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## THE NEURAL BASIS OF SOCIAL PAIN: FINDINGS AND IMPLICATIONS

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According to most studies, people's number one fear is public speaking. Number two is death. Death is number two. Does that sound right? This means that to the average person, if you go to a funeral, you're better off in the casket than doing the eulogy.

—Jerry Seinfeld

Many would agree with the notion that public speaking is frightening, and some would do nearly anything to avoid it. Indeed, public speaking is often cited as people's number-one fear. However, in the preceding quote, comedian Jerry Seinfeld highlights one of the stranger truths about human beings—namely, that the fear of public speaking is right up there with the fear of death. How can that be possible? When directly compared with the fear of death, the fear of public speaking seems trivial. Death, after all, is the end of one's existence, the termination of all relationships, life, and experience. Public speaking is, well, just that—getting up in front of a group of people and talking. Yet, for many people, just the thought of speaking in front of an audience can make the stomach turn. How is it that the fear of public speaking could even be mentioned in the same discussion as the fear of death?

Research from social psychology suggests that one of the reasons that public speaking may be so feared is because of the evolutionary importance of social inclusion for survival and the increased risk of rejection that comes with speaking to a group (Baumeister & Leary, 1995). The fear of public speaking stems, in part, from the fear of being evaluated negatively and rejected by the people to whom one is speaking. Throughout the evolution of humans and other mammalian species, maintaining close social ties, and thus minimizing

opportunities for social rejection and social isolation, has been critical for survival. From birth, mammals rely on the care and nurturance of a caregiver because of their inability to survive on their own. Later in life, being connected to a social group increases chances of survival by providing shared resources and protection from predators. Thus, over the course of evolutionary history, being separated from a caregiver or from the social group significantly decreased chances of survival. Because of this, even though social rejection may no longer be such a dangerous proposition, modern-day fears of public speaking may be a remnant of human beings' evolutionary past, in which rejection from the social group typically resulted in death.

People's need for social connection not only has left its mark on their most intense fears but also has shaped their underlying neural makeup. My colleagues and I have previously argued that the need for social connection is so important and that the threat of social rejection is so severe that the experience of social rejection is actually processed by some of the same neural machinery that processes physical pain (Eisenberger & Lieberman, 2004), which gives support to the notion that rejection hurts. In fact, some have suggested that over the course of mammalian evolution, the social attachment system, which keeps people connected to close others, may have piggybacked directly onto the physical pain system, borrowing the pain signal to indicate when social ties are threatened or lost (Panksepp, 1998). From an evolutionary standpoint, feeling pain upon social rejection makes a lot of sense. If broken social ties are experienced as painful, individuals will be more likely to avoid situations that might threaten social ties or lead to rejection (e.g., public speaking), hence increasing one's likelihood of inclusion in the group and one's chances of survival. Thus, one of the mechanisms for ensuring social connection may be through the experience of pain during social rejection, and this may be instantiated, in part, through an overlap in the neural systems underlying physical and social pain.

In this chapter, I explore the notion that physical and social pain share similar neurocognitive substrates and discuss what this overlapping neural circuitry means for the experience of social relationships and the dread and pain that come with the possibility or actuality of losing these social bonds. To do this, I examine whether social and physical pain rely on shared neural circuitry and summarize findings from some of my own work that has examined the neural correlates of socially painful experience in humans. I then highlight several studies that have tested novel questions stemming from the hypothesis that physical and social pain processes overlap, such as (a) Are individuals who are more sensitive to physical pain also more sensitive to social pain? and (b) Do factors that increase or decrease social pain (social rejection vs. social support) alter physical pain in a parallel manner? To conclude, I extend the field's understanding of the neural systems underlying physical and

social pain experience by examining related issues such as aggression and race-based rejection.

## IS THERE EVIDENCE FOR A PHYSICAL–SOCIAL PAIN OVERLAP?

One reason to believe that physical and social pain share overlapping mechanisms is that they share a common vocabulary. When individuals describe what it feels like to be rejected or left out, they describe their experience with physical pain words, complaining of hurt feelings, broken hearts, or the pain of rejection. In fact, there are no direct synonyms for the painful feelings that result from broken social bonds other than these physical pain words. It is notable that the use of physical pain words to describe a socially painful experience is a phenomenon that is common to many different languages—not just English (MacDonald & Leary, 2005). However, linguistic evidence alone does not substantiate the claim that physical and social pain processes overlap. A broken heart could simply be a figure of speech and might not actually be experienced as painful.

One way to more convincingly demonstrate an overlap in the mechanisms that support physical and social pain processes is to show that they rely on shared neural circuitry. Here, I review neuropsychological and neuroimaging research suggesting that the dorsal anterior cingulate cortex (dACC), a large structure on the medial wall of the frontal lobe, is one of the key neural structures involved in physical and social pain processes. Though undoubtedly many other neural structures are involved in this overlap, such as the insula, periaqueductal gray (PAG), and dorsomedial thalamus (Panksepp, 2003; see also Chapter 1, this volume), I focus primarily on the dACC, both because of the role that it plays in the distressing experience of physical pain in humans and because of the role that it plays in separation-distress behaviors in nonhuman mammals and social pain experience in humans.

### The dACC and Physical Pain in Humans

Painful experience can be divided into two components: the sensory and affective components (Price, 2000). The sensory component of pain has to do with the intensity of the painful stimulus. Asking about the sensory component of pain can be likened to asking, "How loud is the volume on the radio?" The affective component of pain has to do with the perceived unpleasantness of the painful stimulus, which should be dissociable, at least in part, from the intensity of the painful stimulus. Asking about the affective component of pain can be likened to asking, "How much does the volume of the radio bother you?" The answers to these questions will often be correlated, but each addresses distinct features of experience.

Both neuropsychological and neuroimaging studies demonstrate that the dACC is involved in the affective or distressing component of painful experience, as opposed to the sensory component. Chronic pain patients who have undergone cingulotomy, a surgical procedure in which a portion of the dACC is removed, often report that though they can still identify the source location of the painful stimuli, the pain no longer bothers them (Foltz & White, 1968). Such evidence highlights the unique role that this neural region plays in the distressing or what is sometimes referred to as the "suffering" component of pain experience.

Similarly, neuroimaging studies have shown that the activity of the dACC tracks the affective component of pain experience. Subjects who were hypnotized so as to selectively increase the unpleasantness of noxious stimuli (affective component) without altering the intensity (sensory component) showed increased activity in the dACC without changing activity in primary somatosensory cortex (Rainville, Duncan, Price, Carrier, & Bushnell, 1997). Likewise, self-reports of pain unpleasantness correlate specifically with dACC activity (Peyron et al., 2000; Ploghaus et al., 1999; Sawamoto et al., 2000) and those with greater pain sensitivity show greater dACC responses to painful stimuli (Coghill, McHaffie, & Yen, 2003).

### The dACC and Separation Distress in Nonhuman Mammals

In addition to its role in physical pain, the dACC is also involved in separation-distress behaviors in nonhuman mammals, which suggests that it may also play a role in some forms of social pain experience. Across many mammalian species, infants emit distress vocalizations when separated from their mothers. These vocalizations are thought to reflect separation distress in the infants and serve the purpose of cueing the mother to retrieve the infant to prevent prolonged separation between the two.

With regard to the role that the dACC plays in distress vocalizations specifically, it has been shown that ablation of the dACC in squirrel monkeys leads to decreases in distress vocalizations but not other kinds of vocalizations (Kirzinger & Jürgens, 1982; MacLean & Newman, 1988), whereas electrical stimulation of the dACC in rhesus monkeys leads to the spontaneous production of distress vocalizations (Jürgens & Ploog, 1970; Ploog, 1981; Smith, 1945). In addition, highlighting the specific role of the dACC rather than other neural regions in producing distress vocalizations, stimulation of the area corresponding to Broca's area, an area known to be involved in speech production, elicits movement of the vocal chords but no distress vocalizations in monkeys and apes (Leyton & Sherrington, 1917; Ploog, 1981). Thus, distress vocalizations seem to be distinctly related to dACC activation and not to the activation of neural regions involved in speech production more generally.

On the basis of the involvement of the dACC in physical pain in humans and separation-distress behaviors in nonhuman mammals, the remaining link to be examined is whether this same neural region also plays a role in social pain in human populations. In a series of studies, we examined whether this and other pain-related neural regions were involved in the feelings associated with being socially excluded.

### The dACC and Social Pain in Humans

In the first neuroimaging study of social exclusion in humans (Eisenberger, Lieberman, & Williams, 2003), participants were led to believe that they would be scanned while playing an interactive ball-tossing game—called Cyberball (Williams, Cheung, & Choi, 2000)—over the Internet with two other individuals who were also in functional magnetic resonance imaging (fMRI) scanners. Unbeknownst to participants, they were actually playing with a pre-set computer program. Participants completed one round of the ball-tossing game in which they were included for the entire game and a second round in which they were excluded by the other players, partway through the game (see Figure 2.1). After completing the game, participants exited the scanner and filled out self-report measures of how much social distress they felt in response to being left out (e.g., "I felt rejected," "I felt meaningless").

Upon being excluded from the game, compared with when being included, participants reported feeling significant levels of social distress and showed increased activity in a region of the dACC, very similar to the region of the dACC associated with the unpleasantness of physical pain. Moreover, the magnitude of dACC activity correlated strongly with self-reports of social distress felt during the exclusion episode, such that individuals who showed greater dACC activity in response to social rejection also reported feeling more distressed by the rejection episode. Participants also showed increased activity in the insula, a region known to be involved in processing visceral sensation (e.g., visceral pain) as well as negative affective states (Aziz, Schnitzler, & Enck, 2000; Cechetto & Saper, 1987; Lane, Reiman, Ahern, Schwartz, & Davidson, 1997; Phan, Wager, Taylor, & Liberzon, 2004; Phillips et al., 1997); however, insular activity did not correlate significantly with self-reported social distress in this study.

In addition, in response to social exclusion relative to inclusion, participants showed significant activity in the right ventral prefrontal cortex (RVPPFC), a region of the brain typically associated with regulating physical pain experience or negative affect (Hariri, Bookheimer, & Mazziotta, 2000; Lieberman et al., 2004, 2007; Ochsner & Gross, 2005; Petrovic & Ingvar, 2002; Wager et al., 2004). Consistent with this region's role in emotion regulatory processes, greater RVPPFC activity was associated with lower levels of

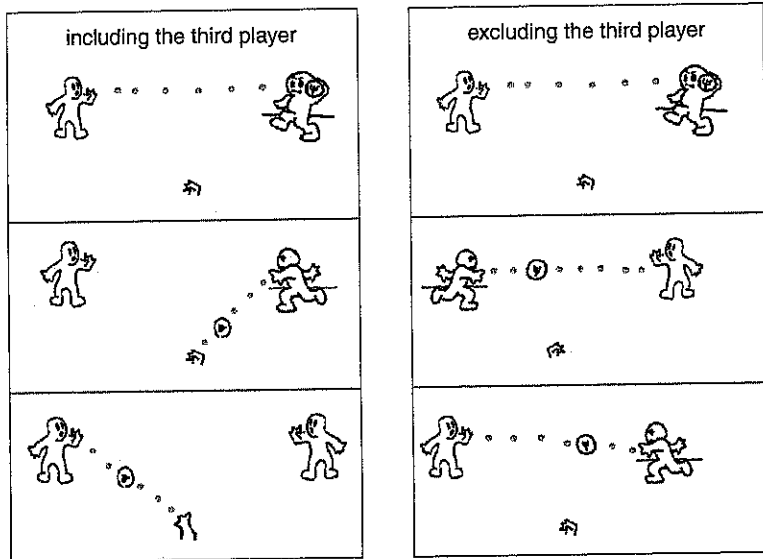


Figure 2.1. Illustration of the Cyberball game during the social inclusion condition (left) and social exclusion condition (right). The subject ("the third player") is represented by the hand at the bottom of the screen. From "Why Rejection Hurts: A Common Neural Alarm System for Physical and Social Pain," by Naomi I. Eisenberger and Matthew D. Lieberman, 2004, *Trends in Cognitive Sciences*, 8, p. 296. Copyright 2004 by Elsevier. Reprinted with permission.

self-reported social distress in response to social exclusion, which suggests that this region may be involved in regulating the distress of being socially excluded. Last, it was found that the dACC was a significant mediator of the RVPFC–distress relationship, such that RVPFC may relate to lower levels of social distress by down-regulating the activity of the dACC.

Thus, neural responses to an episode of social exclusion recruited some of the same neural regions that are involved in the distress (dACC) and regulation (RVPFC) of physical pain experience. In fact, when comparing the neural activations in this study of social pain with those from a study of physical pain in irritable bowel syndrome patients (Lieberman et al., 2004), one notices very similar regions of activation in the dACC and RVPFC (see Figure 2.2; the left panel displays social pain, and the right panel displays physical pain). Moreover, these two studies demonstrate similar patterns of correlations between neural activity and pain distress, such that, in both cases, greater dACC activity was associated with greater reports of social pain or physical pain distress, whereas greater RVPFC activity was associated with

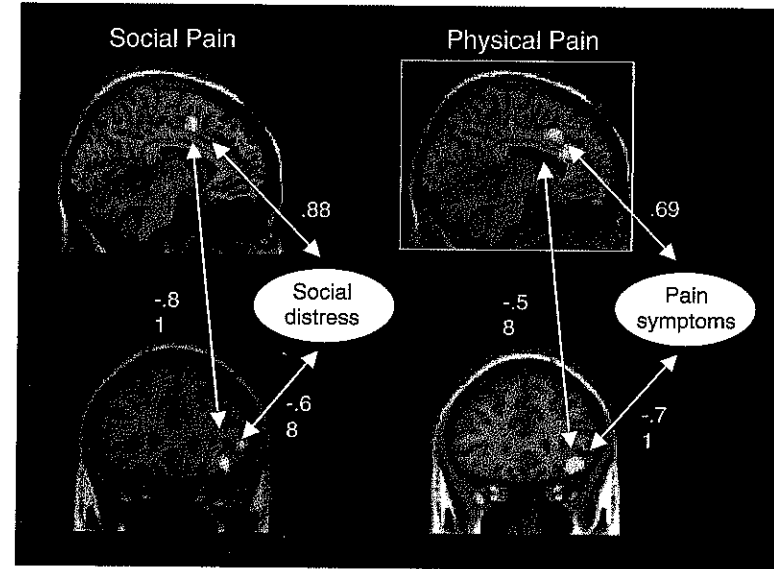


Figure 2.2. The left side of the panel displays the neural activity during social exclusion (compared with social inclusion) that correlates with self-reported social distress. The right side of the panel displays the neural activity during painful visceral stimulation (compared with baseline) that correlates with self-reported pain experience. Data from Eisenberger, Lieberman, and Williams (2003) and Lieberman et al. (2004).

lower reports of distress and less dACC activity. Thus, not only do physical and social pain recruit some of the same neural regions, but, for both types of pain, these neural regions relate to painful or distressing experience in similar ways.

We subsequently demonstrated that individuals who tend to feel more rejected in their everyday social interactions also showed greater activity in pain-related neural regions in response to social rejection (Eisenberger, Gable, & Lieberman, 2007). In this study, participants completed a 10-day experience-sampling study in which they were randomly signaled at different times during their daily lives and were asked to report on their feelings of social distress in their most recent social interaction (momentary social distress: e.g., "I felt accepted/rejected by my interaction partner"). We then examined how individual differences in real-world momentary social distress related to neural responses to the Cyberball social exclusion task.

Results revealed that individuals who reported feeling greater momentary social distress during their real-world social interactions across this 10-day

period also showed greater dACC activity in response to being rejected in the Cyberball social exclusion task. In addition, individuals who reported greater momentary social distress in their daily lives also showed greater activity (in response to social exclusion) in the amygdala, a neural region involved in affective processing (Davis & Whalen, 2001), and in the PAG, a neural region involved in pain processing and attachment-related behaviors (Bandler & Shipley, 1994). Thus, even real-world experiences of social rejection seem to relate to greater activity in pain-related regions of the brain.

As further evidence that social pain stimuli activate pain-related neural circuitry, we examined neural activity as individuals watched short film clips of actors making "disapproving" facial expressions (Burklund, Eisenberger, & Lieberman, 2007). A disapproving facial expression connotes that one has done something wrong or inappropriate and that one risks being rejected because of it. To the extent that disapproving facial expressions signify the possibility of social rejection, participants, particularly those who are rejection-sensitive (Downey & Feldman, 1996), should show greater activity in regions such as the dACC in response to watching these facial expressions unfold.

In this study, participants watched a series of 3-s film clips in which targets displayed specific facial expressions. Participants watched blocks of disapproving facial expressions, in which the actor raised one side of the upper lip, lowered the inner corners of the brow in a fashion similar to that displayed when expressing confusion, and slightly tilted or pulled the head backwards—similar to the "contempt" faces described by Darwin (1872/1998) and Matsumoto and Ekman (2004). Participants also watched blocks of anger expressions, blocks of disgust expressions, and blocks of a neutral crosshair fixation. Anger and disgust expressions were included to contrast with the disapproving expressions because they share both similar and distinguishing features. Like disapproval, anger and disgust are negative emotional expressions. However, although anger and disgust expressions typically signify physical and contamination threats, respectively (although, in some cases, they can also signify threats to social connection), a disapproving face signifies only a threat to social connection and has no alternative meaning.

As in previous studies of neural responses to emotional faces (Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006; Morris et al., 1996; Phillips et al., 1997; Whalen et al., 2001), participants showed significant activity in the amygdala and various regions of the prefrontal cortex in response to viewing each of these emotional expressions compared with when viewing a neutral crosshair fixation. However, when examining individual differences in rejection sensitivity, we found that individuals who scored higher in rejection sensitivity showed greater dACC activity while viewing the disapproving faces but not while viewing the anger or disgust faces, highlighting a specific role

for the dACC in responding to disapproving faces among rejection-sensitive individuals. Moreover, rejection sensitivity correlated specifically with activity in the dACC in response to viewing disapproving faces but not with the activity of other limbic regions (e.g., amygdala, insula), which suggests that dACC activity, rather than limbic system activity more generally, may be specifically responsive to these cues of rejection.

## Summary

Across diverse languages, individuals use the same words to describe the negative feelings associated with physical injury and social rejection. Moreover, neural data from both animal and human subjects converge to show that some of the same neural regions support both physical and social pain experience. One of these regions, the dACC, has been shown to be involved in the experienced unpleasantness of physical pain, the elicitation of separation-distress behaviors in nonhuman mammals, and the experience of distress following social rejection in humans. Other regions that have also been shown to play a role in these pain processes include the insula and PAG, which encode physical pain experience (Aziz et al., 2000; Bandler & Shipley, 1994; Cechetti & Saper, 1987), as well as the RVPFC, which has been involved in regulating painful as well as generally negative affective experience (Hariri et al., 2000; Lieberman et al., 2004, 2007; Petrovic & Ingvar, 2002; Wager et al., 2004).

Taken together, these data provide solid evidence for a physical-social pain overlap. In the next section, I highlight some of the research that has examined the consequences of such a functional overlap in these pain systems. For example, one implication of a physical-social pain overlap is that individuals who are more sensitive to one type of pain should also be more sensitive to the other. I examine this hypothesis and others in the next section. It should be noted, however, that even though there is evidence to support a functional overlap in physical and social pain processes, these processes certainly do not overlap completely. This fact can be known intuitively because one can differentiate between pain resulting from a relationship snub and pain resulting from physical injury. Moreover, research has identified specific differences between these two types of pain experience. For example, Chen, Williams, Fitness, and Newton (2008) showed that individuals can easily relive the pain of previous relationship breakups or other socially painful events; however, it is much harder, and sometimes impossible, to relive the pain of physical injury. Thus, even though there are certainly ways in which physical and social pain experiences are different, this next section focuses on ways in which these pain processes are similar and the consequences of this similarity.

## WHAT ARE THE CONSEQUENCES OF A PHYSICAL-SOCIAL PAIN OVERLAP?

To the extent that physical and social pain processes overlap, one can generate novel hypotheses regarding some of the functional consequences of such an overlap. One hypothesis is that if physical and social pain sensitivity are governed by the same underlying system, then individuals who are more sensitive to one type of pain should also be more sensitive to the other. A second hypothesis is that regulating or potentiating one type of pain should influence the other type of pain in a similar manner, because influencing one type of pain process should alter the underlying neural system that supports both types of painful experience. I review evidence for each of these hypotheses here.

### Hypothesis 1

*Individual differences in sensitivity to one kind of pain should relate to individual differences in sensitivity to the other.*

To examine whether individual differences in physical and social pain sensitivity correlate with each other, my colleagues and I conducted a behavioral study that measured the extent to which baseline sensitivity to physical pain correlated with sensitivity to a socially painful experience (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006). Participants in this study provided a baseline measure of physical pain sensitivity by rating the temperature at which they perceived a painful heat stimulus delivered to their forearm to be very unpleasant ("pain threshold"). After this, participants were randomly assigned to play one round of the Cyberball game in which they were either included for the entire game or excluded midway through the game and were then asked to rate how rejected they felt during the game.

Results demonstrated that individuals who were more sensitive to physical pain at baseline (e.g., lower baseline pain thresholds) were also more distressed by the social rejection episode (but not by the inclusion episode). Moreover, this relationship remained significant after controlling for neuroticism, which suggests that this relationship cannot be explained solely by a general tendency to report higher levels of negative experience. Thus, as predicted, those who were more sensitive to physical pain at baseline were also more sensitive to the pain of social rejection.

As another test of this hypothesis, we investigated whether genetic differences in opioid-related activity, known for its role in physical pain processes, were also associated with differences in social pain sensitivity (Way, Taylor, Lieberman, & Eisenberger, 2008). In this study, 30 subjects were genotyped so we could examine functional differences in a mu-opioid receptor gene polymorphism (OPRM1; A118G), which has been shown to

relate to individual differences in physical pain sensitivity (Fillingim et al., 2005). Each subject also completed the Cyberball social exclusion task in the scanner so we could examine neural sensitivity to social exclusion and filled out several self-report measures assessing individual differences in traits related to social pain sensitivity.

Results demonstrated that individuals with one or more of the minor G alleles, compared with those homozygous for the A allele, scored higher in self-reported trait rejection sensitivity (Mehrabian, 1976). In addition, in response to social exclusion, individuals with one or more of the minor G alleles showed greater activity in the dACC and anterior insula, neural regions involved in processing physical pain stimuli. These findings map nicely onto recent work showing that infant rhesus monkeys with the minor G allele, compared with those with the common allele, exhibit higher levels of distress vocalizations when separated from their mothers (Barr et al., 2008). Thus, individual differences in a pain-related genetic polymorphism also predicted individual differences in trait social pain sensitivity, evidenced by greater rejection-sensitivity scores, as well as individual differences in state social pain sensitivity, evidenced by greater pain-related neural activity in response to social rejection.

### Hypothesis 2

*Factors that regulate or potentiate one type of pain should have a similar effect on the other type of pain.*

To the extent that physical and social pain processes overlap, factors that reduce or enhance one type of painful experience should affect the other type of pain in a similar manner. Few studies have directly examined this hypothesis because it is not necessarily intuitive to measure feelings of social and physical pain in the same study. However, some studies have shown these types of effects, and the number of studies that have explicitly tested this notion is increasing.

### Pain-Regulation Effects

Pharmacological studies provide strong evidence for the notion that regulating one type of pain can inadvertently regulate the other type of pain in a complementary manner. For example, opiate-based medications, known primarily for their powerful analgesic properties, have also been shown to reduce the pain of social separation in nonhuman mammals (Panksepp, 1998); infants treated with opiates demonstrated fewer distress vocalizations when separated from their mothers than did those treated with saline (Herman & Panksepp, 1978; Kalin, Shelton, & Barksdale, 1988; Panksepp, 1998; Panksepp, Herman, Conner, Bishop, & Scott, 1978).

Although it is more difficult to experimentally examine opioid-related processes in humans, it is known anecdotally that opiates reduce social pain in humans, potentially contributing to their powerfully addictive nature (Panksepp, 1998). Moreover, an experimental study has shown that acetaminophen, another pain-reducing drug, can reduce social pain in a human sample (DeWall et al., in press). In this study, a daily dose of acetaminophen (i.e., Tylenol), compared with placebo, led to a significant reduction in daily "hurt feelings" over the course of a 3-week period. Moreover, in a subsequent study, participants who took acetaminophen for a 3-week period, compared with those who took placebo, showed reduced pain-related neural activity (dACC, anterior insula) in response to a scanner-based episode of social exclusion.

In a similar fashion, drugs that are primarily thought to reduce social pain have been shown to reduce physical pain as well. For example, antidepressants (e.g., selective serotonin reuptake inhibitors, or SSRIs), known for their capacity to reduce depression and anxiety, which often result from social stressors, have also been shown to alleviate physical pain in humans (Nemoto et al., 2003; Shimodozono et al., 2002; Singh, Jain, & Kulkarni, 2001). In fact, antidepressants are now commonly prescribed to treat chronic pain conditions (e.g., Fishbain, Cutler, Rosomoff, & Rosomoff, 1998).

In addition to pharmacological studies, studies of social support have also demonstrated the regulatory effects of social factors on physical pain. Across numerous studies, social support, a variable that should primarily relate to reduced social pain, also relates to reduced physical pain experience. For example, correlational evidence shows that individuals with more social support also reported feeling less pain during childbirth (Chalmers, Wolman, Nikodem, Gulmezoglu, & Hofmeyer, 1995; Kennell, Klaus, McGrath, Robertson, & Hinkley, 1991), following coronary artery bypass surgery (King, Reis, Porter, & Norsen, 1993; Kulik & Mahler, 1989), and during cancer (Zaza & Baine, 2002). More convincing, however, is experimental evidence that has shown that individuals who were randomly assigned to receive social support from a friend or stranger, compared with those who received no support, reported experiencing less pain during a cold pressor task, a painful task that involves submerging one's arm in ice water (Brown, Sheffield, Leary, & Robinson, 2003). Moreover, merely viewing a picture of one's significant other while receiving a painful heat stimulus led to lower reports of pain unpleasantness than did viewing a picture of a stranger or neutral object (Master et al., 2009).

### Pain-Potential Effects

To date, no experimental studies have examined whether potentiating physical pain also increases social pain; however, correlational evidence

suggests that the two experiences are related. For example, Bowlby (1969) noted that when children experience physical pain, they become much more sensitive to the whereabouts of their caregiver, experiencing distress more frequently and easily upon noting distance from the caregiver. Similarly, compared with healthy controls, adults with chronic pain are more likely to have an anxious attachment style, characterized by a heightened sense of concern with their partner's relationship commitment (Ciechanowski, Sullivan, Jensen, Romano, & Summers, 2003).

A few studies have started to examine the reverse—namely, that experiences that increase social pain can increase physical pain experience too. For example, we tested the notion that feeling socially excluded would lead to an increase in sensitivity to physical pain (Eisenberger et al., 2006). Participants in this study were randomly assigned to play one round of the Cyberball game in which they were either included during the game or excluded midway through the game. During the last 30 s of the Cyberball game (when participants were being either included or excluded), participants were exposed to three painful heat stimuli and were asked to rate the unpleasantness of each. This was done to determine whether being included or excluded changed physical pain sensitivity. After completing this task, participants were asked to rate how rejected they felt during the Cyberball game.

Although there was no main effect of exclusion on pain sensitivity (i.e., rejected subjects did not become more pain-sensitive than did included subjects), results demonstrated that individuals who felt the most distressed by the social rejection episode also reported the highest pain ratings to the heat stimuli that were delivered at the end of the rejection episode, and this effect remained significant after controlling for neuroticism. Thus, even though this finding is correlational, it suggests that augmented sensitivity to one type of pain is related to augmented sensitivity to the other.

It should be noted that these findings are somewhat different from those of another study that examined the effect of social exclusion (using a different manipulation) on physical pain sensitivity (DeWall & Baumeister, 2006). This study was based on the observation that extreme physical pain can sometimes turn off the pain system itself, leading to temporary analgesia or numbness (Gear, Aley, & Levine, 1999). On the basis of this observation, it was hypothesized that, to the extent that physical and social pain overlap, extreme forms of social exclusion should lead to numbness, not only to negative social experiences but to physical pain as well.

DeWall and Baumeister (2006) manipulated social exclusion by telling participants that they would be alone in the future. Participants in this "future alone" condition, compared with those who were given no feedback or who were told that they would have satisfying relationships in the future, showed a reduced (rather than an increased) sensitivity to physical pain. Differences

between these two sets of findings could be due to the underlying nature of the pain system, such that mild pain (e.g., being excluded by strangers during the Cyberball game) augments pain sensitivity, whereas more intense pain (e.g., being told that one will be alone in the future) leads to analgesia (Gear et al., 1999; Price, 2000). It is also possible that the "future alone" manipulation may have induced more depression-like affect, which in some cases has been associated with reduced experimental pain sensitivity (Adler & Gattaz, 1993; Dickens, McGowan, & Dale, 2003; Orbach, Mikulincer, King, Cohen, & Stein, 1997), whereas the Cyberball manipulation may have induced more anxiety-like affect, which has been linked with increased experimental pain sensitivity (Cornwall & Donderi, 1988; Lautenbacher & Krieg, 1994; Melzack & Wall, 1999). Nonetheless, it is important to note that in both studies, physical and social pain sensitivity still seem to be working in parallel. In the first study, greater sensitivity to social rejection was correlated with greater sensitivity to physical pain; in the second, an extreme form of social exclusion resulted in general emotional insensitivity to both social and physical pain.

A final example of the effect of social pain potentiation on physical pain is Gray and Wegner's (2008) examination of whether an intentional interpersonal transgression (i.e., stepping on someone's toe on purpose), which is typically more emotionally hurtful than an accidental transgression, was also more physically painful. Participants believed that another subject, who was actually a confederate, was going to choose which of two tasks the participant was going to complete. In the intentional transgression condition, the confederate chose a task that involved the participant receiving electric shock; in the unintentional transgression condition, the confederate chose a pitch judgment task for the participant to complete, but the participant still received shock as a result of study constraints. Participants were told which task the confederate chose for them and then rated pain unpleasantness as they received a series of electric shocks.

Results revealed that physical pain ratings following the intentional transgression were higher than were those following the unintentional transgression. In addition, though participants in the unintentional transgression condition showed habituation to repeated painful stimulation, those in the intentional transgression condition did not. Thus, social factors that are primarily thought to increase emotional pain seem to affect physical pain in a congruent manner.

## Summary

Identification of an overlap in the neural substrates that underlie physical and social pain leads to several novel hypotheses regarding the ways in which

these two types of painful experiences interact. For example, the studies reviewed here demonstrated that those more sensitive to physical pain were also more sensitive to social pain and that factors that regulate or potentiate one kind of pain have similar effects on the other. There are likely many other consequences of this functional overlap, and it will be interesting to watch as new hypotheses are developed and explored.

## WHAT CAN BE LEARNED FROM UNDERSTANDING THE NEURAL CORRELATES OF SOCIAL PAIN?

Understanding the shared neural circuitry that underlies physical and social pain allows one not only to make novel predictions about how one type of painful experience may influence the other but also to answer questions about other social processes that are challenging to assess with self-reports alone. For example, it is known that aggressive behavior can be either the product of blunted emotional sensitivity, leading an individual to aggress because he or she cares little about the feelings of others, or the product of enhanced emotional sensitivity, leading an individual to aggress defensively in response to negative social treatment (Berkowitz, 1993). Which type of aggressive behavior occurs among those who are predisposed to aggressive behavior on the basis of their underlying genetic makeup?

As a second example, racial discrimination is one form of negative social treatment that some have to deal with on a daily basis. Are experiences of racial discrimination just as painful as the sting of social rejection, or does attributing negative social treatment to race change the dynamics of these events? Later in this section, I review two studies that begin to answer these questions by using neuroimaging tools to investigate the experiential substrates that underlie these social events.

## Understanding a Genetic Precursor to Aggression

Previous work has demonstrated a link between aggressive behavior and a specific genetic polymorphism that encodes monoamine oxidase-A (MAOA), an enzyme that degrades monoamines such as serotonin (Caspi et al., 2002). MAOA-deficient men from a single Dutch kindred demonstrated elevated levels of impulsive aggression, arson, and attempted rape (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993). In addition, when exposed to early adversity, men with the low expression allele (MAOA-L) of the 30-bp variable number tandem repeats polymorphism in the MAOA promoter region (MAOA-uVNTR) were more likely to develop antisocial behavior than were men with the high expression allele (MAOA-H; Caspi et al., 2002). Despite



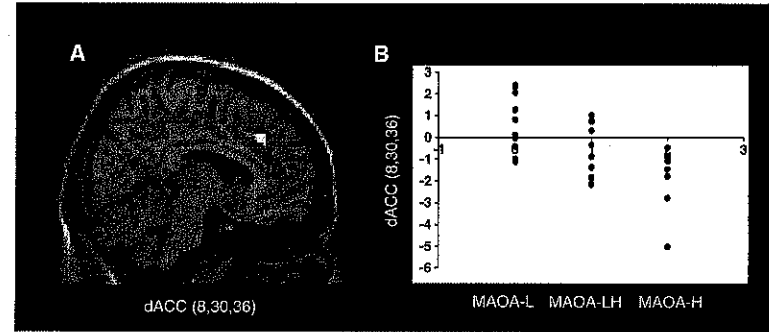
mounting evidence suggesting a relationship between the MAOA-uVNTR and aggressive behavior, it is unclear how this genetic polymorphism predisposes individuals to aggressive behavior.

Many possible mechanisms might help to explain the functional relationship between the MAOA polymorphism and aggressive behavior. One possibility is that MAOA-L individuals show blunted socioemotional sensitivity, making them less concerned with the feelings of others, less empathic, and thus more likely to commit violent crimes because they care less about harming others or the repercussions of doing so (Blair, 2007). Another possibility is that MAOA-L individuals show heightened socioemotional sensitivity, making them more sensitive to negative social experiences such as social rejection and more likely to respond to these experiences with defensively aggressive behavior (Crick & Dodge, 1996; Dodge et al., 2003; Dodge & Pettit 2003; Twenge, 2005; Twenge, Baumeister, Tice, & Stucke, 2001).

To investigate the underlying socioemotional experience that links this genetic polymorphism with aggression, we examined how different allelic variants in the MAOA polymorphism related to neural responses to social rejection (using the Cyberball game) as well as self-report measures of trait aggression (Eisenberg, Way, Taylor, Welch, & Lieberman, 2007). To the extent that the link between MAOA and aggression is a function of blunted socioemotional sensitivity, MAOA-L individuals should show less dACC activity to social rejection (less social pain) than do MAOA-H individuals. On the other hand, to the extent that the link between MAOA and aggression is a function of heightened socioemotional sensitivity, MAOA-L individuals should show greater dACC activity to social rejection (more social pain) than do MAOA-H individuals. In either case, MAOA-L individuals should report higher levels of trait aggression than do MAOA-H individuals.

Consistent with previous work, we found that MAOA-L individuals reported higher levels of trait aggression than did MAOA-H individuals. More important, results indicated that MAOA-L individuals, compared with MAOA-H individuals, showed greater dACC responses to social rejection (see Figure 2.3), which suggests that the relationship between MAOA and trait aggression may be due to heightened, rather than blunted, socioemotional sensitivity, fitting with previous findings (Meyer-Lindenberg et al., 2006). Moreover, the relationship between the MAOA polymorphism and trait aggression was partially mediated by dACC responses to social rejection.

Thus, using knowledge of the neural systems underlying social pain experience, we were able to show that MAOA-related aggression was a function of heightened socioemotional sensitivity—a finding that may have been difficult to assess with self-reports alone, given the sensitive and personal nature of the underlying psychological experience. Clarifying the underlying socioemotional mechanisms that link MAOA to aggression is critical for both



**Figure 2.3.** dACC activity (8,30,36) that varies as a function of the MAOA polymorphism. (A) Activity in the dACC, during social exclusion versus inclusion, that correlates with individual differences in the MAOA polymorphism (maximum activation at 8,30,36) and shows greater activity for MAOA-L, compared with MAOA-H or MAOA-LH (females with one low expression and one high expression allele) individuals. (B) Scatterplot showing the relationship between the MAOA polymorphism and dACC (8,30,36) responses to social exclusion versus inclusion. From "Understanding Genetic Risk for Aggression: Clues from the Brain's Response to Social Exclusion," by N. I. Eisenberger, B. M. Way, S. E. Taylor, W. T. Welch, and M. D. Lieberman, 2007, *Biological Psychiatry*, 61, p. 1105. Copyright 2007 by Elsevier. Reprinted with permission.

understanding the experience of individuals at risk for aggression and for identifying appropriate interventions for treating these aggressive behaviors. Moreover, identifying a genetic correlate of social pain sensitivity may aid not only in the identification and treatment of aggressive disorders but also in the identification and treatment of other clinical disorders that relate closely to sensitivity to social pain as well (e.g., social anxiety, depression).

### Exploring the Neural Correlates of Racial Discrimination

In some ways, racial discrimination can be conceptualized as the routine experience of social rejection. Those who experience racial discrimination perceive that they are being treated unfairly—rejected from a social group, not granted a promotion, or given poorer treatment—because of their race. In light of these hardships, it is natural to ask about the psychological consequences of racial discrimination. In other words, does the repeated experience of rejection, unfair treatment, and disadvantage have negative psychological consequences for the person who is experiencing these events? One would think that it must. Research, however, has debated whether attributing unfair treatment to race makes the experience better or worse.

It is not surprising that some have suggested that attributing negative social treatment to race can be psychologically harmful; if one is maltreated because of one's race, one is likely to continue to be a target of such maltreatment (Schmitt & Branscombe, 2002). However, others have suggested the counterintuitive notion that perceptions of racial discrimination may actually serve a "self-protective" function (Crocker & Major, 1989). To the extent that negative social treatment is due to one's race and not something personal about one's character, the cause of the negative social treatment is external to one's self and thus less psychologically damaging. Because it is inherently difficult to ask individuals who are the target of racial discrimination about whether perceptions of discrimination are painful or protective, we examined the neural correlates associated with feeling that one is the target of racial discrimination. Using neuroimaging tools, one can examine the feelings associated with racial discrimination in a more covert way that is less subject to demand characteristics or self-presentational concerns.

In this study (Masten, Telzer, & Eisenberger, 2008), we examined the neural correlates of felt racial discrimination by investigating the neural regions that are involved when one judges a rejection episode to be due to one's race as opposed to some other factor (e.g., one's personality, one's behavior). African American (AA) subjects ( $n = 18$ , nine female) completed the Cyberball social exclusion task. Each participant was greeted by a Caucasian American (CA) experimenter and two CA confederates, and participants were led to believe that they would be playing the ball-tossing game with the two CA confederates. The subject and confederates were instructed in how to play the game and then spent a few minutes introducing themselves to each other. Race was never mentioned during this meeting session, nor was it mentioned in any of the recruitment materials. Each participant was then scanned while supposedly playing the Cyberball game with the two CA players. In reality, however, participants played with a preset computer game. As in previous studies, the participant was included during the first round of the game and excluded partway through the second round (thus all AA subjects were ultimately rejected by two CA players).

Immediately following the game, participants rated how upset they felt in response to the rejection episode (social distress: e.g., "I felt rejected," "I felt meaningless"). They also rated the extent to which they thought they were rejected because of their race. After completing these self-report measures, each participant completed a face-to-face interview with the experimenter. Here, each participant was asked about his or her thoughts and feelings about the rejection episode as well as his or her possible reasons for why the rejection occurred. Again, race was never mentioned by the experimenter unless the participant spontaneously brought it up, in which case the experimenter asked

the participant to elaborate on his or her thoughts. This interview was videotaped for later analysis.

Consistent with previous work on social pain, AA participants who were judged to be the most distressed during their videotaped interviews showed the most pain-related neural activity (dACC, anterior insula) in response to the social rejection episode and the least emotion regulatory activity (right ventrolateral prefrontal cortex). However, to the extent that AA participants believed that they were rejected because of their race, they showed significantly less activity in the dACC and more activity in prefrontal regions associated with regulating negative affect (medial prefrontal cortex, rostral anterior cingulate cortex; Petrovic & Ingvar, 2002; Phelps, Delgado, Nearing & LeDoux, 2004).

Thus, although observed distress following social exclusion was associated with greater pain-related neural activity, attributing rejection to race was associated with less pain-related neural activity, consistent with the counterintuitive notion that perceiving discrimination can, in some cases, protect one's well-being (Crocker & Major, 1989). Findings such as these would have been difficult to assess with self-reports alone and have important implications for understanding the experiences of those who have to face negative social treatment on a daily basis.

## CONCLUSIONS

Taken together, the research presented here puts forth a strong case for the notion that being rejected hurts. Not only do people use physical pain words to describe rejection experiences, but some of the same neural regions that process physical pain process social pain too. One of the implications of these findings is that episodes of rejection or relationship dissolution can be just as damaging and debilitating to the person experiencing those events as can episodes of physical pain. Thus, even though physical pain conditions may be treated more seriously and regarded as more valid ailments, the pain of social loss can be equally as distressing, as demonstrated by the activation of pain-related neural circuitry to social disconnection as well.

It is important not to forget, though, that though painful in the short term, feelings of distress and heartache following broken social relationships also serve a valuable function—namely, to ensure the maintenance of close social ties. To the extent that being rejected hurts, individuals are motivated to avoid situations in which rejection is likely. Over the course of evolutionary history, avoiding social rejection and staying socially connected to others likely increased chances of survival, because being part of a group provided additional resources, protection, and safety. Thus, the experience of social

pain, while distressing and hurtful in the short term, is an evolutionary adaptation that promotes social bonding and ultimately survival.

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## 3

PHYSIOLOGICAL RESPONSES TO  
EXPERIENCES OF SOCIAL PAIN

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Children who are consistently teased or ostracized, or are always the last ones chosen for the team; people who make fools of themselves in public presentations, or are ridiculed by superiors; and individuals who are put down, criticized, or rejected by relationship partners or because they possess devalued characteristics or social stigmas often experience *social evaluative threat* (SET), which occurs when the self could be negatively judged by others (Dickerson & Kemeny, 2004). Social evaluative experiences can lead to *social pain*—the emotional response to the perception that one is being excluded, rejected, or devalued by a significant individual or group (MacDonald & Leary, 2005). A growing body of evidence suggests that SET is accompanied by specific physiological responses, including changes in the cardiovascular, neuroendocrine, and immune systems (e.g., Dickerson, 2008; Dickerson, Gruenewald, & Kemeny, 2004). Therefore, social evaluative events that induce social pain are capable of eliciting not only intense emotional responses but physiological responses as well. Though the acute experience of social pain may produce short-term changes in physiological parameters, more chronic or prolonged experiences could result in dysregulation of physiological systems, which could lead to negative health outcomes.

# Social Pain

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Neuropsychological and  
Health Implications of  
Loss and Exclusion

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