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ORIGINAL RESEARCH ARTICLE

# A sociocultural neuroscience approach to pain

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**Abstract** A significant body of research has identified ethnic, racial, and national differences in pain report. Although a number of contemporary models of the pain experience include top-down modulation by social and cultural factors, the neurobiological mechanisms underlying these group differences in pain remain unknown. We argue that a sociocultural neuroscience approach to pain may elucidate the sociocultural and neurobiological mechanisms underlying group differences in pain report. As a foundation for this approach to pain we will (1) review examples of group differences in pain report, (2) propose a neurocultural model of pain that outlines and connects cultural and neurobiological mechanisms that may account for these group differences, (3) review the literature that supports the connections between culture, pain, and the brain in each stage of our model, and (4) discuss the novel contributions that a sociocultural neuroscience approach to pain can make to our understanding of pain and to improving pain diagnosis and treatment.

**Keywords** Sociocultural · Cultural neuroscience · Pain · Pain modulation · Neuroimaging · fMRI

Over the past decade, neuroscience methods have been used to investigate social behavior and cultural variation. This sociocultural neuroscience approach has revealed neurobiological mechanisms underlying cultural variation in a variety of social and cognitive domains, including emotion processing, perception of the self and others, sensory perception, and attention (For reviews see Han 2015a, b). A long-standing literature suggests that cultural norms and practices may also influence the incidence and presentation of disease as well as its diagnosis and

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treatment (Spector 2002), yet little is known about the neurobiological mechanisms underlying these culture-health interactions.

Pain is an ideal target for investigating neurobiological mechanisms underlying sociocultural influences on health for several reasons. First, pain is part of most major medical disorders and is the most common reason patients seek medical treatment (Loeser and Melzack 1999). Second, pain accounts for a large proportion of both the financial and disability burden of illness and disease on society (Murray et al. 2013; IOM 2011). Finally, anthropological and psychological investigations of pain have revealed ethnic, racial, and national group differences in pain report (Rahim-Williams et al. 2012), collectively referred to here as *group differences*. Although contemporary models of acute and chronic pain mechanisms include top-down modulation by social, cultural, and contextual factors (Bates 1987; Craig 2009; Gatchel et al. 2007; Kirmayer 2008; Loeser and Melzack 1999; Melzack 2001; Merskey et al. 1979), the neurobiological mechanisms underlying these group differences in pain report remain unclear (Rahim-Williams et al. 2012). Here, we will (1) review examples of group differences in pain report, (2) propose a neurocultural model of pain that outlines and connects cultural and neurobiological mechanisms that may account for these group differences, (3) review the literature that supports the connections between culture, pain, and the brain in each stage of our model, and (4) discuss the novel contributions that a sociocultural neuroscience approach to pain can make to our understanding of pain, its diagnosis, and its treatment.

## Group differences in pain report

As evidence supporting a culture-pain connection, a substantial body of literature has identified differences in pain report and the incidence of pain conditions between ethnic, racial, and national groups (Rahim-Williams et al. 2012). In pioneering work by Zborowski (1952) at the Kingsbridge Veterans Hospital, medical personnel reported that the Jewish and Italian patients had lower pain thresholds and were more sensitive to pain than the Irish and Anglo-American patients. In more recent experimental pain studies, African Americans, and in some cases Hispanics, have been found to report more pain than Non-Hispanic Whites in response to multiple experimental pain modalities (Campbell et al. 2008; Edwards and Fillingim 1999; Mechlin et al. 2005; Rahim-Williams et al. 2007) and clinical conditions (Breitbart et al. 1996; Creamer et al. 1999; Edwards and Fillingim 1999; Green et al. 2003; Greenwald 1991; Riley et al. 2002). A few studies have examined pain sensitivity in East and South Asians, with most finding that Asians report lower pain threshold and tolerance levels to acute experimental pain compared to Non-Hispanic Whites (Gazerani and Arendt-Nielsen 2005; Rowell et al. 2011; Watson et al. 2005; Woodrow et al. 1972). Although the majority of studies finding group differences in pain have been conducted with groups residing in the US, group differences in pain have been documented in other countries (Tan et al. 2008). These findings suggest that the culture-pain connection may be a cross-national phenomenon.

As can be seen from these findings, studies of group differences in pain report have employed different constructs to define groups for comparison, including ethnicity—typically referring to a common nationality, culture, or language; race—typically referring to categories tied to phenotypic characteristics, and nationality (Betancourt and Lopez 1993). The definition, validity, and utility of these group constructs is controversial. Regardless of the particular construct used to identify group differences, however, our goal in the present review is to outline potential cultural mechanisms—those related to shared beliefs, values, and practices—and potential neurobiological mechanisms that may underlie those differences.

## Sociocultural neuroscience approach to pain

Despite substantial documentation of group differences in pain report, very little is known about their underlying sociocultural and neurobiological mechanisms (Rahim-Williams et al. 2012). One reason for this lack of understanding is that neurobiological conceptions of pain historically focused on bottom-up processes (e.g., Descartes 1644), whereas culture likely acts as a top-down modulator of the pain experience (Loeser and Melzack 1999). More recent conceptions of pain have incorporated the concept of top-down pain modulation. The gate control theory of pain (Melzack and Wall 1967) proposed the first nervous system mechanism of pain modulation. Since then, other models of pain have included top-down pain modulation and even spelled out the role that social behavior and culture may play in this modulation (e.g., Bates 1987; Craig 2009; Gatchel et al. 2007; Kirmayer 2008; Melzack 2001). We argue, however, that a sociocultural neuroscience approach to pain will be necessary in order to fully elucidate the sociocultural and neurobiological mechanisms underlying group differences in pain report. This approach aims to connect the cultural influences on pain described in the anthropological and cross-cultural psychology literature with the neurobiological mechanisms of pain now being revealed using neuroimaging.

Another reason group differences in pain are not well understood may be a conflict between the multidimensional nature of culture (Bates and Plog 1990) and the often unidimensional nature of pain measurement used in clinical and research settings (Jensen and Karoly 2011). Bates and Plog (1990) famously defined culture as including shared beliefs, values, customs, and behaviors. Although different dimensions of pain perception can be measured, including intensity, affect, quality, and location, in clinical and experimental settings pain measurement is often limited to a numeric rating scale (NRS) on which the patient provides a number between 0 and 10 indicating how much pain (intensity) they are experiencing (Jensen and Karoly 2011). In reality, this single number provided by the patient is underlain by the cumulative effects of cultural experience beginning at birth, which in turn affect each stage of the pain experience and its underlying neurobiological mechanisms (Melzack 2001).

As a foundation for a sociocultural neuroscience approach to pain, we propose a neurocultural model of pain that aims to explain group differences in pain report by delineating the specific aspects of culture affecting each stage of the pain experience and the potential neurobiological mechanisms underlying these pain-culture connections (Fig. 1). Compared to previous pain models (e.g., Bates 1987; Craig 2009; Gatchel et al. 2007; Kirmayer 2008; Melzack 2001), our model has a more explicitly temporal organization. Furthermore, we conceptualize the "pain experience" more broadly as encompassing influences from birth until the moment of pain report in order to address the cumulative effects of cultural experience throughout the life course. We divide potential influences on pain report based on this temporal organization, with those occurring between birth and the painful event referred to as *pain precursors*. Influences on pain report occurring between the painful event and pain report are further divided into those affecting *nociception*, or the sensory transduction of a potentially injurious stimulus; *pain responses*, or the internal responses to the nociceptive information; and *pain communication*, or the external responses to the previous two stages. For each temporal stage we (1) delineate the particular aspects of culture which may influence pain at that stage, and (2) connect these cultural elements to psychological and neural mechanisms known to influence pain report.

Our model is not intended to imply or provide a simple predictive relationship between a given group and the level of reported pain. Instead, our model is meant to

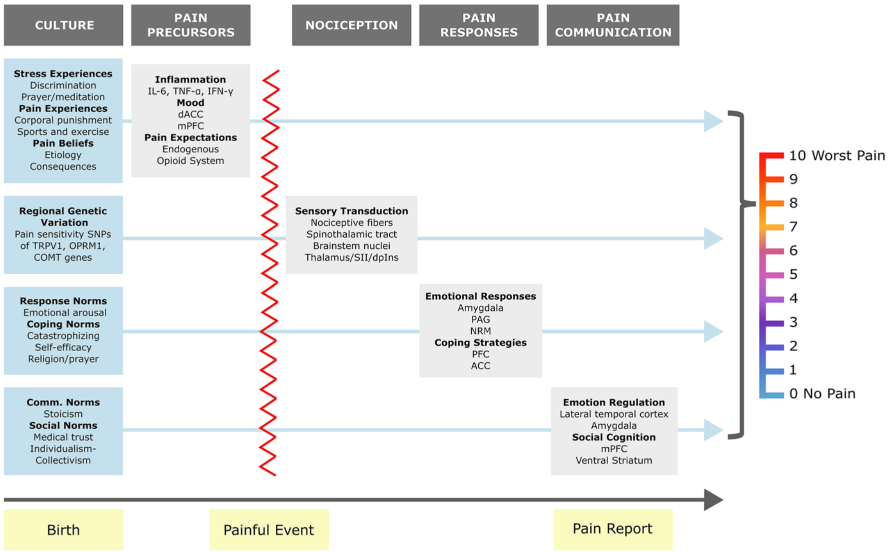


**Fig. 1** Neurocultural model of pain. The model delineates the specific aspects of culture affecting each stage of the pain experience (*top boxes*), and the potential neurobiological mechanisms underlying these pain-culture connections. Time over the lifespan leading up to pain report is represented on the x-axis. For each aspect of culture represented in the *far-left boxes*, *bolded descriptions* refer to the specific cultural constructs described in the review, while *non-bolded descriptions* are examples of cultural variability. *Moving right, the boxes* under each section of the model refer to the psychological and neural mechanisms that may connect each cultural construct to pain. *Bolded descriptions* refer to psychological/physiological mechanisms, while *non-bolded descriptions* are examples of underlying brain regions/systems. All contribute to a single number provided during pain report (*far right*). Abbreviations: *dACC* dorsal anterior cingulate cortex, *mPFC* medial prefrontal cortex, *SII* secondary somatosensory cortex, *dpIns* dorsal posterior insula, *PAG* periaqueductal gray, *NRM* nucleus raphe magnus, *PFC* prefrontal cortex, *ACC* anterior cingulate cortex

highlight the ways in which cultural influences on the pain experience may contribute to group differences in pain report. In the following sections, we review the extant literature supporting each component of our model. Although the examples of group differences discussed in each section may not apply to everyone in a given culture, or to a given culture over time, they are highlighted to illustrate the significant cultural variability that exists in the experience of pain worldwide.

Because neurobiological mechanisms may appear in multiple sections of our model, here we provide a brief general overview of the peripheral and central mechanisms of pain processing. The ascending acute pain pathway begins with a noxious (injurious or potentially injurious) stimulus activating a set of specialized cutaneous receptors, called nociceptors. Nociceptive signals are transmitted via the spinal cord and brainstem to the amygdala and thalamus, which in turn project to subcortical and cortical brain regions (Ringkamp et al. 2013). Cortical regions include the primary and secondary somatosensory cortices (SI & SII), dorsal posterior insula (dpIns), and the most caudal area of the parietal operculum (OP1), which are thought to be specific to the nociceptive aspects of pain; the anterior cingulate cortex (ACC) and anterior insula (AI) are thought to be involved in the affective aspects of pain (Apkarian et al. 2005; Bushnell et al. 2013; May 2007). Many of these same brain regions, especially the ACC, exert top-down influences on pain through activation of brainstem nuclei, including the periaqueductal gray (PAG) and nucleus raphe magnus (NRM), which ultimately suppress incoming pain signals from the spinal cord (Gebhart 2004).

## Culture, brain and pain precursors

The first section of our model examines how the cultural experiences, practices, and beliefs acquired over the lifespan may influence the physiological, emotional, and cognitive states that an individual is in when they encounter a painful event, as these pain precursors have been found to shape the pain experience (Enck et al. 2008; Villemure and Bushnell 2002; Zhang and An 2007).

### Cultural differences in physiological and emotional precursors of pain

Culturally embedded stressful experiences, such as having low socioeconomic status (SES) (Chen et al. 2011) and experiencing racial and ethnic discrimination (Burgess et al. 2009), have been linked to physiological and emotional states that are known to increase pain sensitivity, including increased pro-inflammatory cytokine production and gene expression (Brody et al. 2015; Miller et al. 2009; Zhang and An 2007), and anxiety and depression (Gallo and Matthews 2003; Gee et al. 2007; Villemure and Bushnell 2002). Direct links between culturally embedded stressful experiences and pain have also been found. For example, low SES has been associated with increased pain related to dental cavities (Nomura et al. 2004), childbirth (Weisenberg and Caspi 1989), low back pain (Katz 2006), and increased incidence of chronic pain (Andersson et al. 1993). Experiences of racial discrimination have been associated with decreased experimental pain tolerance

(Goodin et al. 2013) and increased incidence of chronic pain (Edwards 2008). There is also evidence that certain cultural practices may positively influence pain-related physiology and mood and have pain-reducing effects. For example, experienced practitioners of various forms of yoga (Kiecolt-Glaser et al. 2010) and meditation (Kaliman et al. 2014) have been found to have lower baseline levels of pro-inflammatory cytokines and reduced inflammatory responses to stressors (Black et al. 2013; Kabat-Zinn et al. 1985; Morone et al. 2008; Zeidan et al. 2010). Judeo-Christian religious practices, including religious service attendance and prayer, have also been associated with lower levels of pro-inflammatory cytokines (Koenig et al. 1997), improved mood (Loewenthal et al. 2000), and decreased pain (Harrison et al. 2005). Together, these findings suggest that cultural variability in stress-modulating experiences and practices may contribute to group differences in the pain experience affecting pain report.

*Neurobiological mechanisms linking physiological and emotional precursors of pain to pain*

Pro-inflammatory cytokines, small regulatory proteins that help regulate the body's immune response (e.g., IL-6, TNF-$\alpha$, and IFN-$\gamma$), are thought to increase pain by lowering activation thresholds of peripheral nociceptors and triggering cascades of other inflammatory mediators (Sommer and Kress 2004). Culturally embedded stressors such as racial discrimination and stress reducing cultural practices, such as prayer, may thus act peripherally to modulate pain by increasing or decreasing peripheral inflammation. Additionally, central mechanisms of pain modulation related to anxiety and depression and meditation have been found. Neuroimaging studies of experimentally induced (Ploghaus et al. 2001) and trait level pain-related anxiety (Ochsner et al. 2006), and a review of studies linking negative affect, cognitive control, and pain (Shackman et al. 2011), have highlighted increased activation within the dorsal anterior cingulate cortex (dACC) as a brain mechanism that may underlie the increased pain perception associated with anxiety. Heightened activation within the amygdala (Giesecke et al. 2005) and medial prefrontal cortex (mPFC) (Schweinhardt et al. 2008), two brain regions associated with the evaluative and affective aspects of pain, has been implicated in greater pain sensitivity in major depression. In contrast, several of these same brain regions have been found to be less active in Zen meditation practitioners during pain (Grant and Rainville 2009). Thus, in addition to altering peripheral inflammation, culturally embedded stressors and stress reducing practices may modulate pain via brain regions associated with the affective and evaluative aspects of pain.

## Cultural differences in cognitive precursors of pain: normative pain experiences and pain expectations

Studies within anthropology and cross-cultural psychology have revealed cultural variability in normative experiences with pain, which may influence expectations about the intensity of future painful experiences. For example, although there is moderate global usage of corporal (physically painful) punishment of children, usage varies widely across cultures (Lansford and Dodge 2008; Murdock and White 1969).

Similarly, cultural practices typically accompanied by regular physical discomfort and injury, such as participation in sports and vigorous exercise, also vary widely by culture (Bauman et al. 2009). A final example of cultural variability in normative pain experiences is the presence of painful rites of passage. For example, many cultural and religious groups including Jews, Muslims, and a number of ethnic and religious groups in sub-Saharan Africa practice male and/or female genital surgery, often without anesthesia, to mark birth or the passage into adolescence (Toubia 1994; Weiss 2008). Both athletics and physical abuse, which may have similar effects to corporal punishment, have been linked to decreased experimental pain sensitivity (Fillingim and Edwards 2005; Tesarz et al. 2012). However, history of physical abuse has also been linked to an increased incidence of clinical pain complaints and chronic pain (Davis et al. 2005; Fillingim and Edwards 2005), highlighting the potential complexity of relationships between previous pain experiences and future pain. Thus, in cultures where early life experiences with intense pain are common, normalization of pain and altered expectations of future pain may contribute to either a reduction or an increase in pain sensitivity and risk for developing chronic pain.

*Neurobiological mechanisms linking pain expectations to pain*

Placebo analgesia, in which belief in pain reduction reduces perceived pain, is a prototypical example of the pain altering effects of expectations (Hróbjartsson and Gøtzsche 2001). Pharmacological and positron emission tomography (PET) studies have suggested that placebo effects are caused by the release of endogenous opioids, such as endorphins, as part of the body's own pain relieving system (Levine et al. 1978). Additionally, fMRI has been used to demonstrate that placebo analgesia is associated with decreased brain activity in pain-related brain regions, including the thalamus, insula, dACC, and PAG, suggesting that reduced expectations in placebo analgesia can alter both pain experience and report (Amanzio et al. 2013; Wager and Atlas 2015; Wager et al. 2004). Conversely, negative expectations may lead to increased pain perception (Petersen et al. 2014) through a process known as nocebo hyperalgesia, which is associated with increased activity in many of the same brain regions involved in placebo analgesia, as well as the hippocampus (Tracey 2010). Brain mechanisms similar to those underlying placebo analgesia/nocebo hyperalgesia may connect pain expectations associated with culturally mediated exposure to painful experiences with reduced/heightened pain sensitivity.

## Cultural differences in cognitive precursors of pain: pain beliefs

Anthropological studies have demonstrated cross-cultural variability in beliefs regarding the causes and consequences of pain. For example, ethnographic descriptions of pain beliefs suggest that cultural belief systems place different levels of emphasis on biophysical, psychosocial, and supernatural causes of pain. An emphasis on supernatural and divine causes of pain has been described in Mexican-American culture in which pain is sometimes viewed as being due to God's will, a punishment for immoral behavior or penance (Calvillo 2013). Similarly, spiritual causes of pain among Hindus and Muslims include divine will embodied in the Hindu concept of *karma* and the Muslim concept of *qismat* (Pugh 2013). In contrast, a detailed ethnographic survey of pain beliefs of the Quichua, an indigenous group in Ecuador, revealed a heavier emphasis on psychosocial causes of pain, such as the stress associated with married life (Incayawar and Maldonado-Bouchard 2013). European American samples have been found to emphasize biophysical causes of pain, such as injury, illness, and the physical environment, and therefore more frequently view pain as something that needs to be managed by the individual through the use of medication and lifestyle practices (Sharp and Koopman 2013). Belief in an external or divine locus of control for pain and illness has been associated with increased pain intensity and disability in both Western (Arraras et al. 2002; Gustafsson and Gaston-Johansson 1996) and Eastern (Cheng and Leung 2000) cultures. Therefore, cultural pain belief systems that emphasize divine or psychosocial causes of pain could contribute to increased pain sensitivity compared to pain belief systems emphasizing internal biophysical causes of pain. The meaning of pain within a given belief system, e.g., as divine punishment or a means to salvation, is also likely to play an important role in modulating the pain experience and affecting pain report.

*Neurobiological mechanisms linking pain beliefs to pain*

Brain mechanisms related to the amount of control one has over experimentally induced pain may play a role in the pain modulating effects of cultural beliefs about the causes of pain. Human and animal studies suggest that the lateral PFC, a brain region implicated in emotion regulation, contributes to the reduction in pain perception associated with perceived control over pain (Amat et al. 2005; Borckardt et al. 2011), particularly in those who believe other forces, such as divine will, have control over their lives (Wiech et al. 2006). In contrast, pain modulatory brain regions, including the mPFC, amygdala, and PAG, show increased activation when pain is uncontrollable (Mohr et al. 2005; Salomons et al. 2007; Wiech et al. 2006). Furthermore, Brascher et al. (2016) found increased connectivity between the dorsolateral PFC (dlPFC) and insula during controllable pain and increased connectivity between the mPFC and AI during uncontrollable pain, suggesting that these pain modulatory regions exert their effects on pain perception through modulation of brain regions associated with the affective aspects of pain. Similar pain modulation through connections between the dlPFC and AI may contribute to lowered pain perception that may be associated with cultural traditions that emphasize a biological (internal) versus divine (external) control over pain.

## Culture, brain, and nociception

### Population genetic differences and nociception

Because nociception, the sensory transduction of an injurious or potentially injurious stimulus, is the stage of the pain experience most directly tied to an external physical stimulus, it may be least influenced by cultural factors. However, a handful of studies have documented population differences in the frequency of single nucleotide polymorphisms (SNPs) in genes related to nociception and descending pain modulation (Denk et al. 2014). Interactions between gender, ethnicity, and SNPs in the capsaicin receptor gene (*TRPV1*), mu-opioid receptor gene (*OPRM1*), and catechol-O-methyltransferase gene (*COMT*) have been found to explain a proportion of variability in sensitivity to cold pressor, thermal heat, and pressure pain (Fillingim et al. 2005; Kim et al. 2004; Martínez-Jauand et al. 2013). The A118G allele of the mu-opioid receptor gene OPRM1, which African Americans are less likely to carry than other ethnic groups (Gelernter et al. 1999), is associated with decreased experimental pain sensitivity in Non-Hispanic Whites and increased pain sensitivity in Hispanics and Asians (Hastie et al. 2012; Tan et al. 2009). Finally, the short (S) allele of the serotonin transporter polymorphism (5-HTTLPR), more prevalent in collectivistic cultures (Chiao and Blizinsky 2010), has been associated with decreased analgesic efficacy (Ma et al. 2016), increased distress and distress-related brain activation (Ma et al. 2014), and an increased risk for mood disorders, which may be reduced by collectivistic cultural values (Chiao and Blizinsky 2010). Despite limitations to pain genetics research, such as the frequent use of small sample sizes (Kim et al. 2009; Nielsen et al. 2008), these group differences in pain-related SNPs may contribute to sociocultural differences in pain report.

*Neurobiological mechanisms connecting population genetic differences to nociception*

Previously identified SNPs related to pain sensitivity may exert effects at various points in the nociceptive process. For example, SNPs in the capsaicin receptor TRPV1 gene may affect pain sensitivity through increased sensitization of central and peripheral TRPV1 receptor-expressing nociceptive fibers (Jara-Oseguera et al. 2010). The A118G allele of the OPRM1 gene may influence pain sensitivity through increased binding affinity for beta-endorphin, an endogenous opioid (Fillingim et al. 2005). Finally, the Met allele of the COMT gene may affect the enzymes that regulate dopamine and norepinephrine levels in the brain, subsequently influencing mood and pain sensation (Zubieta et al. 2003).

## Culture, brain and pain responses

The third section of our model examines how cultural factors may influence an individual's response to a painful event, focusing on emotional responses to pain and pain coping styles.

### Cultural differences in emotional responses

Psychological studies have documented cultural variability in emotional response styles, which may contribute to sociocultural group differences in pain report. For example, individuals from East Asian cultural contexts, such as China, have been found to prefer low arousal emotions such as calm (Tsai et al. 2006) and be more tolerant of negative emotions (Curhan et al. 2014), whereas individuals from Western contexts, such as the US, have been found to prefer high arousal emotions. These East–West differences in emotional response preferences are thought to stem from Confucian, Taoist, and Buddhist teachings (Peng and Nisbett 1999). There is also some more limited evidence of similar cultural differences in actual emotional responses. For example, Tsai and Levenson (1997) found that Chinese-American couples reported less positive and less variable emotional responses to a relationship stressor than European American couples, and Lang and Bradley (2007) found that Italians reported higher emotional arousal than both Germans and Americans in response to pictures in the International Affective Picture System (IAPS). Some parallel cultural differences in emotional behavior and peripheral physiology have also been found (Soto et al. 2005; Tsai and Levenson 1997), suggesting that at least some cultural differences in reported responses may reflect differences in internal emotional experience. As heightened emotional responses to pain have been associated with increased pain perception (Lumley et al. 2011), cultural preferences for lower arousal emotions may serve to decrease pain perception and pain report.

*Neurobiological mechanisms connecting emotional responses to pain*

Heightened activity within the ACC and amygdala in response to pain has been associated with greater negative affect (Wiech and Tracey 2009), heightened physiological responses to pain (Dube et al. 2009), and increased perceptions of pain unpleasantness (Rainville et al. 1997). One of the mechanisms by which negative emotion may increase perceived pain is through the descending pain modulatory system, including the PAG and NRM in the brainstem, which ultimately suppress or enhance incoming pain signals from the spinal cord (Gebhart 2004; Roy et al. 2009). Emotion-related facilitation of ascending pain signals may play a role in enhancing perceived pain in cultures where high arousal emotions are favored. Brain regions such as the mPFC have been consistently implicated in more general (non-somatic) emotional responses (Phan et al. 2002), and thus may also be important in connecting culturally tuned emotional responses with group differences in pain report.

## Cultural differences in pain coping

Cognitive responses to pain such as coping are also shaped by social and cultural factors (Quartana et al. 2009). For example, the use of catastrophizing, a negative pain coping style that involves exaggerated negative thinking (Sullivan et al. 2001), has been found to vary by culture and nationality (Hsieh et al. 2010). In the US, African Americans report more pain catastrophizing than Whites in response to clinical and experimental pain (Edwards et al. 2005; Hastie et al. 2004). The perceived controllability of pain, and the closely related concept of self-efficacy, are two positive aspects of pain coping that have also been found to vary by cultural context (Crisson and Keefe 1988; Scholz et al. 2002). Another common positive coping strategy, the use of religion and spirituality, has been found to differ between

US ethnic groups, with Hispanics and African Americans more likely to utilize religion and prayer to cope with pain than non-Hispanic Whites (Abraído-Lanza et al. 2004; Jordan et al. 1998). As the use of catastrophizing has been found to predict greater chronic (Severeijns et al. 2001) and acute (Papaioannou et al. 2009) pain, cultural norms favoring catastrophizing may serve to increase pain. In contrast, perceived controllability and self-efficacy (Samwel et al. 2006), and religious coping (Jegindø et al. 2013) have been associated with decreased experimental and clinical pain perception and improved health outcomes, suggesting cultural emphasis on the use of these pain coping strategies may serve to decrease pain perception and report.

*Neurobiological mechanisms connecting coping to pain*

Brain mechanisms associated with the use of different pain coping styles are starting to be understood. As a negative emotional pain coping style, catastrophizing is associated with greater activation of the brain areas associated with pain's affective and attentional components, in particular the PFC and ACC (Gracely et al. 2004; Seminowicz and Davis 2006). Thus, cultural norms favoring catastrophizing may increase pain through increased activation of these regions. The neural correlates of positive pain coping styles, such as perceived controllability and religious belief, have also been explored. Using real time fMRI feedback, individuals were able to learn to decrease activity within their rostral ACC, implicated in pain affect and modulation, which was in turn associated with decreased pain report (deCharms et al. 2005). Similar pain modulation via the ACC may underlie the pain decreasing effects of cultural emphases on self-efficacy and perceived control in more individualistic cultural contexts. In a study on religious pain coping, Catholics who viewed an image of the Virgin Mary during experimental pain stimulation showed increased pain analgesia and activation of the right ventrolateral PFC (vlPFC) compared to controls (Wiech et al. 2008), suggesting that pain modulation via the vlPFC may be an important mechanism connecting cultural variability in the use of religious pain coping and cultural group differences in pain.

## Culture, brain, and pain communication

The fifth section of our model focuses on the communication and social norms that influence how the pain experience is communicated to others. Cultural differences in communication norms may exert important effects on the relationship between the pain experience and pain report, and may be highly sensitive to context, such as communicating pain to family members or medical professionals (Craig 2009; Fordyce 1988).

### Cultural differences in communication norms

Most relevant to understanding cultural differences in pain report is that cultures have been found to vary widely in the value placed on stoicism, or the endurance of discomfort without external expression. In experimental pain studies, stoicism has been used to explain lower levels of pain reported by East Asians (Hobara 2005), South Asians (Nayak et al. 2000), older adults (Yong 2006), and men (Robinson et al. 2001). Paradoxically, studies ascribing lower pain sensitivity among Asians to stoicism stand in contrast to other experimental studies finding that Asians report more sensitivity to pain compared to Non-Hispanic Whites (Rowell et al. 2011). Cultural factors, such as acculturation, have been proposed as an explanation for these divergent findings (Chan et al. 2013). Individualism-collectivism (Triandis et al. 1988) may also help explain the relationship between culture, pain, and stoicism. Because the needs of the group are prioritized in collectivistic cultures (e.g., in East Asia), outward expressions of negative emotions and physical pain may be perceived as a threat to group harmony, and are therefore discouraged (Gudykunst et al. 1988; Raval et al. 2007). As a result, it is possible that cultural values encouraging stoicism and/or collectivism may contribute to lower pain ratings relative to the internal experience of pain compared to cultural values encouraging pain expressiveness and/or individualism.

*Neurobiological mechanisms connecting communication norms to pain*

Brain mechanisms related to emotion regulation, or the goal-directed practice of consciously or unconsciously modulating one's response to an emotion (Gross 2002), may underlie the pain modulating effects of cultural variability in stoicism. Studies of cognitive reappraisal, the most frequently studied emotion regulation strategy, suggest that general reappraisal consistently activates cognitive control regions in the lateral temporal cortex, which in turn down regulate activity within the amygdala (Buhle et al. 2014). Additionally, a pain specific reappraisal study suggested that connections between the nucleus accumbens (NAc) and ventromedial PFC (vmPFC), implicated in valuation in emotional appraisal, are important for the cognitive modulation of pain (Woo et al. 2015). In contrast, the suppression of emotional expression, an emotion regulation strategy more similar to stoicism, has been associated with an increase, rather than a decrease, in activity within the amygdala and insula, regions associated with negative pain affect (Goldin et al. 2008). The findings of Goldin et al. (2008) suggest that stoic responses to pain emphasized in many East Asian and other cultural contexts may come at a cost of up regulating some aspects of the internal pain experience, which may explain the paradoxically higher experimental pain reports observed in Asians in some studies (Rowell et al. 2011). However, in a cross-cultural study, Murata et al. (2012) found that, compared to European Americans, East Asians were able to effectively suppress electrophysiological responses associated with amygdala activity in response to emotionally aversive pictures, suggesting that the extent to which stoicism down regulates neural responses associated with negative affect may be due to the extent to which stoicism is a culturally normative communication style.

## Cultural differences in social norms

Cultural variability in social norms governing communication with medical professionals likely affects the communication of pain. For example, trust in medical practitioners has been found to vary across cultures, with higher levels of trust typically associated with freer communication (Fuertes et al. 2007). In the US, religiously active individuals have been found to have more trust in physicians (Benjamins 2006; Tarn et al. 2005), and those practicing Judaism, Catholicism, and mainline Protestants expressed more trust in physicians than evangelical Protestants (Benjamins 2006). Another cultural factor affecting provider trust and communication is minority status, with ethnic minorities reporting lower trust in providers overall (Doescher et al. 2000), and particularly poorer trust and communication with providers who do not share their cultural or ethnic background (Cooper et al. 2003; Schouten and Meeuwesen 2006). Finally, cultures that place a high value on social hierarchy, such as cultures in Indonesia (Claramita et al. 2013) and China (Kaba and Sooriakumaran 2007), have been associated with a more paternalistic physician communication style, compared to the more collaborative communication style preferred in the US. The family is another context in which cross-cultural differences in norms affect the communication of pain. In collectivistic cultures, although stoicism is generally encouraged, the communication of negative emotions, such as sadness and pain, is viewed as more appropriate in the context of family and close friends than in more distant relationships (Matsumoto et al. 2008). For example, parents among the collectivistic Isan people of northeastern Thailand encourage children to only express pain in the presence of parents rather than strangers or health care providers until the pain becomes unbearable (Jongudomkarn et al. 2006). A similar discouragement of children's pain expression in front of strangers has been found in Arab-Muslim cultures (Zahr and Hattar-Pollara 1998). Thus, a cultural emphasis on social hierarchy and collectivistic values as well as minority status may increase the experienced pain to communicated pain ratio, whereas more intense religious practice, at least in a US cultural context, may serve to decrease the experienced pain to communicated pain ratio.

*Neurobiological mechanisms connecting social norms to pain*

Brain mechanisms related to trust, social hierarchy, and individualism-collectivism are relevant to understanding cultural variability in pain communication. In terms of trust, higher amygdala activation has been observed toward individuals perceived as less trustworthy (Winston et al. 2002), an effect thought to be due to increased threat detection toward individuals deemed untrustworthy (Gordon and Platek 2009). Similar increases in amygdala activity have been associated with perceiving racial outgroup members (Hart et al. 2000). Thus, amygdala activation may be heightened during physician-patient interactions in cultural contexts associated with less trust in physicians, such as holding minority status. Amygdala activation has also been associated with heightened pain perception (Simons et al. 2014), suggesting that cultural norms that decrease physician trust and communication may increase perceived pain, which may in turn contribute to the higher levels of pain reported by minority compared to majority group members (Campbell et al. 2005; Edwards et al. 2005). In terms of social hierarchy, Zink et al. (2008) found higher ventral striatum (reward system) activity when participants interacted with high status individuals; however, Ly et al. (2011) demonstrated that this effect was reversed for individuals who were relatively low status themselves. Thus, physician-patient interactions in cultural contexts where social hierarchy is emphasized may be accompanied by decreased reward system activation, which may also play a role in both decreased pain communication (Nayak et al. 2000) and increased pain (Rowell et al. 2011). Finally, collectivism has been associated with similar levels of brain activity in the mPFC when making social judgments (mentalizing) about the self and close others (Zhu et al. 2007), which may play a role in decisions to communicate pain and negative emotions more freely with family in collectivistic cultures.

## A sociocultural neuroscience approach to pain: novel contributions

In support of our neurocultural model of pain, we have described numerous examples of connections between culture and pain, from *precursor* states to *pain communication*. We have also described brain mechanisms that have been found to connect each of these stages of the pain experience to pain perception and report. Our model thus serves as a foundation for a sociocultural neuroscience approach to pain that aims to identify the mechanisms underlying group differences in pain report. Our approach differs from previous efforts to describe the multifaceted contributors to pain (e.g., Bates 1987; Craig 2009; Gatchel et al. 2007; Kirmayer 2008; Melzack 2001) in that it delineates the specific cultural processes that may affect the pain experience starting from birth, and proposes potential neurobiological mechanisms underlying these pain-culture connections. Importantly, studies directly testing connections between culture, pain, and the brain are lacking. As a result, the majority of the connections between sociocultural and neural mechanisms related to pain perception and report presented here have been speculative, highlighting the need for more research in the future.

The benefits of studies employing this sociocultural neuroscience approach to pain are twofold. First, understanding how cultural norms, beliefs, and practices may modulate the pain experience, pain report, and their underlying neural mechanisms will broaden our basic understanding of the mechanisms underlying group differences in pain (Losin et al. 2010). Without this mechanistic understanding, group differences in pain report may stand to reify biological, essentialist conceptualizations of race and ethnicity found to contribute to disparities in pain treatment (Hoffman et al. 2016). Second, a sociocultural neuroscience approach to pain may identify mechanisms that can inform culturally sensitive guidelines to improve pain treatment. As transnational migration increases and countries around the world become more diverse, a fuller understanding of the neurobiological differences related to culture will be critical to addressing pain disparities, which remain persistent (Campbell et al. 2012). In conclusion, by connecting the cultural influences on pain described in the anthropological and cross-cultural psychology literature with the neurobiological mechanisms of pain revealed through neuroimaging, a sociocultural neuroscience approach may yield unique insights into how cultural and biological mechanisms work together to shape human behavior, as well as more effectively elucidate how "deep" cultural influences on pain truly penetrate.

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