NEURAL CORRELATES OF SOCIAL PAIN

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In elementary school, many schoolchildren are introduced to the idiom, “Sticks and stones may break my bones, but words will never hurt me.” This phrase implies not only that physical pain is entirely distinct from the negative feelings associated with a social blow, but that physical pain is far more crushing than anything inflicted by social experience, whether it be unintentionally insulting words or explicit social exclusion. However, as decades of psychological research have shown, this common schoolyard phrase does not hold true. Words and actions intended to cause psychological harm, including social exclusion, can hurt very much. In fact, research from the field of social neuroscience has helped us to understand that social pain—the painful feelings accompanying social rejection, exclusion, or loss—and physical pain are not such distinct entities. In fact, findings from social neuroscience have shown that social and physical pain are processed by overlapping neural regions, providing a solid foundation for understanding why social exclusion “hurts.”

As we will outline in this chapter, social and physical pain appear to share underlying neurobiological mechanisms. We will begin by discussing potential reasons for why social and physical pain evolved to share overlapping neural substrates, followed by neuroscience findings that support the social–physical pain overlap. Finally, we will conclude by discussing potential consequences of the shared neural circuitry.

Why Would Social and Physical Pain Share Neural Substrates?

When thinking of a list of basic human needs, vital items such as water, food, and shelter probably come to mind. Social psychologists have suggested that in addition to these basic needs, humans have a fundamental need to seek and
maintain social relationships. In fact, it has been posited that the motivation to seek social connection is necessary for human survival, providing evolutionary advantages such as protection, mating opportunities, and physical resources (Baumeister, Brewer, Tice, & Twenge, 2007). The need to belong, or the drive to establish and maintain social relationships, has been posited to be essential to human existence, just like other basic needs such as the need for water, food, or shelter (Baumeister & Leary, 1995). Failure to sustain strong social ties would thus be associated with detrimental effects on an individual’s health and survival. In fact, decades of research have shown a strong relationship between social isolation and mortality (Holt-Lunstad, Smith, & Layton, 2010), supporting the idea that social relationships are crucial to our survival.

Furthermore, it is generally agreed that discomfort and pain can accompany the lack of any basic needs. This pain can serve as a signal to take action (e.g., seek food, avoid similarly threatening stimuli in the future) in order ultimately to improve the chances of survival. Similarly, given the aforementioned importance of social groups in promoting survival, a lack of social connection may be experienced as painful in order to increase the likelihood that social bonds are maintained. Thus, experiencing social rejection or events that could signal social exclusion (e.g., social evaluation, loss of social ties) may feel painful because this was associated with greater chances of injury and death in ancestral times. Indeed, it has been suggested that over the course of mammalian history, the physical pain system was co-opted by the social attachment system, using pain signals to regulate social relationships (Eisenberger, 2012; Eisenberger & Lieberman, 2004; Panksepp, 1998).

Some evidence supporting this overlap between the systems regulating physical and social pain comes from the shared language used to describe both situations. Words used to describe physical pain are also used in the context of socially painful experiences. For example, individuals often describe their experiences of social rejection or loss of social connection by complaining of broken hearts and hurt feelings much in the same way they would complain of a broken bone or a hurt finger. After a particularly painful episode of social rejection, individuals may describe their experience by saying they feel crushed or emotionally scarred. In fact, because the English language has no synonym for the term hurt feelings, words used for physical pain are the only way to describe this particular phenomenon. Moreover, using these physical pain words to describe socially painful experiences is not unique to the English language. Numerous languages across the globe have overlapping language to recount moments of exclusion and physical pain (MacDonald & Leary, 2005).

In addition to this linguistic evidence, pharmacological studies provide further evidence of the overlap between physical and social pain by showing that both types of pain rely on shared neurochemistry. Indeed, certain types of drugs can have similar effects on both types of pain. Opiates (e.g., morphine, heroin), which are often used to treat physical pain, can also have alleviating
effects on social distress. For example, low doses of opiates can reduce behaviors associated with social distress in non-human infant mammals when they are separated from their mothers (Panksepp, Herman, Conner, Bishop, & Scott, 1978). It has also been suggested that opiate abuse in humans may partly be a result of desires to alleviate negative social experiences (Panksepp, 1998), consistent with animal studies showing greater opiate consumption among animals who have been socially separated (Alexander, Coambs, & Hadaway, 1978). Similar to opiates, anti-depressants are also used to relieve both types of pain. Although typically prescribed to treat anxiety and depression, anti-depressants are also effective in alleviating physical pain (Shimodozono et al., 2002; Singh, Jain, & Kulkarni, 2001) and are commonly used to treat chronic pain conditions.

Finally, as outlined in the following sections, neuroscience research suggests that social and physical pain may share underlying neural structures, providing further evidence for the overlap of these two systems.

**Physical Pain in Humans**

When asked to describe an experience of physical pain, such as having joint pain from a recent injury, we may say something such as, “I felt a burning sensation in my left knee,” describing the quality and location of the pain. Alternatively, we could say something like, “My knee pain is really bothering me,” revealing the distressing or unpleasant nature of the pain. Pain researchers refer to these two subcomponents of pain as the sensory and affective components of pain. Although these two components are certainly interrelated, they are also separable. The sensory component is involved in detecting discriminative aspects of pain such as the location, quality, and intensity of the pain; and the affective component is associated with detecting the subjective experience of pain, such as how distressing, unpleasant, or bothersome it is.

These two subcomponents of pain also have distinguishable neural substrates (see Figure 11.1). The sensory component of pain is largely processed by the primary and secondary somatosensory cortices (S1 and S2, respectively) and the posterior insula (PI; Apkarian, Bushnell, Treede, & Zubieta, 2005; Bushnell et al., 1999; Schnitzler & Ploner, 2000). Supporting the idea that these regions underlie the sensory component of pain, patients with lesions in the S1, S2, and/or PI demonstrate deficits in pain sensation (Greenspan, Lee, & Lenz, 1999; Greenspan & Winfield, 1992; Ploner, Freund, & Schnitzler, 1999), as well as other sensory deficits (e.g., thermal sensation; Greenspan & Winfield, 1992). Interestingly, some of these lesion patients describe the sensation of experiencing physically noxious stimuli as “clearly unpleasant,” suggesting that the affective component of pain may be preserved, even with loss of the sensory component (Ploner et al., 1999). Furthermore, other work has found that activity in the somatosensory cortex correlates with intensity of pain sensation.
FIGURE 11.1 Neural Substrates of the Sensory and Affective Components of Pain.

Note: In the left panel, the medial view of the brain shows the dorsal anterior cingulate cortex (dACC). In the right panel, the lateral view of the brain shows the anterior insula (AI), the primary somatosensory cortex (S1), the secondary somatosensory cortex (S2), and the posterior insula (PI). The neural regions underlying the sensory component of pain (S1, S2, and PI) and those underlying the affective component (the dACC and AI) are labeled.

(Bushnell et al., 1999), but it does not correlate with ratings of pain unpleasantness (Rainville, Duncan, Price, Carrier, & Bushnell, 1997).

Conversely, the affective component of pain is processed cortically by the dorsal anterior cingulate cortex (dACC) and the anterior insula (AI; Price, 2000; Rainville et al., 1997; Schnitzler & Ploner, 2000; Treede, Kenshalo, Gracely, & Jones, 1999). Lesion studies provide some evidence for the role of the dACC in processing the affective dimension of pain. Chronic pain patients who have undergone a cingulotomy, a surgical procedure in which a portion of the dACC is lesioned in order to treat pain, show reductions in pain-related emotion (Foltz & White, 1962). Interestingly, patients who undergo this procedure can still localize the pain and acknowledge that the pain is “still present,” suggesting that the sensory component of the pain is still intact, but they report that the pain is no longer “distressing,” “bothersome,” or worrying them, indicating that the affective component of pain, processed by the dACC, is impacted (Foltz & White, 1962). Similar to findings for the dACC, lesions of the insula lead to disruptions of pain-related emotion, such that patients can perceive pain, but they are not distressed by it (Berthier, Starkstein, & Leiguarda, 1988).

Neuroimaging studies provide additional evidence for the role of the dACC and AI in the affective component of pain. In one study, healthy control participants, through the use of hypnotic suggestion, were able to increase the unpleasantness of painful physical stimuli (increasing the affective component) without experiencing the stimuli as more intense (not impacting the sensory component); furthermore, increases in the experience of unpleasantness were
correlated with increases in dACC activity (Rainville et al., 1997). Other work has shown that self-reports of the unpleasantness of pain correlate specifically with activity in the dACC (Tölle et al., 1999). Similar to the dACC findings, it has been shown that activity in the AI correlates with the self-reported unpleasantness of pain (Schreckenberger et al., 2005). Together, these studies provide support for the involvement of the dACC and AI in processing the affective component of pain.

In addition to signaling the distress or unpleasantness of a painful experience, the affective component of pain is also believed to be involved in motivating behavior to reduce or escape the source of pain (Eccleston & Crombez, 1999). Given this role of the affective component in driving the termination of a painful experience, it has been hypothesized that the neural regions associated with the affective component of pain may underlie the experience of social pain in order to prevent the dangers that may result from social exclusion or loss (Eisenberger, 2012; Eisenberger & Lieberman, 2004; Eisenberger, Lieberman, & Williams, 2003). While the sensory component of pain may also be involved in some instances of social pain (Gudmundsdottir, 2009), the affective component of pain and its neural correlates appear to be directly involved in negative social experiences.

Social Pain Processes in Non-human Animals

One line of evidence for the involvement of the affective component of pain in processing social pain comes from animal studies. Although findings from animal studies cannot speak to the experiential component of social pain, they can provide evidence for neural regions underlying behaviors related to threats to social connection, including social separation. In mammalian species, infants are dependent on a caregiver for care and protection, and separation from the caregiver dramatically reduces the chances of infant survival. Because of this, when infants are separated from a caregiver, they will emit distress vocalizations, which are thought to convey separation-related distress to reduce separation from the caregiver by signaling the caregiver to return to the infant (MacLean, 1985).

Although these distress vocalizations in animals are not necessarily the product of socially painful experiences, they are a behavioral indicator of sensitivity to social separation, and interestingly, the ACC—a neural region involved in the affective component of pain—appears to be a major contributor to these attachment-related distress vocalizations. For example, when dorsal and/or ventral regions of the ACC are lesioned in monkeys, these distress vocalizations are diminished, but other vocalizations remain intact (Hadland, Rushworth, Gaffan, & Passingham, 2003; MacLean & Newman, 1988). Conversely, when these subregions of the ACC are electrically stimulated, monkeys spontaneously produce distress-related vocalizations (Smith, 1945). Furthermore, when other neural regions (e.g., regions responsible for speech production) are stimulated,
monkeys do not emit these distress-related vocalizations (Ploog, 1981). Together, these findings suggest a specific role for the ACC and the affective component of pain in this behavior related to social separation.

Social Pain in Humans

As discussed above, lesion studies have provided important information about the neural regions underlying physical pain in humans and social pain in non-human animals. However, there have been very few studies examining the effect of dACC lesions on social behavior in humans. From the few studies that have examined the impact of cingulotomy on social changes in humans, there is evidence for the idea that the dACC contributes to social motivation. For example, patients who have undergone cingulotomy show reduced concern about the social judgments of others (Tow & Whitty, 1953) and reduced self-consciousness (Le Beau, 1954; Tow & Whitty, 1953). These studies suggest that the dACC plays a role in social psychological experience, but no studies have specifically examined the impact of dACC lesions on changes in socially painful experiences, such as social rejection, or on experimentally induced social pain.

Although lesion studies in humans have not looked explicitly at changes in social pain experience, experimental social neuroscience research since the mid-2000s has supported the notion that many of the neural regions involved in physical pain, particularly the areas associated with the affective component of pain (dACC, AI), are also key regions for experiences of social pain. In addition to the evidence that social pain activates regions related to the affective components of physical pain, a few studies (reviewed in Eisenberger, 2012) have found that socially painful experiences also increase activity in sensory-related regions (e.g., S2, P1) involved in physical pain processes. Although the majority of experimental tasks examining social pain have focused on rejection or evaluation by strangers, a few studies have also looked at rejection by close others and neural responses to bereavement. These findings will be discussed in detail below.

Cyberball

In the first study to examine neural responses to social exclusion (Eisenberger et al., 2003), participants were told that they would be playing a virtual ball-tossing game called “Cyberball” (Williams, Cheung, & Choi, 2000). They were led to believe that they would be playing the game with two players over the Internet while they were in the scanner, but in reality, the game was preset so that the other two players were computer-controlled. The game was programmed so that the participant was at first included in the game, tossing the ball to the two other players and receiving it from them for the entire duration of the scan. After this, the game was set to exclude the participant, so
that the two other computer-controlled players stopped throwing the ball to
the participant.

In response to being excluded from the game, participants reported feeling
significant levels of social distress, assessed by items such as “I felt rejected,”
and “I felt invisible.” Neurally, participants had increased activation in areas
associated with the affective component of pain; participants showed increased
activation in the dACC and AI in response to social exclusion versus inclusion.
Moreover, greater activity in the dACC was associated with increased self-
reported feelings of social distress; participants who showed greater dACC
activation in response to social exclusion also reported feeling more distressed
by being socially excluded.

Several other neuroimaging studies have subsequently been conducted using
the Cyberball paradigm and have found similar results, including increased activity
in the dACC and/or AI in response to social exclusion (Bolling, Pelphrey, &
Vander Wyk, 2012; Bolling et al., 2011b; DeWall et al., 2010 (all reviewed in
Eisenberger, 2012); see also Krill & Platek, 2009; Masten, Telzer, & Eisenberger,
2011; Masten, Telzer, Fuligni, Lieberman, & Eisenberger, 2012). Many studies have
also found a positive correlation between greater self-reported social pain
following the Cyberball game and greater activity in the dACC and/or AI
(DeWall et al., 2012; Eisenberger, Taylor, Gable, Hilmert, & Lieberman, 2007; Krill
& Platek, 2009; Masten et al., 2009; Masten, Telzer et al., 2011; Masten et al., 2012;
Onoda et al., 2009). Moreover, a recent meta-analysis has confirmed a role for
the dACC in social pain (Rotge et al., 2014). These findings across many studies
provide converging evidence for the notion that neural regions involved in the
affective component of physical pain also play a role in processing social pain.

Furthermore, neuroimaging studies using the Cyberball paradigm have also
found that individual difference factors that are related to sensitivity to social pain
also moderate neural responses to social exclusion—factors thought to decrease
sensitivity to social rejection are associated with decreased activity in the dACC
and/or AI. For example, higher levels of social support are typically thought to
attenuate social distress; neuroimaging findings parallel this notion: Individuals
with higher levels of social support or who spent more time with friends show
reduced dACC activation in response to social exclusion (Eisenberger, Taylor,
et al., 2007; Masten et al., 2012). Similarly, individuals with an avoidant attach-
ment style are expected to be less sensitive to signs of social rejection (Fraley &
Shaver, 2000). Indeed, an avoidant attachment style is associated with dampened
activity in the dACC and AI in response to social exclusion (DeWall et al., 2012).

Conversely, factors that are thought to increase sensitivity to social exclusion
are associated with increased activity in the dACC and/or AI. Interpersonal
sensitivity, defined as an increased sensitivity to the thoughts, behaviors, and
feelings of others, is an obvious candidate for increased neural activity in pain-
related regions; in fact, interpersonal sensitivity is associated with increased
activation in the dACC (Eisenberger, Way, Taylor, Welch, & Lieberman, 2007).
Similarly, low self-esteem is associated with both increased activation in the dACC and feeling more hurt in response to social exclusion (Onoda et al., 2010). Finally, while an avoidant attachment style is associated with decreased sensitivity to social exclusion, an anxious attachment style is thought to increase sensitivity to social exclusion. In fact, anxious attachment style is associated with greater activity in the dACC and AI (DeWall et al., 2012). Together, these findings suggest that individual difference factors that moderate sensitivity to experiences of social rejection are also relevant to neural activation in the dACC and/or AI in response to social exclusion.

Interestingly, Cyberball studies using adolescent populations have shown that another portion of the ACC is also activated in adolescents in response to social rejection. The subgenual ACC (subACC) is frequently activated in response to the Cyberball social exclusion task in adolescents (Gunther Moor et al., 2012; Masten et al., 2009; Sebastian et al., 2011), suggesting that there may be a potential developmental role for the subACC in processing social pain. Indeed, this is consistent with prior work showing that dACC responses to threatening stimuli do not become evident until later in development (Hung, Smith, & Taylor, 2012). However, more work is needed to flesh out in more detail the role of the subACC in processing social exclusion in adolescence and its developmental relationship with the dACC in the context of social exclusion.

Neural responses to social exclusion in certain clinical populations have also been studied using Cyberball, including populations with autism spectrum disorders (Bolling et al., 2011a; Masten, Colich, et al., 2011), alcohol dependence (Maurage et al., 2012), and schizophrenia (Gradin et al., 2012). Many of these studies have found that neural responses to social exclusion are significantly different in these clinical populations compared to healthy controls, and even studies using sub-clinical assessments of psychopathology (e.g., sub-clinical individual differences in core features of alexithymia; Chester, Pond, & DeWall, 2015) have found alterations in dACC activity in response to social exclusion, suggesting that neural processing of social pain may be involved and important to understand in affective and psychiatric disorders.

_**Romantic Rejection**_

While the majority of neuroimaging studies of social rejection have looked at responses to rejection by strangers (e.g., Cyberball), a handful of studies have examined neural activity during rejection from a romantic partner, which is a potentially more painful rejection experience. In the first of these studies (Fisher, Brown, Aron, Strong, & Mashek, 2010), all participants had recently experienced rejection from a romantic partner in the form of an unwanted break-up, and participants reported still being intensely “in love” with the partner who rejected them. While in the MRI scanner, participants were
shown pictures of the partner who had romantically rejected them and were told to think about events that had occurred with the former partner. As a control, participants were also shown pictures of a neutral, familiar acquaintance and told to think of neutral events that had occurred with this person. Thinking about the former partner, compared to thinking about the neutral acquaintance, was associated with heightened activation in the dACC and AI (affective component of pain), as well as the PI (sensory component of pain), supporting other work on neural responses to social pain.

Another study examining neural responses to romantic rejection used a similar paradigm (Kross, Berman, Mischel, Smith, & Wager, 2011). Participants, who had all recently experienced an unwanted romantic break-up, were asked to look at pictures of their ex-partner while reflecting on the break-up, and photographs of a friend were used as a control condition. In line with other work on social pain, heightened activity in the dACC and AI were found in response to reflecting on rejection by the former romantic partner (versus thinking about the friend). Interestingly, in addition to the increased activity in these affective components of pain, this study also found increased activity in multiple regions associated with the sensory-related components of pain, including the S2 and the PI. Furthermore, this study also included a physical pain task, and the authors found overlapping neural activity in these pain-related regions in response to the physical and social pain tasks. The overlap in neural activity across physical and social pain tasks in this study provides further support for the notion of shared neural circuitry between physical and social pain.

Another study using a “speed-dating” paradigm looked at neural responses to rejection from potential romantic partners (Cooper, Dunne, Furey, & O’Doherty, 2014). Prior to a scanning session, participants in this study met potential romantic partners in a series of 5-minute “dates,” and they decided whether they wanted to see each partner again. In the scanner, participants were shown pictures of all the potential partners they met. They were also reminded of their own interest in seeing the partner again, and were shown whether the partner was interested in them or rejected them. In order to determine neural activity in response to romantic rejection, the authors compared instances in which participants were rejected (i.e., the participant was interested, but the partner was not) to instances in which both parties were uninterested (i.e., the participant and the partner both indicated that they were not interested). They found that trials in which participants were rejected by potential romantic partners in whom they were interested activated a cluster in the dACC, similar to findings in other social exclusion studies.

Social Evaluation

In addition to studies looking at outright exclusion, studies examining responses to social evaluation (which signals the potential for social rejection) have found
increased neural activity in affective pain regions. In a study examining neural responses to social evaluation (Eisenberger, Inagaki, Muscatell, Haltom, & Leary, 2011), participants completed an interview about their personality characteristics and were told that another participant (actually a confederate) would listen to and evaluate their interview. While in the MRI scanner, participants believed that the other participant was evaluating their interview, and they saw what they believed to be feedback in response to their interview. The feedback words had been pre-selected to be rejecting (e.g., “insecure”), neutral (e.g., “practical”), and positive (e.g., “intelligent”). In response to receiving rejecting feedback, participants reported feeling significantly worse, and interestingly, to the extent that participants felt worse, they showed increased activity in the dACC and AI.

Similarly, another study found that when participants were preparing a speech that was later to be delivered to and evaluated by a panel, participants showed heightened activity in the dACC (Wager et al., 2009). Furthermore, another study found that when participants were primed with social-evaluative threat (i.e., told that between 60% and 80% of raters found them unlikable) before having to evaluate themselves on various personality traits compared to their average peer, there was increased activity in the AI (Hughes & Beer, 2013). In another neural study of social evaluation, when participants were made to view themselves negatively, in comparison to a superior peer, there was heightened activation in the dACC (Takahashi et al., 2009).

Finally, in a study looking at neural responses to negative social evaluation, participants were asked to evaluate to what extent various negative personality traits described them (Ma et al., 2014). While in the MRI scanner, participants were shown both positive and negative adjectives and asked to rate to what extent each word could describe them. When participants were rating themselves on negative personality traits, compared to positive traits, there was heightened activity in the dACC and AI. Together, studies examining neural responses to social evaluation, which may signal impending social rejection, provide additional evidence for the role of these affective pain regions in the negative experience of social pain.

Rejection-themed Images and Faces

Other studies have shown that simply viewing rejection-themed images or disapproving faces, cues that signal potential social disconnection, can activate affective pain regions. In one of these studies, participants were shown paintings with rejection themes (Kross, Egner, Ochsner, Hirsch, & Downey, 2007). Paintings by Edward Hopper were pre-rated as depicting themes related to social rejection, disconnection, and loneliness; whereas paintings by Pierre-Auguste Renoir were pre-rated as eliciting thoughts and feelings related to social connection and acceptance. Viewing the rejection-themed paintings, as
compared to the acceptance-themed paintings, led to increased activation in the dACC and AI. Participants in this study were not explicitly rejected, but they still showed heightened activity in affective pain regions in response to even subtle cues of social exclusion.

In another study looking at responses to potential cues of social disconnection, participants were shown videos of individuals making disapproving facial expressions (Burklund, Eisenberger, & Lieberman, 2007). In response to viewing the disapproving facial expressions, individuals who were more sensitive to rejection showed greater dACC activity. Even though the videos were not personally relevant, rejection-sensitive individuals may have interpreted these ambiguous stimuli as potential cues of social rejection, showing greater dACC activation in response.

**Bereavement**

Social loss, another form of social pain, has also been examined using fMRI. Similar to other forms of social pain, grief cues have been found to activate affective pain regions, such as the dACC and AI (reviewed in O’Connor, 2012). In the first study to examine neural responses to grief, participants who had experienced the death of a first-degree relative were asked to look at pictures of their deceased loved one as well as pictures of an age- and gender-matched stranger while inside an MRI scanner (Gündel, O’Connor, Littrell, Fort, & Lane, 2003). The authors compared neural activity while participants viewed pictures of their lost loved one to activity when they viewed pictures of the stranger. They found that viewing pictures of a deceased loved one, compared to viewing pictures of a stranger, was associated with heightened activation of the dACC and AI, among other regions. Being reminded of social loss was associated with increased activity in these pain-related regions.

Another study examining neural responses to grieving a deceased loved one found similar results (O’Connor et al., 2008). Participants in this study, who had experienced the death of a mother or sister to breast cancer, viewed cues of their lost loved one (i.e., a photograph and a word related to the death event) as well as cues of a matched stranger (i.e., a photograph and a matched neutral word). When participants viewed these cues of their deceased loved ones, compared to viewing the cues of a stranger, there was increased activity in the dACC and AI, supporting the findings from the previous study. Interestingly, this study also found that one additional pain-related region, the periaqueductal gray (PAG), had increased activation when comparing neural activity from trials where participants viewed cues of loved ones compared to the cues of strangers.

Similar results were observed in a neuroimaging study of women grieving the loss of an unborn child (Kersting et al., 2009). Women who had experienced the recent loss of an unborn child after induced termination, as well as a control
group of women who had recently healthily delivered a baby, were shown images of happy babies (all of whom were unfamiliar to the participant) while undergoing an MRI scan. The bereaved group, compared to the control group, showed greater activation in the dACC and PAG when viewing the happy faces of babies. Thus, this study found further evidence for the involvement of pain-related neural regions in social loss, adding support to the idea of an overlap between the neural regions involved in physical and social pain.

Summary

In response to various forms of social pain, there is increased activation in regions associated with the affective components of pain, including the dACC and AI. A few studies have also found heightened activation in neural regions that process the sensory component of pain, including the S2 and PI. Whether looking at responses to subtle cues of rejection, social evaluation, exclusion by strangers, or rejection from a close other, social neuroscience findings converge on evidence for an overlap between the neural regions responsible for physical and social pain.

However, even though the dACC has been reliably activated in studies of social pain, it has been argued that the dACC response to social exclusion may reflect the processing of an expectancy violation (i.e., being excluded when expecting to be included) rather than the processing of the emotional component of social pain (Somerville, Heatherton, & Kelley, 2006). The idea that expectancy violation alone underlies the dACC activation in social rejection studies is belied by the fact that self-reports of social disconnection often correlate with neural activity. As previously mentioned, many of the aforementioned studies have found correlations between neural activity in the dACC (and in some cases, AI) and self-reported distress in response to social pain (DeWall et al., 2012; Eisenberger et al., 2003; Eisenberger, Taylor, et al., 2007; Krill & Platek, 2009; Masten et al., 2012; Onoda et al., 2009), such that greater reported social distress was associated with greater activity in the dACC and/or AI. These correlations between neural activity in pain-related regions and self-reports of social distress provide evidence that activation in these regions is relevant to the negative experience of social rejection, not just expectancy violation alone.

Furthermore, in an attempt to determine whether dACC activation underlies social exclusion or expectancy violation, a recent study investigated the neural substrates of both social exclusion and over-inclusion (i.e., also an expectancy violation, but one that does not involve exclusion) in Cyberball (Kawamoto et al., 2012). In the over-inclusion condition, participants were thrown the ball a surprisingly large percentage (80%) of the time. The results indicated that there was greater activation in the dACC in the exclusion condition relative to the over-inclusion condition, implying that dACC activation during episodes
of social exclusion cannot be attributed to expectancy violation alone, and can probably be attributed to the experience of social exclusion.

Consequences of a Social and Physical Pain Overlap

As we have outlined in this chapter, there is substantial linguistic, pharmacological, and neural evidence supporting the hypothesis that there is an overlap in the systems responsible for physical and social pain. Although the fact of the shared biological substrates of physical and social pain is interesting in and of itself, perhaps even more intriguing are the potential consequences of such an overlap.

Individual Differences

One interesting hypothesis that results from the shared neurobiological underpinnings of physical and social pain is that those who are more sensitive to social pain (e.g., high on rejection sensitivity) should be more sensitive to physical pain, and vice versa. Indeed, there is both correlational and experimental evidence to support this idea of shared sensitivities to both types of pain. For example, individuals who are high on measures of anxious attachment report more physical pain than those with more secure attachment styles (Tremblay & Sullivan, 2010). Additionally, greater levels of state rejection sensitivity, defined as the tendency to anxiously expect and/or intensely react to experiences of social rejection, are correlated with increases in symptoms of physical pain (Ehnvall, Mitchell, Hadzi-Pavlovic, Malhi, & Parker, 2009). Together, these findings support the notion that individuals who are more sensitive to social pain are more sensitive to physical pain. Similarly, patients who have chronic pain have greater fear and avoidance of social interactions than healthy controls (Asmundson, Norton, & Jacobson, 1996), indicating that those who experience greater amounts of physical pain exhibit greater sensitivity to social pain. Experimental work in healthy controls further supports this notion; individuals who are more sensitive to experimental physical pain also report higher levels of social pain in response to social exclusion (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006).

Factors that Increase or Decrease Pain

Given that physical and social pain have overlapping biological processes, factors that influence one type of pain should affect the other type of pain in a parallel manner. For example, factors that decrease physical pain (e.g., opiates, pain relievers) should also decrease social pain. In fact, there is some evidence for this. In a double-blind, placebo-controlled study, taking an over-the-counter pain reliever (Tylenol) over 2 weeks reduced daily self-reported hurt feelings (DeWall et al., 2010). Furthermore, those who took Tylenol showed decreased dACC and
AI activity in response to social exclusion, suggesting that factors which decrease physical pain may also decrease social pain, both experientially and neurally.

Similarly, there is evidence supporting the idea that factors typically thought to decrease social pain (e.g., social support) also decrease physical pain. Individuals who have higher levels of social support also report lower levels of physical pain (Zaza & Baine, 2002). Experimental neuroimaging work provides further support for the notion that social support can attenuate the experience of physical pain. For example, viewing an image of a socially supportive figure leads to reductions in self-reported pain (Master et al., 2009), as well as reductions in dACC and AI activity (Eisenberger, Master, et al., 2011).

Conversely, factors that increase one type of pain should also increase the other type of pain. For example, inflammation, which is known to increase physical pain (Watkins & Maier, 2000), should also increase social pain. Indeed, inflammation can lead to increases in feelings of social disconnection (Eisenberger, Inagaki, Mashal, & Irwin, 2010). Furthermore, greater increases in inflammation are associated with greater activity in the dACC and AI in response to social exclusion (Eisenberger, Inagaki, Rameson, Mashal, & Irwin, 2009).

Finally, factors that increase social pain (e.g., social exclusion) should lead to increases in physical pain. Although there have been some contradictory findings (e.g., DeWall & Baumeister, 2006), there is some evidence that social exclusion is related to increased physical pain sensitivity. In experimental studies examining physical pain sensitivity following social exclusion, participants were randomly assigned to play a game of Cyberball in which they were excluded or included; following the game, participants were exposed to physically painful stimuli. In one study, participants who were socially excluded in the game reported feeling more physical pain to subsequent painful stimuli than participants who were socially included (Bernstein & Claypool, 2012). In another study, among participants who were excluded, those who felt the most social distress about being socially excluded also reported the highest levels of pain in response to the physically painful stimuli (Bernstein & Claypool, 2012; Eisenberger et al., 2006).

Together, these studies provide further evidence for the overlap between physical and social pain by highlighting the expected consequences of such an overlap. While it may not be intuitive that Tylenol should reduce social distress, it is logical when framing it as a consequence of the physical–social pain overlap. There are probably many other consequences of this overlap that have not been discussed here, and future research is also needed to test the boundaries of this overlap.

**Conclusions and Future Directions**

In summary, the findings presented here make a strong case for the notion that social pain “hurts.” Social neuroscience research since the mid-2000s has
provided consistent evidence for an overlap in the neural substrates processing physical and social pain. Although these findings represent great advances in understanding socially painful experiences, there are still some issues that need to be addressed by future research. For example, it is possible that the dACC and AI are more general neural indicators of various kinds of threat, harm, or negative affect (Eisenberger, 2015; Eisenberger & Lieberman, 2004). Thus, future research is needed to determine the specificity of the dACC and AI for pain, as opposed to being a broader “neural alarm” system for survival-relevant threats.

Another issue that warrants further research is whether social pain reliably activates neural regions associated with the sensory component of pain. While most of the current research supports a role for the affective-related neural regions of pain in processing socially painful experiences, some studies have also found activation in sensory-related neural regions in response to social pain. It will be important for future research to determine when social pain activates sensory-related neural regions in addition to affective-related regions.

Finally, it is important to note that although the current research supports an overlap in the neurobiological underpinnings of physical and social pain, this does not mean that physical and social pain are equivalent, or that they are experienced in identical ways. However, the linguistic, pharmacological, and neural evidence pointing to an overlap in the biological processes underlying physical and social pain does help us to understand why experiencing social rejection or loss can feel so painful. Over the course of mammalian history, social pain has served a very important purpose, helping us to avoid loss of social connections, which would minimize our chances of survival.

Given that the ability to process social pain is crucial to survival, similar to the ability to recognize and manage physical pain, it is not surprising that our biological systems, including our brains, have adapted to process social pain similarly to physical pain. By further studying the biological overlap between physical and social pain, we can gain even richer insight into why negative social experiences “hurt,” in much the same way as physically painful experiences. Furthermore, we can develop a more nuanced understanding of physical and social pain, which are both essential to our survival.

References


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