

Feature Review

Neurocognitive Basis of Racial Ingroup Bias in Empathy

Shihui Han^{1,*}

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

Box 1. Racial Discrimination in Social Decision Making and Behavior

Despite cultural values and social norms that counteract racial discrimination, research literature have documented widespread racial discrimination in contemporary societies. Early studies revealed under treatment of African-Americans and other racial/ethnic minorities in the US, in prescription 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 departments 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 dataset 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?

Fortunately, brain imaging research has contributed greatly to our understanding of the neural underpinnings of empathy and the 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 in 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 group 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 other's 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 stimulus 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 non-painful stimulation [13-18], video clips or photos of faces with painful neutral expressions [19-21], or symbolic cues indicating others receiving painful 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-phase-locked neural oscillations elicited by viewing other individuals' pain. ERPs in response to perceived painful stimulation versus non-painful stimulation 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 stimulation to other individuals' faces increases the amplitude of an early negative ERP component at 80-140 ms over the frontal lobe [50] and induces a 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 others' 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 others' pain and one's own feeling of unpleasantness are positively correlated with the 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 not

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. Naïve 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

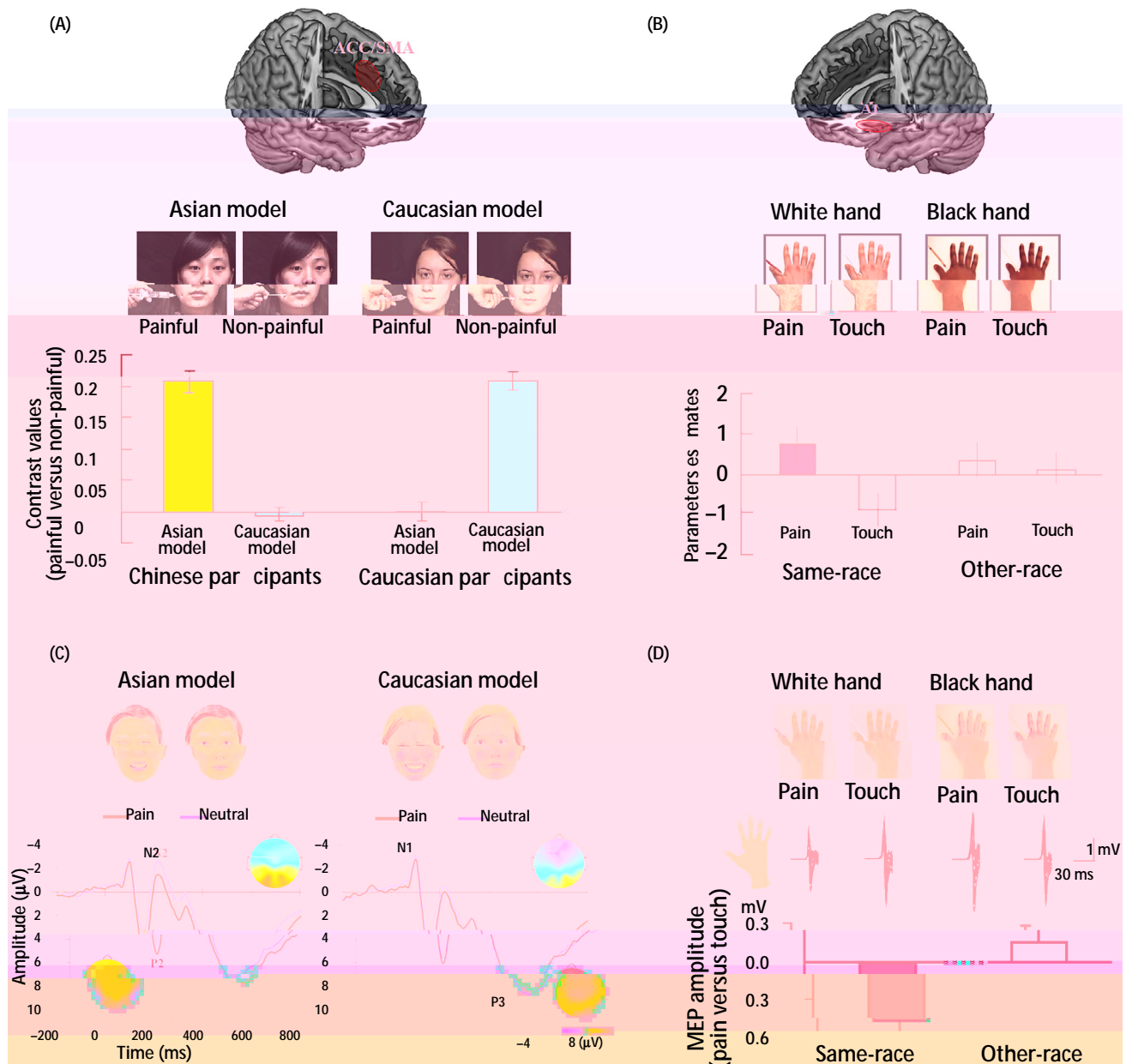
The first brain imaging study of RIBE scanned Chinese and white college students in China, using fMRI, while participants viewed video clips showing faces of six Asian and six Caucasian models [16]. Each 3-s video clip depicted a face with a neutral facial expression, receiving either painful (needle 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 to press. To examine whether the 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 stimulation being applied to the models significantly activated the ACC/SMA and the inferior frontal (IF)/AOrtex in both ethnic groups. However, the ACC/SMA activation was significantly decreased in response to painful stimulation 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) stimulation and self-reported measures did not differ significantly between same-race and other-race models. Thus, racial in-group bias in empathic brain activity was evident regardless of the absence of self-reported estimation of RIBE.

Racial in-group 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) stimulation to blacks and white 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 face judgment 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 in-group. These fMRI findings demonstrate racial in-group 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) stimulation 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 in-group 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 amplitude 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 in-group bias in empathic neural



Trends in Cognitive Sciences

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/non-painful stimulation 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/non-painful stimulation 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 favors same-race individuals.

Perceiving painful versus non-painful stimulation 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 stimulation (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 stimulation 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 in-group bias in empathic neural responses in the N1 time-window, suggesting strong effects of interracial (versus minimal) in-group 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 in-group bias in sensorimotor responses to the pain of others, motor-evoked potentials (MEPs) elicited by 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 stimulation 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 in-group 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 in-group 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 in-group bias in motor-evoked potentials (MEP) elicited by transcranial magnetic stimulation. The top panel illustrates video clips showing painful/non-painful stimulation 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 stimulation 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

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 movements 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.

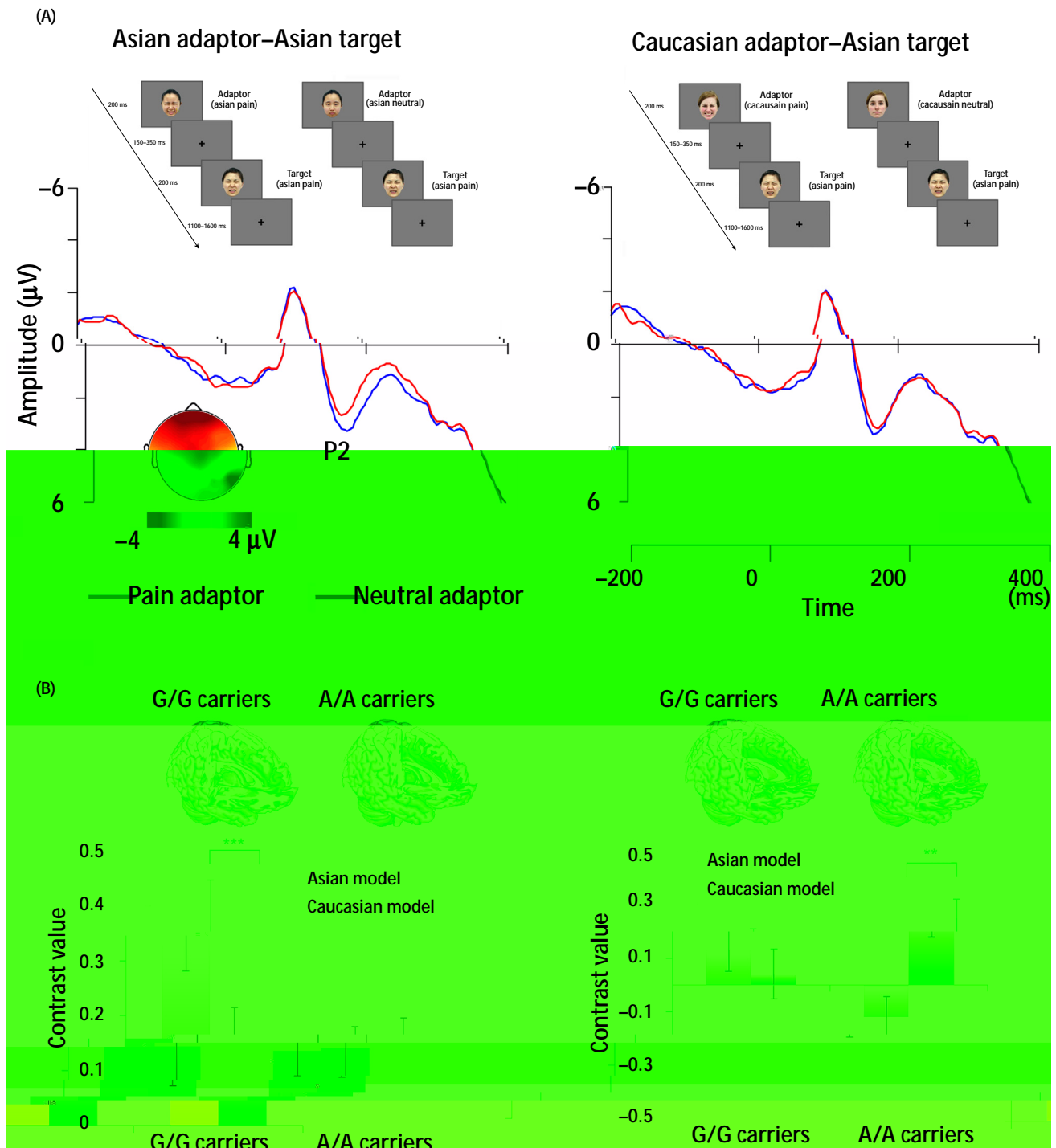
Neurobiological Mechanisms

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 suppression). If empathic responses to same-race and other-race pain are encoded by distinct neuronal populations, the repetition suppression effect related to painful expressions 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) expressions of same-race but not other-race faces [44], resulting in greater racial group 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 group bias in ACC/SMA activity positively predicted participants' racial group bias in implicit attitudes, and the NAcc activity in response to racial outgroup individuals' pain negatively predicted participants' motivation 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 collectivist than individualistic cultures [64], one may expect stronger RIBE in samples dominated by collectivist than



Trends in Cognitive Sciences

Figure 2. Neurobiological Mechanisms Underlying Racial Ingroup Bias in Empathy. (A) Repetitions suppression effects on empathic neural responses in Chinese participants. The top panel illustrates the experimental procedure wherein Asian target faces with a painful expression is preceded by Asian or Caucasian adaptor faces with painful or neutral expressions. The repetitions suppression effects defined by the differential event-related potentials to target faces preceded by adaptor faces with neutral versus painful expressions. The bottom panel illustrates the repetitions 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 no or salient racial in-group bias in the mPFC activity in response to the suffering of others in African-Americans than in Caucasian-Americans [53,4] and greater racial in-group 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 in-group 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 in-group 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 in-group 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 environment 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 in-group 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 in-group 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.

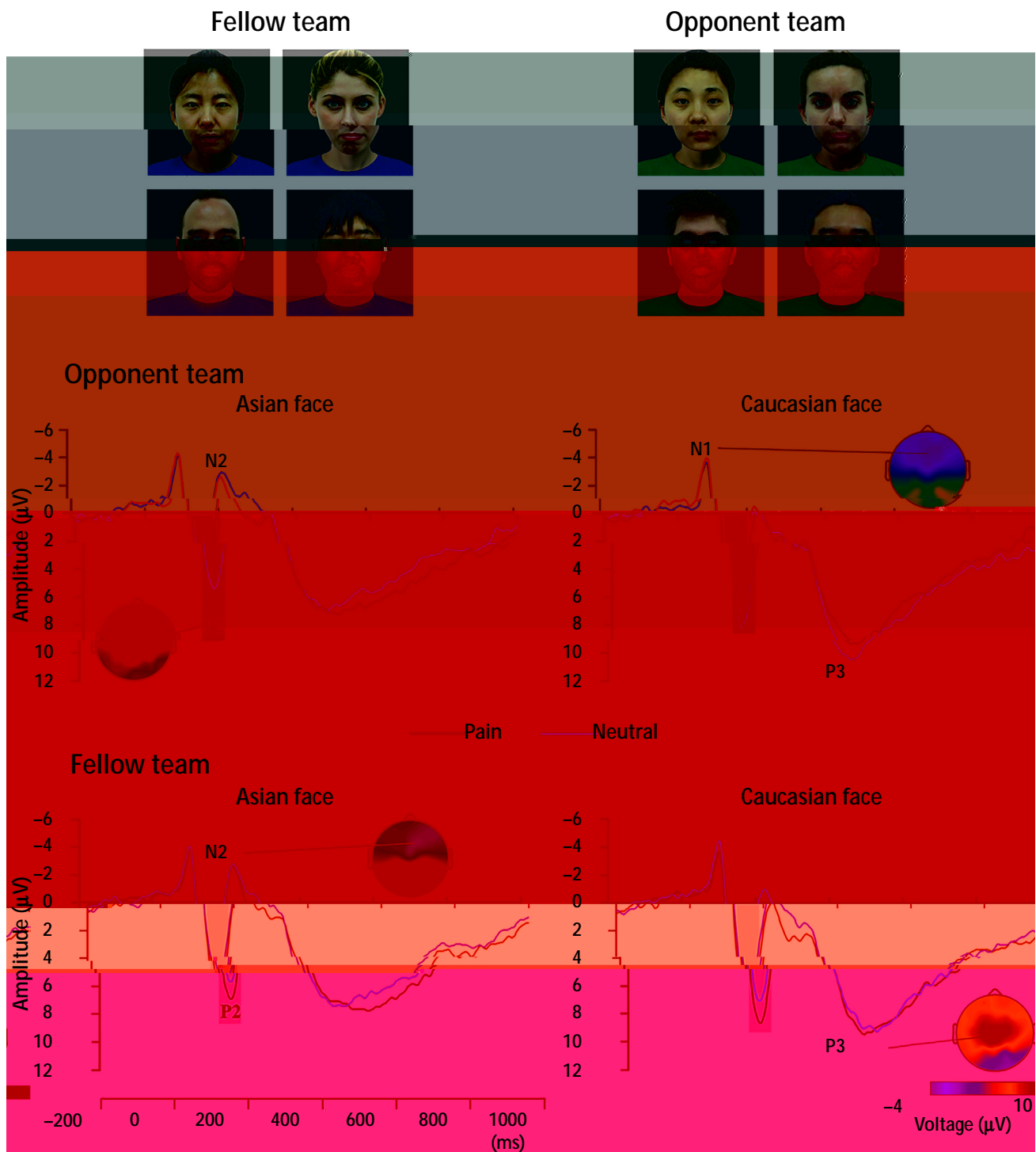
Because the worst consequence of inclement environments would be the loss of human lives, researchers also examined whether making individuals think about this consequence (i.e., death) would increase racial in-group bias in empathic brain activity. Reminders of death lead to increased group affiliation [73,74] which may then increase RIBE. Consistent with this, both fMRI and ERP evidences showed that asking Chinese students to think about death increased racial in-group bias in empathic neural responses in the ACC and in the P3 time-window when viewing painful (versus non-painful) stimulation 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 In-group 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) Association 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 in-group bias in neural responses to video clips showing painful or non-painful stimulation 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 _____.



Trends in Cognitive Sciences

Figure 3. Decreased Racial Ingroup Bias in Empathy in a Minima 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 effective responses to other-race pain, supported by the ACC and AI activity and thereby reduce RIBE.

Concluding Remarks

The aforementioned section summarizes 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

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

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 in-group 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 inter-group/interracial interactions on cognitive and affective processes. This understanding might in turn increase conscious efforts to counteract RIBE.

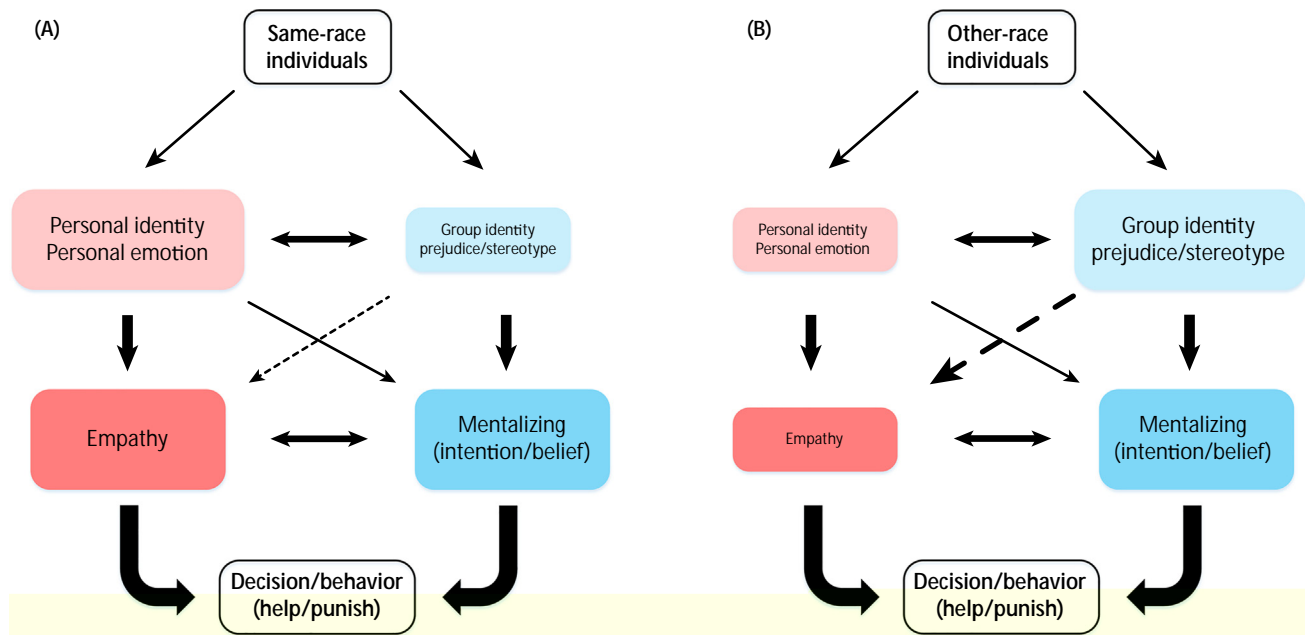
Additionally, the findings that RIBE in neural activity can be weakened or even erased through laboratory manipulations and real-life interracial interactions provide a neuroscientific basis for developing intervention programs to mitigate racial discrimination in social behavior. Moreover, the findings suggest potential methods for interventions, 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 [85,96], 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 inferences 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 modulation 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 intention and beliefs of others, promotes altruistic decision making and behavior toward racial in-group members. By contrast, the processing of other-race



Trends in Cognitive Sciences

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 personal 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 interaction 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 continues to play a key role in behavior towards social outgroup members, the enhanced processing of group identity and the activation of prejudices/stereotypes consistent with that group identity dampen 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 activation and connections across time.

Finally, in-group 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 the 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 context 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 context. 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 3166114303931470986, 31421003). The author is grateful to Daniela M. Pfabigan, Yina Ma, and Elizabeth R. Losin for proofreading of the manuscript and to Feng Sheng and Xiaochun Han for help with modification of the figures.

References

- Moya, P. and Markus, H.R. (2011) Doing race: a conceptual overview. In *Doing Race: 21 Essays for the 21st Century* (Markus, H.R. and Moya, P., eds), pp. 1–102, Norton.
- Richeson, J.A. and Sommers, S.R. (2016) Toward a social psychology of race and race relations for the twenty-first century. *Annu. Rev. Psychol.* 67, 439–463.
- Bowers, W.J. et al. (2001) Race, crime and the constitution: death sentencing in black and white: an empirical analysis of the role of jurors' race and jury racial composition. *Univ. Pa. J. Const. Law* 3, 171–274.
- Hunt, J.S. (2015) Race, ethnicity and culture in jury decision making. *Annu. Rev. Law Soc. Sci.* 11, 269–288.
- Stout, E. et al. (2017) Racial differences in adherence to prescribed analgesia in cancer patients: an integrated review of quantitative research. *JCOM* 24, 39–48.
- Batson, C.D. (2009) These things called empathy: eight related but distinct phenomena. In *The Social Neuroscience of Empathy* (Decety, J. and Ickes, W.J., eds), pp. 3–15, MIT Press.
- Batson, C.D. (2011) *Altruism in Humans*, Oxford University Press.
- Batson, C.D. et al. (1981) Is empathic emotion a source of altruistic motivation? *J. Pers. Soc. Psychol.* 40, 290–302.
- Batson, C.D. et al. (1983) Influence of self-reported distress and empathy on egoistic versus altruistic motivation to help. *J. Pers. Soc. Psychol.* 45, 706–718.
- Schroeder, D.A. et al. (1988) Empathic concern and helping behavior: egoism or altruism? *J. Exp. Soc. Psychol.* 24, 333–353.
- Decety, J. et al. (2016) Empathy as a driver of prosocial behaviour: highly conserved neurobehavioural mechanisms across species. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* 371, 20150077.
- de Waal, F.B. and Preston, S.D. (2017) Mammalian empathy: behavioural manifestations and neural basis. *Nat. Rev. Neurosci.* 18, 498–509.
- Jackson, P.L. et al. (2005) How do we perceive the pain of others? A window into the neural processes involved in empathy. *Neuroimage* 24, 771–779.
- Gu, X. and Han, S. (2007) Attention and reality constrain the neural processes of empathy for pain. *Neuroimage* 36, 256–267.
- Lamm, C. et al. (2007) What are you feeling? Using functional magnetic resonance imaging to assess the modulation of sensory and affective responses during empathy for pain. *PLoS One* 2, e1292.
- Xu, X. et al. (2009) Do you feel my pain? Racial group membership modulates empathic neural responses. *J. Neurosci.* 29, 8525–8529.
- Azevedo, R.T. et al. (2013) Their pain is not our pain: brain and autonomic correlates of empathic resonance with the pain of same and different race individuals. *Hum. Brain Mapp.* 34, 3168–3181.
- Fan, Y. and Han, S. (2008) Temporal dynamics of neural mechanisms involved in empathy for pain: an event-related brain potential study. *Neuropsychologia* 46, 160–173.
- Sheng, F. et al. (2014) Task modulation of racial bias in neural responses to others suffering. *Neuroimage* 88, 263–270.
- Saarela, M.V. et al. (2007) The compassionate brain: humans detect intensity of pain from another's face. *Cereb. Cortex* 17, 230–237.
- Han, S. et al. (2009) Empathic neural responses to others' pain are modulated by emotional contexts. *Hum. Brain Mapp.* 30, 3227–3237.
- Singer, T. et al. (2004) Empathy for pain involves the affective but not sensory components of pain. *Science* 303, 1157–1162.
- Singer, T. et al. (2006) Empathic neural responses are modulated by the perceived fairness of others. *Nature* 439, 466–469.
- Fan, Y. et al. (2011) Is there a core neural network in empathy? An fMRI-based quantitative meta-analysis. *Neurosci. Biobehav. Rev.* 35, 903–911.
- Lamm, C. et al. (2011) Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. *Neuroimage* 54, 2492–2502.
- Decety, J. and Michalska, K.J. (2010) Neurodevelopmental changes in the circuits underlying empathy and sympathy from childhood to adulthood. *Dev. Sci.* 13, 886–899.
- Tousignant, B. et al. (2016) A developmental perspective on the neural bases of human empathy. *Infant Behav. Dev.* 48, 5–12.
- Jackson, P.L. et al. (2006) Empathy examined through the neural mechanisms involved in imagining how I feel versus how you feel pain. *Neuropsychologia* 44, 752–761.
- Rütgen, M. et al. (2015) Placebo analgesia and its opioidergic regulation suggest that empathy for pain is grounded in self-pain. *Proc. Natl. Acad. Sci. U. S. A.* 112, E5638–E5646.

30. Betti, V. and Aglioti, S.M. (2016) Dynamic construction of the neural networks underpinning empathy for pain. *Neurosci. Bio-behav. Rev.* 63, 191-206
31. Masten, C.L. et al. (2011) An fMRI investigation of empathy for 'social pain' and subsequent prosocial behavior. *Neuroimage* 55, 381-388
32. Meyer, M.L. et al. (2012) Empathy for the social suffering of friends and strangers recruits distinct patterns of brain activation. *Soc. Cogn. Affect. Neurosci.* 8, 446-454
33. Novembre, G. et al. (2014) Empathy for social exclusion involves the sensory-discriminative component of pain: a within-subject fMRI study. *Soc. Cogn. Affect. Neurosci.* 10, 153-164
34. Mathur, V.A. et al. (2010) Neural basis of extraordinary empathy and altruistic motivation. *Neuroimage* 51, 1468-1475
35. Morelli, S.A. et al. (2014) The neural components of empathy: predicting daily prosocial behavior. *Soc. Cogn. Affect. Neurosci.* 9, 39-47
36. Cheng, Y. et al. (2007) Expertise modulates the perception of pain in others. *Curr. Biol.* 17, 1708-1713
37. Mu, Y. et al. (2008) Event-related theta and alpha oscillations mediate empathy for pain. *Brain Res.* 1234, 128-136
38. Sheng, F. and Han, S. (2012) Manipulations of cognitive strategies and intergroup

80. Staton, L.J. *et al.* (2007) When race matters: disagreement in pain perception between patient and their physicians in primary care. *J. Natl. Med. Assoc.* 99, 532-538
81. Drwecki, B.B. *et al.* (2011) Reducing racial disparities in pain treatment: the role of empathy and perspective-taking. *Pain* 152, 1001-1006
82. Kaseweter, K.A. *et al.* (2012) Racial difference in pain treatment and empathy in a Canadian sample. *Pain Res. Manag.* 17, 381-384
83. Johnson, J.D. *et al.* (2002) Rodney King and O. J.: revisited the impact of race and defendant empathy induction on judicial decisions. *J. Appl. Soc. Psychol.* 32, 1208-1223
84. Batson, C.D. *et al.* (1995) Immorality from empathy-induced altruism: when compassion and justice conflict. *J. Pers. Soc. Psychol.* 68, 1042-1054
85. Hoffman, M.L. (ed.) (2001) *Empathy and Moral Development: Implications for Caring and Justice*, Cambridge University Press
86. Bingel, U. *et al.* (2003) Single trial fMRI reveals significant contralateral bias in responses to laser pain within thalamus and somatosensory cortices. Batson,

128. Gutsell, J.N. and Inzlicht, M. (2012) Intergroup differences in the sharing of emotive states: neural evidence of an empathy gap. *Soc. Cogn. Affect. Neurosci.* 7, 596-603
129. Ma, Y. et al. (2011) Neural responses to perceived pain in others predict real-life monetary donations in different socioeconomic contexts. *Neuroimage* 57, 1273-1280
130. Morelli, S.A. and Lieberman, M.D. (2013) The role of automaticity and attention in neural processes underlying empathy for happiness, sadness, and anxiety. *Front. Hum. Neurosci.* 7, 160
131. Lamm, C. et al. (2007) The neural substrate of human empathy: effects of perspective-taking and cognitive appraisal. *J. Cogn. Neurosci.* 19, 42-58
132. Eres, R. and Molenberghs, P. (2013) The influence of group membership on the neural correlates involved in empathy. *Front. Hum. Neurosci.* 7, 176
133. Decety, J. et al. (2010) The blame game: the effect of responsibility and social stigma on empathy for pain. *J. Cogn. Neurosci.* 22, 985-997