part of the same harmonic series. The shifted harmonic is perceptually segregated from the rest of the complex and has little effect on the overall pitch [11].

So although *preference* for harmonicity may be dependent on musical experience, the use of harmonicity in auditory processing is probably not dependent on this specific experience. Instead its use is driven by the adaptation of the auditory system to the acoustic properties of objects in the environment. The preference for consonance reflects the central role of harmonicity in auditory perception, both for the identification of sounds and for the segregation of sounds from different sound sources.

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Human Communication and Deafness Division, The University of Manchester, Manchester M13 9PL, UK.
E-mail: chris.plack@manchester.ac.uk

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Intergroup Empathy: How Does Race Affect Empathic Neural Responses?

How does race affect the human ability to share and respond to the suffering of others? Recent evidence provides novel insight into how and why race alters empathic neural response.

Joan Y. Chiao and Vani A. Mathur

In 1959, John Howard Griffin ingested anti-vitiligo drugs which transformed the color of his skin from white to black, and then travelled through the racially segregated South for the first time from the perspective of a Black man (Figure 1). In his memoir '*Black Like Me*', Griffin would later remark, "I had no idea what they [Blacks] have to go through. I literally bawled myself to sleep some nights. I learned that when it is night, when it is dark, then the Negro feels safest. Langston Hughes's line, 'Night coming tenderly/ Black like me', has real meaning".

How and why does race affect our ability to understand and share the suffering of others? Race is a potent modulator of neural responses underlying social behavior [1,2]. Prior neuroimaging research has demonstrated that racial majority group members, such as Whites (in the US), show greater fusiform and parahippocampal response when perceiving own-race faces [3], and either heightened [4] or attenuated [5,6] amygdala response to other-race faces, depending on social context and presence of unconscious racial bias [7–9]. By contrast, members of racial minority groups, such as Blacks, typically demonstrate greater fusiform [3] as well as amygdala activation to own-race faces [4], suggesting that intergroup status moderates the direction and magnitude of neural responses to ingroup and outgroup members [3–6].

Most recently, studies of race and social brain functioning have focused on the neural basis of intergroup empathy [10–12]: in particular, a study reported in this issue of *Current Biology* [10] using transmagnetic stimulation (TMS) reveals for the first time greater empathic sensorimotor contagion when observing the physical suffering of subjects of the same race, but not those of other races.

Multiple Routes to Empathy

Empathy is the capacity to understand and share the emotional states of others and serves as a key motivator and the proximate mechanism of altruistic behavior, whereby an individual perceives and shares in the distress of another person, and acts to reduce his or her suffering [13].

Convergent evidence suggests the existence of multiple routes to our ability to understand and share the pain of another, including sensorimotor contagion, affect sharing and cognitive perspective-taking or appraisal [14].

During sensorimotor contagion, seeing a painful sensorimotor experience in another person, such as a needle penetrating another's hand, elicits an isomorphic sensorimotor experience in the observer — for example, muscle-specific freeze within the same region of the observer's hand [15]. By contrast, during affect sharing, seeing the emotional pain of another person, such as a painful facial expression, elicits a shared affective experience, while during cognitive perspective-taking, the capacity to

Figure 1. To understand the experience of racial discrimination, Griffin, a white native from Texas, artificially darkened his skin.

take another's perspective facilitates shared emotional experience [14,16]. The former route to empathy is thought to occur automatically and without conscious awareness of one's own emotional state, whereas the latter routes reflect conscious empathic experience and are modulated by top-down contextual factors.

Empathic Neural Response for Same but not Other Races

Prior studies of sensorimotor contagion using TMS have shown that muscle-specific motor-evoked potentials (MEPs) are inhibited when participants observe the physical suffering of another, such as watching a needle penetrating a specific muscle [15]. In their recent study, Avenanti, *et al.* [10] found that both Black and White participants showed greater muscle-specific corticospinal inhibition when watching a needle penetrate the hand, but only when the hand was a person of the same race, indicating an ingroup bias in the activation of pain representations within the perceiver's sensorimotor system (Figure 2A).

Intriguingly, ingroup biases in empathic neural response appeared to occur as a function of culturally acquired racial prejudice, rather than an automatic sensorimotor preference to respond to the physical suffering of same-race targets. When Black and White participants in their study were shown a needle penetrating the hand of a different, but culturally unfamiliar race target, such as a Violet hand, greater sensorimotor contagion was still observed. They found that unconscious racial bias modulated the extent to which corticospinal inhibition was preferential for same-race targets (Figure 2B). Moreover, participants who showed greater unconscious racial bias, as measured by the implicit association test, showed greater ingroup bias in corticospinal inhibition (Figure 2C). Taken together, their results suggest that an empathic neural response to the physical suffering of others occurs readily, but unconscious racial prejudice can lessen the extent to which empathy for other race targets occurs and persists.

In addition to sensorimotor contagion, empathic neural response is facilitated by affect sharing, cognitive perspective taking and appraisal [14,16,17]. A distinct neural pain matrix, including bilateral anterior insula (AI) and anterior cingulate cortex (ACC)

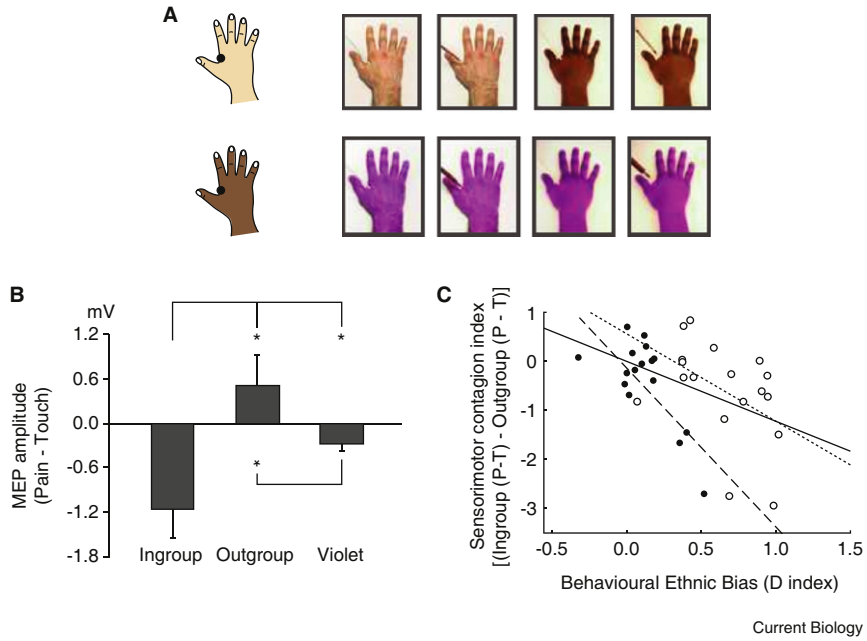

Figure 2. Empathic sensorimotor contagion as a function of race.

(A) Black and White participants observe a needle penetrating a specific muscle in a Black, White or Violet hand. (B) For Black and White participants, MEP inhibition is greater for ingroup relative to outgroup hands. (C) Unconscious racial bias predicts degree of ingroup bias in empathic sensorimotor contagion. (Adapted from [13].)

[14,16,17], is thought to underlie the affective components of empathy. AI and ACC code the autonomic and affective dimension of pain and, in particular, the subjective experience of empathy when perceiving pain or distress in others [14,16,17]. The affective empathic neural response varies among individuals, depending on factors such as the degree to which one prefers social hierarchy over egalitarianism [18]. Cognitive components of empathy, such as the capacity to take another person's perspective, are thought to rely on subregions of medial prefrontal cortex (MPFC) [19]. Hence, the capacity to understand and share another's pain is supported by sensorimotor (for example, contagion), affective (for example, affect resonance) and cognitive (for example, perspective-taking) mechanisms in the brain.

Recent neuroimaging evidence indicates that race modulates affective and cognitive components of empathic neural response. One recent neuroimaging study [11] found that White and Asian participants show increased empathic neural response within the supplementary motor area, ACC, and lateral frontal cortices when perceiving a needle penetrating a same-race face, but decreased ACC response when perceiving a needle penetrating an other-race face. Another recent neuroimaging study [12] showed that, for Black and White participants, empathy for ingroup members was neurally distinct from empathy for humankind more generally. When observing the emotional suffering of others, Black and White participants recruited ACC and bilateral AI, yet Black participants additionally recruited MPFC when observing the suffering of members of their own racial group. Moreover, neural activity within MPFC in response to pain expressed by ingroup relative to outgroup members predicted greater empathy and altruistic motivation for one's ingroup, suggesting that neurocognitive processes associated with self-identity underlie extraordinary empathy and altruistic motivation for members of one's own racial group.

The results of all of these studies indicate that empathic neural response is heightened for members of the same race, but not those of other races. It could be argued that as a social species, humans have evolved for cooperative living in social groups and that effective cooperative living sometimes entails belonging to smaller social groups and limiting resource sharing to members of that group so

that individual costs and risks associated with nonreciprocated empathy and altruism are reduced. By this view, enhanced empathic neural response for same but not other races is a consequence of group selection in prosociality and altruistic behavior. Nevertheless, growing evidence indicates that racial bias in empathic neural responses is not inevitable, but instead results from culturally acquired prejudice. This in turn demonstrates flexibility in empathic neural circuitry and highlights a pivotal role for culture in changing how and when humans share and respond to the suffering of same and other races.

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Department of Psychology and Interdepartmental Neuroscience Program, Northwestern University, 2029 Sheridan Road, Evanston, IL 60208, USA.
E-mail: joan.chiao@gmail.com

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Epigenetic Switching: Bacteria Hedge Bets about Staying or Moving

Growing populations of *Bacillus subtilis* exhibit bistability: motile cells co-exist with long chains of sessile cells. An epigenetic switch has been characterized that controls the transition between the two cell types.

Patrick Piggot

Motility gives bacteria the distinct advantage of being able to move towards good things, and away from bad things. However, considerable resources need to be devoted to building flagella, becoming motile and displaying chemotaxis. Consequently, if local conditions are good, there is an advantage to staying put, and not wasting resources on these processes. Indeed, motility is typically regulated so that bacteria are sometimes sessile and sometimes motile. In *Bacillus subtilis*, these two types of bacterial cell can occur successively or can co-exist as distinct cell lineages within a genetically homogeneous population. A recent paper by Chai *et al.* [1] elucidates the nature of the epigenetic switch between the two lineages. The switch has a double-negative feedback loop involving protein–protein and protein–DNA interactions.

In species such as *Escherichia coli*, motility may be associated with a particular growth phase: the bacteria are not motile during exponential growth in batch cultures, when the times are good, and food is plentiful. They become motile during the transition to stationary phase, bad times with starvation approaching [2]. Similar behavior is exhibited by *B. subtilis* when it is grown in a rich medium [3]. With *B. subtilis*, the non-motile cells are not simply sessile, and devoid of flagella: they are present in long chains because separation of the sessile cells lags far behind their formation by cell division. This behavior means that any switch between non-motile and motile is also a switch between low and high activity of the autolysins responsible for cell separation. In appropriate circumstances, motile *B. subtilis* can go on to initiate formation of biofilms, in which the bacteria have again become sessile, and are in long chains that are held together by an extracellular matrix [4,5].

In the contrasting case of *Caulobacter crescentus*, both motile and sessile bacteria are present throughout exponential growth. Sessile, stalked bacteria grow and divide by binary fission to give one daughter that is motile, with the other being sessile [6]. Thus, after every division half the population stays and half is able to move to better conditions. The sessile daughter is primed to undergo another division; the motile daughter must first differentiate into a sessile cell before it is able to divide. Both sessile and motile bacteria are also observed throughout growth for *B. subtilis* when it is grown in a minimal medium [3,7] (Figure 1). However, the mechanism controlling this bifurcation is very different. Within the same growing population the two