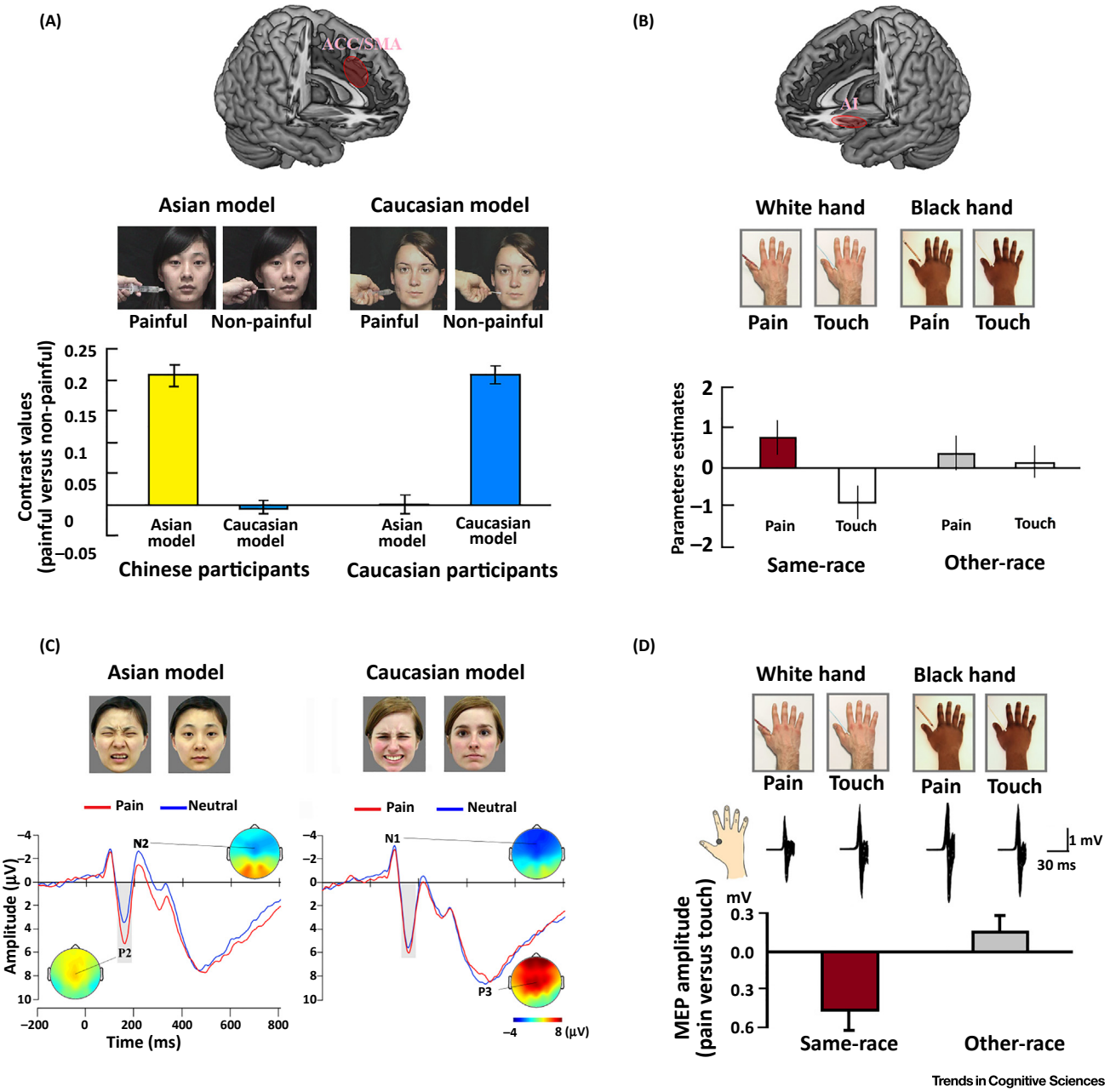


Figure 1. Racial Ingroup Bias in Brain Activity Underlying Empathy for Pain. (A) Racial ingroup bias in empathic neural responses in the anterior cingulate cortex (ACC) and supplementary motor area (SMA). The top figure illustrates the ACC/SMA in which the contrast value of painful versus non-painful stimuli is extracted. The middle panel illustrates video clips showing painful or non-painful stimulations of Asian and Caucasian models. The bottom panel shows the contrast values of painful versus non-painful stimuli in different conditions. Both Chinese and white students show greater ACC/SMA activation in response to same-race rather than other-race pain. Adapted, with permission, from [16]. (B) Racial ingroup bias in empathic neural responses in the left anterior insula (AI). The top figure illustrates the brain region in which parameter estimates of neural activity in response to painful versus non-painful stimuli are extracted. The middle panel illustrates video clips showing painful or non-painful stimulations of black and white hands. The bottom panel shows the parameter estimates of neural activity in response to painful and non-painful stimuli to same-race or other-race models. Both black and white participants show greater AI activity in response to same-race than other-race pain. Adapted, with permission, from [17]. (C) Racial ingroup bias in event-related potentials (ERPs) in response to painful versus neutral expressions. The top panel illustrates painful and neutral expressions of Asian and Caucasian models. The bottom panel shows ERPs in response to painful versus neutral expressions of Asian and Caucasian models. Chinese participants exhibit enlarged P2 amplitude in response to painful versus neutral expressions of Asian but not Caucasian models. The voltage topography reveals the

(See figure legend on the bottom of the next page.)

responses to painful expressions in P2 and N2 time-windows has been replicated in other studies of Asian and white participants [44–48]. The ERP results demonstrate modulations of empathic neural responses by target/onlooker interracial relationships in multiple time-windows, which obviously favor same-race individuals.

Perceiving painful versus non-painful stimulations applied to same-race and other-race faces also modulated empathic neural responses in multiple time-windows. White students in Italy showed decreased N2 amplitudes to painful (a needle penetrating the skin) versus non-painful stimulations (a cotton swab touching the skin) applied to white but not black models with neutral expressions [49]. The modulation of empathic neural responses by target/onlooker interracial relationships occurred in an even earlier time-window in whites in Australia, who showed larger amplitude of a frontal/central ERP component at 80–140 ms (N1) when perceiving painful versus non-painful stimulations applied to white but not Asian models [50]. Moreover, a minimal group manipulation that affiliated onlookers and other-race targets to one group could not reduce racial ingroup bias in empathic neural responses in the N1 time-window, suggesting stronger effects of interracial (versus minimal) ingroup relationships on empathic brain activity. Time-frequency analysis of EEG data also revealed that event-related desynchronization of beta band (13–30 Hz) neural oscillations at 300–1500 ms after stimulus onset was stronger in response to painful stimulation to same-race than to other-race hands in whites in Austria [51]. The EEG/ERP results are consistent with the reported fMRI findings by showing enhanced empathic activity of same-race compared with other-race pain in multiple time-windows.

Motor-Evoked Potential Evidence for RIBE

To investigate racial ingroup bias in sensorimotor responses to the pain of others, motor-evoked potentials to single-pulse **transcranial magnetic stimulation** of the left motor cortex were recorded from blacks and whites in Italy [52] to examine sensorimotor contagion: an automatic reduction of the corticospinal excitability of onlookers who observe painful stimulations delivered to others. The authors found that the excitability of corticospinal body representations, indexed by amplitude reduction of the motor-evoked potentials, decreased significantly when watching painful stimulations to same-race, compared with other-race, hands in both white and black participants (Figure 1D). This finding demonstrates greater sensorimotor contagion associated with same-race than with other-race pain and suggests greater sensorimotor resonance between same-race targets and onlookers.

Implicit versus Explicit RIBE

While the aforementioned neuroimaging studies reported evidence for racial ingroup bias in empathic brain activity, self-reported evaluation of empathy (e.g., explicit rating of same-race and other-race pain and one's own feeling of unpleasantness induced by the pain of others) did not always show racial ingroup bias in these studies ([16,17,19,38], for an exception, see [34]). Even in the same study, one racial group (i.e., blacks) showed RIBE in self-reported evaluation of empathy, whereas another racial group did not (i.e., whites) [43]. The dissociation between empathic neural responses and self-reported evaluation of empathy in racial bias is not surprising. Empathic neural responses occur quickly and implicitly, whereas self-reported

maximum P2 amplitude over the frontal/central region. Adapted, with permission, from [38]. (D) Racial ingroup bias in motor-evoked potentials (MEP) elicited by transcranial magnetic stimulation. The top panel illustrates video clips showing painful or non-painful stimulations of black and white hands. The middle panel shows MEPs recorded from a participant's hand in response to painful and non-painful stimulations as an index of the corticospinal excitability. The bottom panel shows the effect of reduction of the corticospinal excitability in black and white participants when observing painful versus non-painful stimulations of same-race but not of other-race hands. Adapted, with permission, from [52].

evaluation of empathy requires deliberate reasoning and explicit assertion of one's own feelings in response to other individuals' pain. It has been widely recognized that distinct implicit and explicit processes are involved in many aspects of cognition and emotion [53–57] and that different processes underlie changes in explicit and implicit attitudes [58]. People can be aware of explicit processes in social interaction but cannot always use them to override implicit processes [59]. In the case of RIBE, neural responses to the pain of others may reflect fast and implicit empathic processes, as indicated by the EEG/ERP findings (Box 2), whereas self-reported measures may depend on the deliberate and explicit empathic processes. This can explain the dissociation between racial ingroup bias in empathic neural responses and RIBE in self-reported measures. Alternatively, participants might consciously realize their RIBE but are unwilling to report it explicitly due to the dominant cultures that condemn racial discrimination. If this is the case, the dissociation between RIBE in brain activity and RIBE in self-reports implies that current dominant cultures regarding interracial attitudes can suppress explicit reports of one's own RIBE but cannot erase RIBE in brain activity. This reflects a stronger cultural influence on self-reported empathy for same-race and other-race pain than on the neural underpinnings of RIBE.

Mechanisms of Racial Ingroup Bias in Empathic Brain Activity

A core issue following the aforementioned neuroimaging studies of RIBE is to clarify the underlying mechanisms of racial ingroup bias in empathic brain activity. Infants are usually raised by same-race parents and their early experiences of perceiving the suffering of others are obtained from their interactions with same-race individuals. Perceiving other-race pain commonly occurs at a later stage of development when watching TV/movies or interacting with peers. The different time courses of perceiving same-race and other-race pain may result in different cognitive and neural strategies for processing same-race/other-race pain. From an evolutionary point of view, if ingroup bias in empathy benefits individuals' survival by driving altruism in same-race ingroup members, one should also expect influences of physical and sociocultural environments on RIBE. Furthermore, evolutionary pressure may associate specific genes with racial ingroup bias in empathic brain activity. Indeed, a number of studies have uncovered distinct cognitive, neurobiological, sociocultural, and environmental mechanisms underlying racial ingroup bias in empathic brain activity.

Cognitive Mechanisms

Negative attitudes towards other-race individuals may contribute to racial ingroup bias in empathic brain activity. Findings supporting this hypothesis include a study showing that lack of sensorimotor empathic reactivity to other-race pain was higher in onlookers with stronger implicit racial ingroup bias in attitude [52], tested using the Implicit Association Test [60]. Greater implicit racial bias in attitude also predicted increased AI activity in response to same-race pain relative to other-race pain [17].

The influences of prejudice on RIBE can be also mediated by the dampened individuation of other-race faces that has been well documented [61]. This was tested by recording ERPs in response to Asian and Caucasian faces with painful or neutral expressions from Chinese students in China, using an oddball paradigm that required responses only to scrambled faces [46]. It was found that the amplitude of the occipitotemporal N170, an ERP component sensitive to faces [62], was decreased for other-race compared with same-race faces and the P2 amplitude to pain (versus neutral) expressions was larger for same-race rather than other-race faces. Moreover, negative attitudes towards other-race individuals predicted the racial ingroup bias evident in the P2 amplitude, and this association was mediated by the racial ingroup bias in the N170 amplitude. These results suggest that the dampened individuation

processes of other-race faces (as indicated by the variation of N170 amplitude) may function as a possible intermediate mechanism of attitude influences on RIBE.

ERP findings also suggest a role of facial mimicry in favoring early empathic responses to same-race pain. Inhibiting facial mimicry by asking Chinese students to hold a pen horizontally using both teeth and lips to prevent facial muscle movement, significantly reduced the amplitude of a frontal ERP component at 100–120 ms (N1) to painful (versus neutral) expressions of same-race but not other-race faces [47]. This finding highlights a functional role of facial mimicry in RIBE.

Neurobiological Mechanisms

Increasing evidence suggests distinct neurobiological mechanisms underlying empathic neural responses to same-race and other-race pain. This was tested using a repetition suppression paradigm in which white and Chinese students recruited in China viewed two faces presented in rapid succession; the first adaptor face with a painful or neutral expression and the second target face with only a painful expression [45]. Recording ERPs to target faces helped to investigate whether different neuronal populations are engaged in coding same-race and other-race pain by examining how empathic responses to target faces are decreased by painful versus neutral expressions of the adaptor face (i.e., repetition suppress). If empathic responses to same-race and other-race pain are encoded by distinct neuronal populations, the repetition suppress effect related to a painful expression should occur only when adaptor and target faces are of the same race. Indeed, it was found that for both ethnic groups, the amplitude of the frontal P2 component at 140–200 ms in response to target faces was significantly decreased by painful versus neutral expressions of adaptor faces only when adaptor and target faces were of the same race (Figure 2A). This finding suggests that distinct neural assemblies are recruited for the processing of painful expressions of same-race and other-race faces in a specific time-window of empathic neural responses.

Empathic neural responses to same-race and other-race pain are also differentially sensitive to **oxytocin**: an evolutionarily ancient neuropeptide that functions as both neurotransmitter and hormone. An ERP study testing Chinese students found that intranasal administration of oxytocin (versus placebo) significantly increased the P2 amplitude to painful (versus neutral) expression of same-race but not to other-race faces [44], resulting in greater racial ingroup bias in empathic brain activity. A following fMRI study further suggests that neural responses to same-race and other-race pain are differentially associated with the two variants of the oxytocin receptor gene (*OXTR rs53576*) [63]. By scanning A/A and G/G homozygous genotypes of *OXTR rs53576* in a Chinese sample, it was found that G/G but not A/A carriers showed stronger ACC/SMA activity in response to painful stimulation applied to same-race than other-race models (Figure 2B). In contrast, A/A but not G/G carriers exhibited greater activity in the nucleus accumbens (NAcc) in response to painful stimulation of other-race rather than same-race models. Moreover, the racial ingroup bias in ACC/SMA activity positively predicted participants' racial ingroup bias in implicit attitudes, and the NAcc activity in response to racial outgroup individuals' pain negatively predicted participants' motivations to reduce racial outgroup members' pain. Together, the findings highlight distinct neurobiological mechanisms (e.g., distinct neuronal populations, neurotransmitter sensitivities, and genes) involved in empathic brain activity in response to same-race and other-race pain.

Sociocultural Influences

As **ingroup favoritism** in behavior is more prominent in collectivistic than individualistic cultures [64], one may expect stronger RIBE in samples dominated by collectivistic than

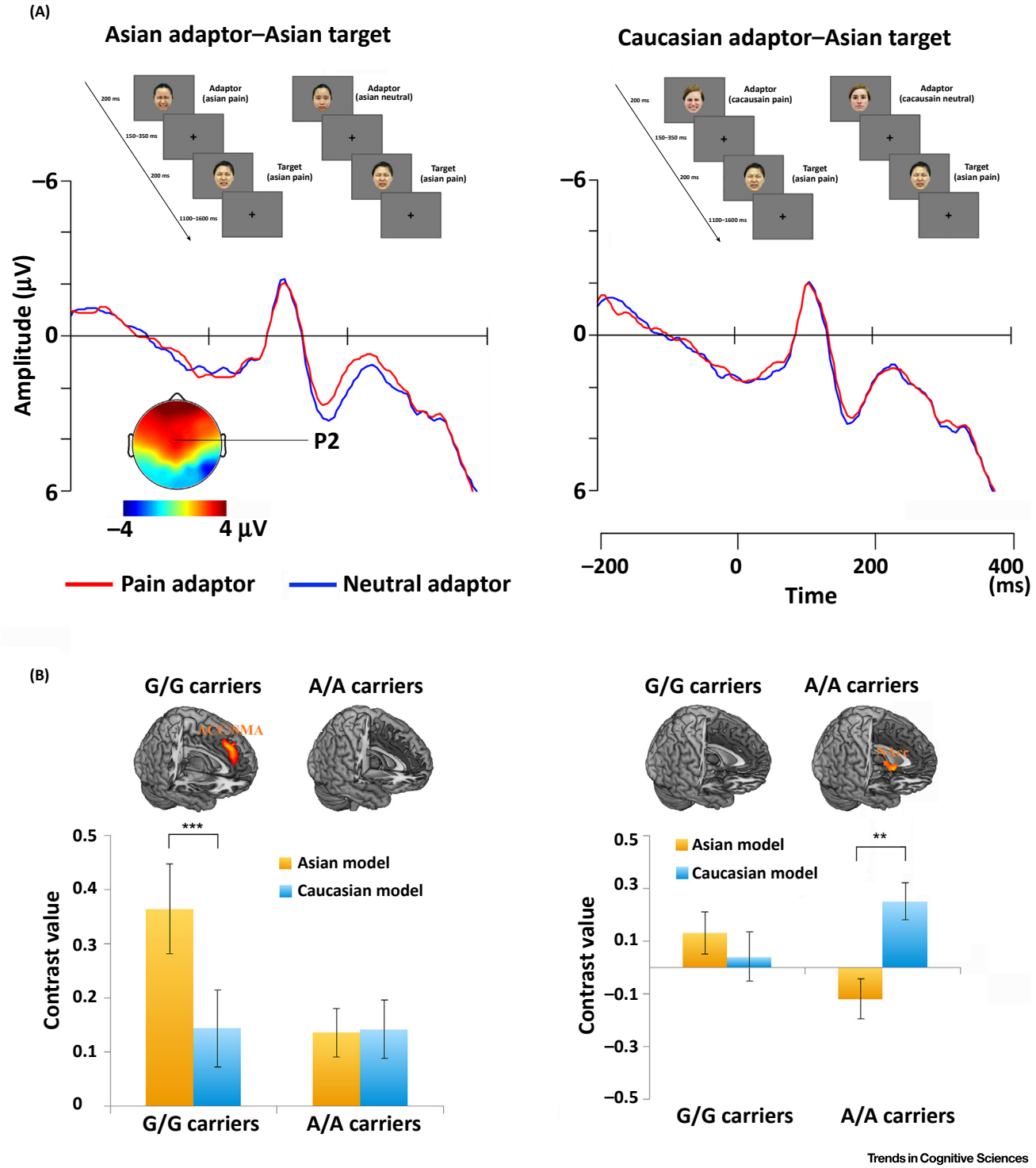


Figure 2. Neurobiological Mechanisms Underlying Racial Ingroup Bias in Empathy. (A) Repetition suppression effects on empathic neural responses in Chinese participants. The top panel illustrates the experimental procedure where an Asian target face with a painful expression is preceded by Asian or Caucasian adaptor faces with painful or neutral expressions. The repetition suppression effect is defined by the differential event-related potentials to target faces preceded by adaptor faces with neutral versus painful expressions. The bottom panel illustrates the repetition suppression effect in the P2 time-window that occurs only when

(See figure legend on the bottom of the next page.)

individualistic cultures. Consistent with this prediction, fMRI studies reported more salient racial ingroup bias in the mPFC activity in response to the suffering of others in African-Americans than in Caucasian-Americans [34], and greater racial ingroup bias in the TPJ activity in response to the suffering of others in Koreans from South Korea relative to Caucasian-Americans in the US. [65]. These findings are consistent with the idea that, relative to Caucasian-Americans, African-Americans [66], and East Asians [67] favor collectivism to a greater degree.

Additional evidence for a direct link between culture and RIBE came from a recent fMRI study demonstrating that Chinese students showed increased ACC/SMA and AI activity in response to painful (versus non-painful) stimulations of Asian compared with white models after being primed with interdependence (a cultural value emphasizing social connections) [68]. In contrast, priming participants with independence (a cultural value emphasizing one's own feeling and desire) significantly reduced the racial ingroup bias in empathic neural responses in these brain regions. The findings provide evidence for significant sociocultural influences on the brain activity underlying RIBE.

Environmental Influences

The finding of greater ingroup favoritism in behavior when coping with harsher climates [69] supports the proposal that an inclement environment with scarce resources threatens human survival and demands increased group affiliation and ingroup favoritism [70]. This finding also suggests increased RIBE in an inclement environment, which can be simulated in laboratories by inducing physical coldness (versus warmth), which has been shown to increase interpersonal distance [71]. The effect of cold versus warm environments on RIBE has been tested by recording ERPs to painful and neutral expressions of same-race and other-race faces from Chinese students who had to hold a cold (6°C) or warm (39°C) pack using the left hand [72]. Racial ingroup bias in empathic neural responses in the N2 (200–340 ms) and P3 (400–600 ms) time-windows over the frontal/central region was significantly enlarged in the cold compared with the warm condition. In addition, the increased racial ingroup bias in empathic neural responses was predicted by self-reports of the temperatures of cold (versus warm) packs, indicating a link between RIBE and subjective feelings of the environment.

Because the worst consequence of inclement environments would be the loss of human lives, researchers also examined whether making individuals think about this consequence (i.e., death) would increase racial ingroup bias in empathic brain activity. Reminders of death lead to increased group affiliation [73,74], which may then increase RIBE. Consistent with this, both fMRI and ERP evidence showed that asking Chinese students to think about death increased racial ingroup bias in empathic neural responses in the ACC and in the P3 time-window when viewing painful (versus non-painful) stimulations applied to same-race and other-race individuals [48]. Together, the findings suggest that RIBE can be increased in harsh environments that induce intergroup competition/conflict and threaten human lives.

Overcome Racial Ingroup Bias in Empathic Brain Activity

The neuroimaging evidence for RIBE raises other important questions (i.e., whether or not RIBE is inevitable and how to reduce RIBE). Because RIBE may play a key role in mediating pain

adaptor and target faces are of the same race. Adapted, with permission, from [45]. (B) Associations between OXTR and distinct empathic neural responses in the anterior cingulate cortex (ACC) and nucleus accumbens (NAcc) in a Chinese sample. The top panel illustrates ACC and NAcc in which racial ingroup bias in neural responses to video clips showing painful or non-painful stimulations applied to Asian and Caucasian models is evident in G/G and A/A allele carriers of *OXTR rs53576*, respectively. The bottom panel shows the contrast values in the ACC and NAcc in response to same-race and other-race pain in G/G and A/A variants, respectively. Adapted, with permission, from [63].

perception and pain treatment, it is important to find out how to reduce RIBE by modulating the underlying neural activity. Several studies have tried different manipulations to reduce racial ingroup bias in empathic brain activity by adjusting cognitive strategies, intergroup relationships, or interracial interactions.

Cognitive Intervention

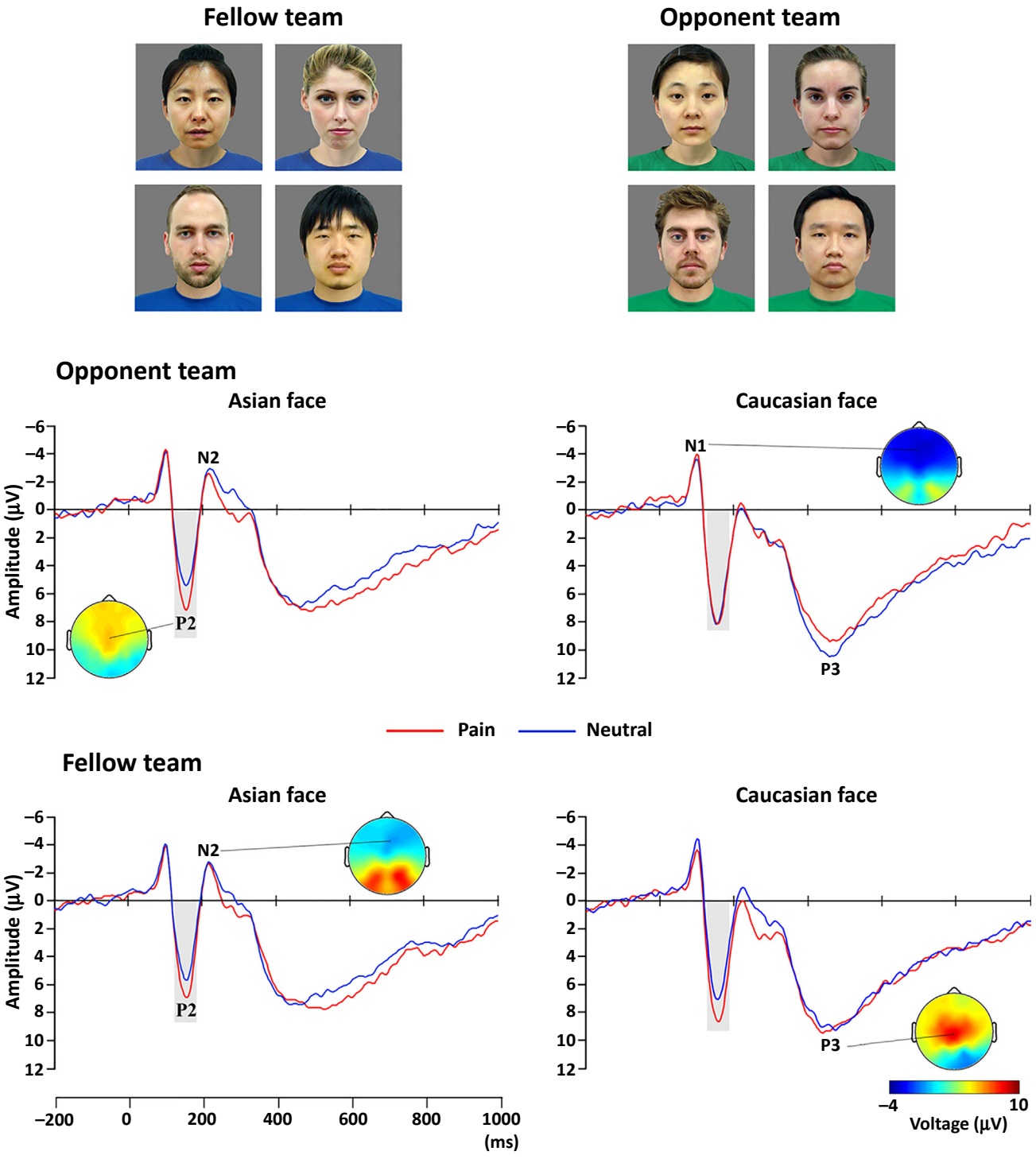
Since the processing of other-race faces is characterized by degraded individuation [61] and the decreased individuation processing of other-race faces contributes to racial ingroup bias in empathic brain activity [46], one may expect to reduce RIBE by enhancing attention to each other-race individual's painful feelings. This was tested by asking Chinese students to judge whether each individual Asian or white face with painful/neutral expressions was feeling pain during EEG recording [38]. This task instruction, which promoted individuation processing but weakened group identity processing of each face, eliminated racial ingroup bias in the P2 empathic response by increasing the P2 amplitude to painful expressions of other-race faces. An fMRI study testing Chinese students further revealed that instructions to pay attention to other individuals' pain versus their ethnicity increased their ACC/SMA and AI activity in response to painful expressions of other-race (Caucasian) models and activated their TPJ [19]: a key region engaged in taking others' perspectives and beliefs into account [75]. The results suggest that cognitive interventions can promote individuation processing of other-race faces by encouraging understanding of other individuals' perspectives, and such changes of cognitive strategy can enhance neural responses to other-race pain and, in turn, reduce RIBE.

Intergroup Relationship Manipulation

If RIBE essentially reflects the influence of intergroup relationship on emotional understanding and sharing, changing intergroup relationships between other-race individuals and oneself should reduce RIBE. An ERP study of Chinese participants tested this by examining the effects of a minimal group manipulation on empathic neural responses to same-race and other-race pain [38]. The minimal group manipulation made participants believe that they would join a team and play a game by working with both same-race and other-race team members to compete against an opponent team that also contained both same-race and other-race individuals. It was found that, while the difference in the P2 amplitude to painful versus neutral expressions was larger for other-race than same-race individuals from the opponent team, the P2 empathic response to others individuals' pain was comparable for same-race and other-race individuals from the fellow team (Figure 3). Thus, changing the intergroup relationships between onlookers and targets by enclosing other-race models into one's own team (i.e., one's own group) can weaken or eliminate RIBE.

Interracial Experience

Experiences of interacting with other-race individuals influence multiple aspects of our lives [76] and reduce differential brain responses to other-race/same-race faces [77]. To test whether experiences of interacting with other-race individuals also reduce RIBE, an fMRI study scanned Chinese students who were born and/or educated in the US, UK, and Canada, and thus had abundant experiences with interracial communication and interaction [78]. Empathic neural responses were assessed by contrasting brain activity in response to painful versus non-painful stimulation applied to Asian and white models. This study observed comparable ACC/SMA, AI, and somatosensory activity in response to same-race and other-race models' pain and the empathic neural responses to Asian and white models were positively correlated with each other. A further fMRI study of Chinese students, who had been living in Australia for 6 months to 5 years, further showed that, although the participants exhibited greater ACC activity in response to painful (versus non-painful) stimulation of Asian rather than white models, the



Trends in Cognitive Sciences

Figure 3. Decreased Racial Ingroup Bias in Empathy in a Minimal Group Manipulation. The top panel illustrates faces of fellow and opponent team members, indicated by t-shirt colors, whom participants had to remember before electroencephalography recording. The bottom panel shows event-related potentials in response to painful and neutral expressions of Asian and Caucasian faces from the fellow team and the opponent team, respectively. Chinese participants show comparable empathic neural responses in the P2 time-window to Asian and Caucasian faces of the fellow team but only to Asian faces of the opponent team. Adapted, with permission, from [38].

ACC activity in response to other-race pain was positively correlated with the self-reports of the overall level of experience with other-race individuals [79]. These studies suggest that the experience of interracial communication and interaction can increase affective responses to other-race pain, supported by the ACC and AI activity, and thereby reduce RIBE.

Concluding Remarks

The aforementioned sections summarize the brain imaging findings that demonstrate the presence of RIBE and uncover the neurocognitive underpinnings. A few issues related to RIBE that emerge from the summarized findings are discussed below. In addition, a theoretical model is proposed, that integrates social categorization and RIBE, to explain discrepant social decisions and behavior toward same-race and other-race individuals.

RIBE is Pervasive

Although studies employing self-reported measures showed limited evidence for RIBE [80–83], brain imaging findings summarized in this paper have shown consistent evidence for RIBE in multiple ethnic groups from different continents, including Asia, Europe, North America, Africa, and Australia. The RIBE reported in the fMRI/ERP studies is commonly characterized by increased empathic neural responses to same-race (versus other-race) pain regardless of variations in language, culture, history, economic development, and physical environments.

The pervasive and similar RIBE demonstrated in brain imaging findings may arise from the two key aspects of social living; that is, the need to coordinate and cooperate with same-race (ingroup) members and the need to compete against other-race (outgroup) members for resources. This can be traced back to the early time of human evolution, and to a certain extent, is still prevalent in current societies. Consistent with this, RIBE influences our lives in two opposite directions. One the one hand, fast and automatic empathic neural responses to same-race individuals' pain promote communication of emotional states and facilitate coordination and cooperation among ingroup members. On the other hand, degraded neural responses to other-race individuals' suffering result in apathy and facilitate intergroup aggression.

The pervasive neural underpinnings of RIBE may influence multiple facets of human lives, such as racial discrimination in clinical pain treatment. Given the connection between empathy and moral development [84,85], the distinct neural responses to same-race and other-race pain may also affect other social domains, such as judgments of criminal justice. If RIBE reflects the consequence of human evolution over hundreds of thousands of years in adaption to lives characterized by ingroup favoritism and outgroup degradation, it is then not surprising that human brains have evolved the distinct patterns of brain responses to same-race and other-race pain, as race is one of the strongest social markers used to categorize others into ingroups and outgroups.

However, relative to the long history of human evolution, antiracial-bias cultures have existed for a much shorter period of time. It is a challenge for human societies to consolidate and popularize the antiracial-bias culture in the hope of modifying and dampening the distinct patterns of empathic neural responses to same-race and other-race pain. Neural plasticity along with changes in sociocultural contexts might allow us to develop equal empathic neural responses to same-race and other-race pain, that in turn motivate unbiased altruistic behavior towards all individuals. Being aware of the pervasiveness of RIBE is critical for understanding human nature related to ingroup favoritism and intergroup conflict and is crucial for improving interracial relationships.

Outstanding Questions

How do the dynamic neural responses involved in perception, empathy, mental inference, decision making, and action execution related to other individuals' suffering drive prosocial behavior? How does the information-flow from one stage to the next contribute to prosocial behavior?

Are there distinct neuronal populations that encode same-race and other-race pain? If yes, where are these neurons located in the neural circuit underlying empathy? Are the distinct neuronal populations located in all the key nodes of the neural circuit of empathy or only in some of them?

Does racial ingroup bias in cognitive and affective processes of empathy share similar neurochemical mechanisms? Is oxytocin involved in racial ingroup bias in both cognitive and affective processes of empathy for individuals in pain? Do other neurotransmitters play a role in mediating distinct empathic neural responses to same-race and other-race pain?

Is racial ingroup bias in empathic brain activity associated with racial discrimination in attitude and social (e.g., prosocial and aggressive) behavior? Does racial ingroup bias in cognitive and affective components of empathy mediate racial ingroup favoritism in social behavior, and if so, how?

Does racial ingroup bias in empathic neural responses to physical pain and social pain mediate racial ingroup favoritism in social behavior via the same or different mechanisms?

Do laboratory manipulations and interracial experiences produce similar effects on racial ingroup bias in cognitive and affective components of empathy? What sort of real-life interracial interactions can efficiently reduce racial ingroup bias in empathic neural responses? Are modulations of racial ingroup bias in empathic neural responses associated with changes in social (altruistic/aggressive) behavior toward racial ingroup and outgroup members?

How can current brain imaging findings of racial ingroup bias in empathy help to develop social programs aimed at reducing racial ingroup favoritism in social decision making and behavior?

RIBE Permeates Both Cognitive and Affective Processes

Previous fMRI research has identified several (sensory, cognitive, and affective) components of empathy that engage distinct brain regions and networks, such as the SII, ACC, AI, and mPFC. ERP research also suggests associations of early and late empathic neural activity patterns with affective and cognitive components of empathy, respectively (Box 2). To date, brain imaging studies of RIBE have revealed modulations of ACC/AI activity related to both affective and cognitive components of empathy [16,17,19], sensorimotor activity involved in sensory and motor processing in response to the pain of others [52], and mPFC activity related to prosocial decisions [34,65] by interracial relationships between targets and onlookers. ERP studies also showed evidence for modulations of both early (sensory and affective) and late (cognitive) empathic neural responses by interracial relationships between targets and onlookers [38,44–49,72].

Empathic neural responses in different brain regions may correspond to different psychological processes. The SII activity is engaged in evaluation of sensory consequences of other individuals' pain [15] and of sensory-discriminative attributes of one's own physical pain [86]. The ACC and AI are engaged in automatic affective response to painful stimuli applied to both others [15] and oneself [87]. The SII and ACC activity in response to other individuals' pain also play a key role in affective motivation to drive onlookers' actions [25,88]. The mPFC is widely known for representations of the mental states of others (e.g., beliefs and intentions) [89,90] and seems to be specifically engaged in empathy for social pain and can predict altruistic behavior [31,34,91].

The brain imaging findings of RIBE indicate that interracial relationships between targets and onlookers generate broad influences on affective, cognitive, and sensorimotor processes involved in empathy. Notably, racial ingroup bias in brain activity underlying cognitive and affective processes was reported in different studies that employed different stimuli and tasks, suggesting that interracial relationships may bias cognitive or affective processes of other individuals' suffering, independent of stimuli and tasks. However, whether and how racial ingroup bias in cognitive and affective processing of empathy drives racial discrimination in social behaviors remains unclear and should be addressed in future research.

RIBE Is Malleable

Current neuroimaging findings have uncovered multiple (cognitive, neurobiological, sociocultural, and environmental) contributions to RIBE. This may not be surprising if one believes that RIBE reflects long-term adaptation to social interactions that favor ingroup members' benefits/survival, and thus requires multiple mechanisms that allow individuals to make fast and appropriate decisions during interracial interactions. However, the adaptive function of pervasive RIBE has been challenged by contemporary cultures that advocate equality, caritas, justice, etc., and condemn racial segregation and discrimination. In addition, the ease of international travel, demands of international cooperation, and large-scale immigration has greatly promoted interracial communication and interaction, which in turn may alter the psychological and neural processes related to representing the emotional states of same-race and other-race individuals.

Current brain imaging findings suggest that either manipulations in laboratories or real-life interracial experiences can alter the brain activity underlying RIBE. How do the brain imaging findings relate to social problems, such as racial bias observed in clinical treatments and jury judgments? Given the strong coupling between empathic neural responses and altruism, one might predict that the lack of empathic neural responses to other-race individuals' pain plays a

key role in producing these social problems, reflecting a consequence of long-term adaptation to interracial interactions on human brain and behavior. As shown in the brain imaging studies, racial ingroup bias in empathic neural responses occurs commonly, while conscious self-reporting often does not show RIBE. Making the public aware of the findings of RIBE in empathic brain activity should strengthen their understanding of the consequence of intergroup/interracial interactions on cognitive and affective processes. This understanding might in turn increase conscious efforts to counteract RIBE.

Additionally, the findings that RIBE in neural activity can be weakened or even erased through laboratory manipulations and real-life interracial interactions, provide a neuroscientific basis for developing intervention programs to mitigate racial discrimination in social behavior. Moreover, the findings suggest potential methods for intervention, such as changing cognitive strategies and intergroup relationships. The brain imaging findings have significant implications for reducing racial bias in social decision making and behavior, such as clinical pain treatment, jury decision making, interracial communication in education and cooperation, political/economic decisions regarding immigrants, and other important social issues. It is challenging to clarify how modifications of RIBE arising from laboratory manipulations and interracial interactions are associated with changes of social (altruistic/aggressive) behavior toward same-race and other-race individuals in everyday life.

Future Direction

RIBE summarized in this paper is only one aspect of distinct cognitive and affective processes of same-race and other-race individuals. Race also modifies other domains of cognition and emotion underlying social interactions, such as perception [92,93], memory (i.e., the own-race bias in memory of faces) [61,94], attitude/prejudice [95,96], stereotype [97–99], and imitation [100]. To date, social neuroscientists have been searching for distinct neural substrates underlying these cognitive and affective processes when interacting with same-race and other-race individuals [101–104]. However, most of the previous studies that focused on racial influences on a specific domain of cognition and emotion have uncovered overlapping neural underpinnings.

For example, perception and categorization of race engage the amygdala, ACC, fusiform gyrus, and orbital frontal cortex (OFC) [101,105,106]. Prejudice and stereotyping related to race engage the amygdala, ACC, AI, mPFC, and OFC, and regulation of prejudice and stereotyping recruits the lateral prefrontal cortex [101,103]. As summarized in this review, the activity in some of these brain regions (e.g., ACC and AI) related to empathy for individuals in pain also demonstrates modulations by interracial relationships. A novel emerging trend in social neuroscience, which is pivotal for understanding racial bias in social decision making and behavior, is to construct a neural model which integrates the neural circuits that have been demonstrated to function in the processing of race in different domains.

Two conceptual models that integrate different domains of race processing and characterize the asymmetric processing of same-race and other-race individuals are suggested on the grounds of previous psychological and neuroscientific findings. As illustrated in the asymmetric race processing (ARP) models in Figure 4, the processing of same-race individuals is characterized by enhanced processing of personal identity and emotion but weakened processing of group identity and related prejudice/stereotype (Figure 4A). The enhanced processing of personal identity and emotion, in turn facilitates empathy for same-race individuals' pain and, together with representing the intentions and beliefs of others, promotes altruistic decision making and behavior toward racial ingroup members. By contrast, the processing of other-race

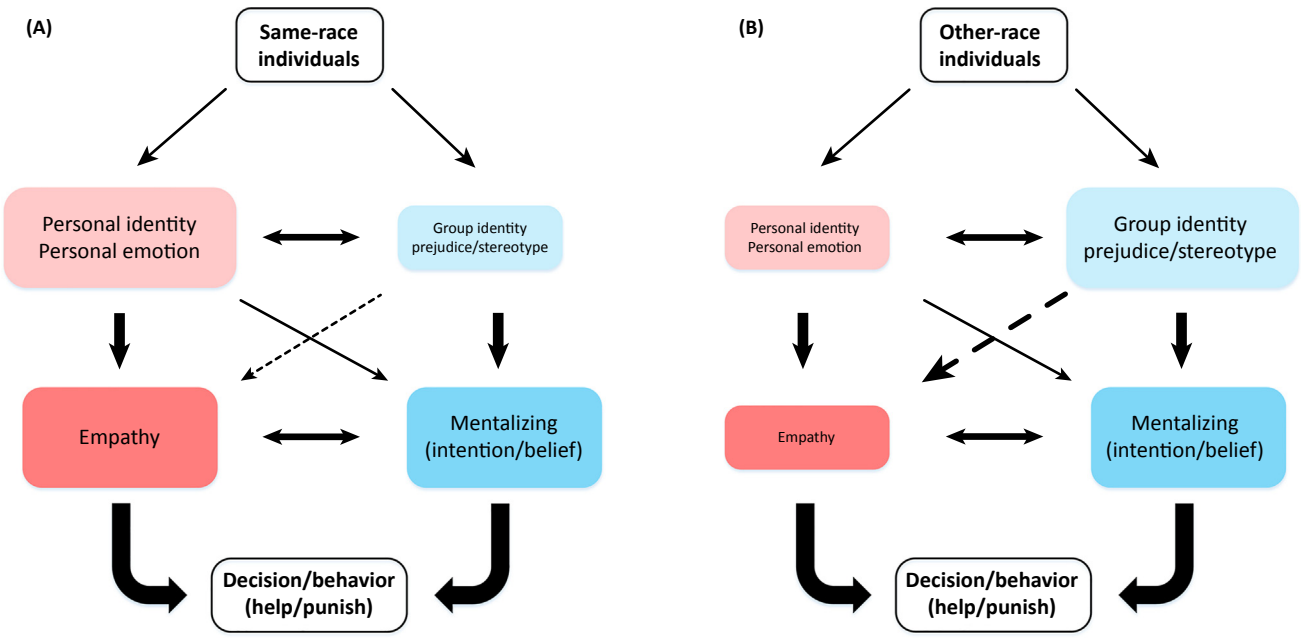


Figure 4. Illustration of the Asymmetric Race Processing Models. (A) shows the model for same-race individuals, and (B) shows the model for other-race individuals. Enhanced (or weakened) processes of person identity/emotion or group identity/prejudice/stereotype are illustrated by large (or small) shapes. The filled, one-direction arrows indicate feed-forward processing. The filled, two-direction arrows indicate mutual interactions between two modules. The dashed, one-direction arrows indicate inhibition processing, which is stronger for other-race than same-race individuals.

individuals is characterized by enhanced processing of group identity and related prejudice/stereotypes but weakened processing of personal identity and emotion (Figure 4B). Thus, although the processing of the intentions and beliefs of others continue to play a key role in behavior towards racial outgroup members, the enhanced processing of group identity and the activation of prejudices/stereotypes consistent with that group identity dampens empathy for other-race pain.

The ARP models proposed here provide a framework for future studies to investigate how the neural circuits involved in different domains of race processing interact with each other to guide social decision making and behavior. A key issue related to RIBE is to clarify how the neural circuits involved in racial categorization and prejudice connect and modulate the neural circuit underlying empathy for same-race and other-race pain. It is also challenging to combine different neuroimaging methods with high spatial resolution (e.g., fMRI) and high temporal resolution [e.g., EEG/ERP and **magnetoencephalography (MEG)**] to examine how the same set of brain regions are involved in different domains of race processing through dynamic activations and connections across time.

Finally, ingroup bias in empathy is evident in Asians, whites, and blacks, as summarized in this review; racial group identities are defined by physical markers such as skin tone that can be easily perceived. Cultural heritage and sociopolitical relationships likewise contribute greatly to formation of social group identity [2], such as Jewish-Israeli and Arab-Palestinian. In such cases, group identity also modulates empathy and compassion for the suffering of others. It has been shown that Arab and Israeli adult immigrants in the US reported significantly less compassion for each other's pain and suffering [107]. Americans, Hungarians, and Greeks

reported greater empathy for their ingroup than outgroup (Arabs or Germans), and this 'parochial empathy' predicted self-reports of intentions to support or help the outgroup [108]. A recent MEG study found that Arab-Palestinian adolescents expressed less empathic behavior toward their Jewish peers and their behavioral empathy was correlated with brain-to-brain synchrony [109]. The findings suggest prevalence of ingroup bias in empathy for other individuals' suffering, regardless of whether group identity is defined by physical markers or cultural heritage. Future research should examine whether the conceptual models proposed here can be similarly applied to the processing of ingroups and outgroups formed by physical markers versus cultural heritage. In addition, as group conflict usually characterizes sociopolitical intergroup relationships, it is important to investigate how the neural circuits involved in the models in Figure 4 are modulated by intergroup conflict. A comprehensive understanding of these issues will expand contributions of neuroscientific research to address social problems related to interracial communication and behavior.

Acknowledgements

This work was supported by the National Natural Science Foundation of China (Projects 31661143039, 31470986, 31421003). The author is grateful to Daniela M. Pfabigan, Yina Ma, and Elizabeth R. Losin for proof reading of the manuscript, and to Feng Sheng and Xiaochun Han for help with modification of the figures.

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