



# Why It Hurts to Be Left Out

## The Neurocognitive Overlap Between Physical and Social Pain

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Pain Overlap Theory

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Conclusion

*Without friends no one would choose to live, though he had all  
other goods.*

Aristotle

Replace the word “friends,” as quoted above, with the word “air,” “water,” or “food” and Aristotle’s claim is indisputable. Without amending his statement, however, Aristotle’s claim seems more hyperbolic than truthful. If granted all the ‘real’ necessities of life, such as air, water, and food, would we not be able to live or, at least, not want to live, without the companionship of others? Are social relationships something we actually need or are they better

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described as desirable but not necessary? In this chapter we will suggest that social connection is a need as basic as air, water, or food and that like these more basic needs, the absence of social connections causes pain. Indeed, we propose that the pain of social separation or rejection may not be very different from some kinds of physical pain.

We are not alone in this claim. For centuries, writers, musicians, playwrights, and poets have noted that the loss of social bonds can unleash the most profound forms of human pain and suffering. The legal systems of many countries have also recognized this, as evidenced by their use of social isolation as one of the most extreme forms of punishment, at times issued interchangeably with the death penalty for the most severe crimes (Baumeister, 2000). Likewise, the pain of broken social bonds permeates the English language, illustrated by the use of physical pain words to describe episodes of socially painful experiences, such as when speaking of “broken hearts” or “hurt feelings” (MacDonald & Shaw, this volume). Could Aristotle have been right?

Until a half-century ago, most psychologists would have responded with a resounding “no.” Psychologists believed that an infant’s attachment to his or her caregiver was exclusively the result of the association of the caregiver’s face or form with the alleviation of certain drive states such as hunger or thirst (Dollard & Miller, 1950). However, in a series of seminal studies, Harlow (1958) demonstrated that infant rhesus monkeys separated from their natural mothers preferred a cloth surrogate mother that provided them with contact comfort to a wire-mesh mother that provided them with food, indicating the existence of a need, over and above the need for food. This study, along with the others that have followed, emphasized the importance of a mammalian drive that is primarily social, unrelated to hunger or thermoregulation, aimed at maintaining social closeness or social contact. Though it is possible that this need for social closeness may have originally evolved to support a drive for food or warmth, Harlow’s studies indicate that it is now clearly a separate, autonomous need.

We propose that along with the evolution of mammals, a species unique in their need for early nurturance and care, came a corresponding lifelong need for social connection (Baumeister & Leary, 1995; see also, Pickett & Gardner; Williams & Zadro; this volume). Indeed, this need has proved so essential to survival that social separation, like other unmet needs, is experienced as painful. We hypothesize that the pain mechanisms involved in preventing physical harm were co-opted during our evolution to prevent social separation. In this chapter, we suggest that social and physical pain share the same underlying system and that this overlap has several consequences for the way that these types of pain are detected, experienced, and overcome (see also MacDonald & Shaw, this volume). We will refer to this theory as *pain overlap theory* and will present evidence for four hypotheses derived from this proposed overlap.

## PAIN OVERLAP THEORY

Pain overlap theory proposes that social pain, the pain that we experience when social relationships are damaged or lost, and physical pain, the pain that we experience upon physical injury, share parts of the same underlying processing system (Eisenberger & Lieberman, 2004). This system is responsible for detecting the presence or possibility of physical or social harm and recruiting attention once something has gone wrong in order to fix it. Evolutionarily, this overlap makes good sense. Based on mammalian infants' lengthy period of immaturity and their critical need for substantial maternal contact and care, it is possible that the social attachment system, the system that keeps us near close others, may have piggybacked onto the pre-existing pain system, borrowing the pain signal to signify and prevent the danger of social separation (Nelson & Panksepp, 1998; Panksepp, 1998).

This evolutionary hypothesis was first proposed to explain the similar effects of opiates on both physical and social pain. Panksepp (1998) noted that opiate-based drugs, known for their effectiveness in alleviating physical pain, were also effective in alleviating distress vocalizations emitted by the young of different mammalian species when separated from others. Panksepp suggested that the social attachment system may have co-opted the opiate substrates of the physical pain system to maintain proximity with others, eliciting distress upon separation and comfort upon reunion (Nelson & Panksepp, 1998).<sup>1</sup>

For most mammalian species, an initial connection between mother and child is essential for survival as mammalian infants are born relatively immature, without the capacity to feed or fend for themselves. The Latin root of the word *mammal* is *mamma* which means *breast* and bears a striking resemblance to the first word uttered by many infants across many countries, namely the colloquial word for *mother* (English: *mom*, *mommy*; Spanish: *mami*, *mama*; French: *maman*; German: *mami*, *mama*; Hindi: *ma*; Korean: *ama*; Hebrew: *ima*). Thus, the need to maintain closeness with the mother is so critical that the first word uttered by many human infants typically reflects this important underlying motivation, the need for the mother.

Because maintaining closeness with caregivers for food and protection is necessary for the survival of mammalian young, a system that monitors for distance from the caregiver and alerts the individual once a certain distance has been exceeded is critical. Indeed, the pain system may have been co-opted for just this purpose. Due to its aversiveness, pain grabs attention, interrupts ongoing behavior, and urges actions aimed at mitigating painful experience (Williams, 2002). To the extent that social distance is harmful to survival, experiencing pain upon social separation would be an adaptive way to prevent social distance.

The value of pain overlap theory, however, comes primarily from its corollary hypotheses. In this chapter, we will present four of these hypotheses along with

the evidence relevant to each. Pain overlap theory provides an overarching structure and organization to these findings that on their own tend to be interesting but atheoretical. The first hypothesis is that physical and social pain should share a common phenomenological basis and should rely on some of the same neural structures. Second, if both types of pain rely on some of the same neural structures, they should also share some of the same underlying cognitive or computational mechanisms. Third, potentiating or regulating one type of pain should similarly influence the other type of pain. Fourth, traits related to a heightened sensitivity to one type of pain should also relate to a heightened sensitivity to the other type of pain. Before examining these hypotheses, we will first define the terms “physical pain” and “social pain.”

Physical pain has previously been defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (International Association for the Study of Pain, 1979). For social pain, however, there is no pre-existing definition of this term that captures our intended meaning. We conceptualize social pain as analogous to Bowlby’s description of the separation distress that occurs when an infant feels distress due to separation from a caregiver (Bowlby, 1969). In a similar manner, we define social pain as *the distressing experience arising from actual or potential psychological distance from close others or from the social group*.

Psychological distance could include perceptions of rejection, exclusion, non-inclusion, or any socially-relevant cue that makes an individual feel unimportant to, distant from, or not valued by important relationship partners (see also Leary; Baumeister, & DeWall, this volume). Whereas infants may only be capable of detecting actual physical distance from a caregiver, emergent cognitive capacities soon enable young children with the ability to monitor not only objective distance from the caregiver, but also perceived psychological distance from the caregiver, an assessment that relies on a more complex understanding of socio-relational information. In short, social pain can be thought of as the distressing experience associated with *perceived social distance*.

Two factors make social pain a broader and more expansive social experience than Bowlby’s conception of separation distress. First, unlike separation distress, social pain is posited to be an experience that persists throughout the life span. Typically, separation distress is thought to diminish as a child matures and becomes capable of taking care of himself (Bowlby, 1969). However, if the social attachment system borrowed the mechanisms underlying the physical pain system, perceived social distance should continue to cause social pain for as long as the physical pain system is in tact. Indeed this seems to be the case, as evidenced by the occurrence of grieving responses, social anxiety disorders, and depression from social isolation in individuals of all ages. Whether this continued sensitivity to social distance remains adaptive in adulthood or is merely a vestige of the merging of these two systems is not yet known.

Second, based on expanding cognitive capacities that allow certain species to represent, manipulate, imagine, and predict complex social information, many

more cues may be capable of eliciting social pain in mature humans than are capable of eliciting separation distress in infants or other mammals. For example, human adults can experience social pain not only based on the perception of psychological distance from an individual but also based on the perception of psychological distance from a *social group*, a more complex mental representation. In addition, humans can experience social pain or anxiety at the mere *possibility* of social distance. The capacity to represent complex ideas such as the social group or the possibility of social distance may only be possible for those species with cognitive resources that allow symbolic and propositional representations (Deacon, 1992; Lieberman et al., 2002). For instance, human infants can only begin to show fear of *anticipated* situations once they have undergone a critical period of prefrontal cortex maturation, at the end of their first year (Bowlby, 1973; Schore, 2001). Species that show the most expansion of neocortical areas, such as primates, humans, and possibly cetaceans (whales and dolphins; Panksepp, 1998) may be the only mammals capable of showing distress at these more subtle cues of social distance. In the remainder of this chapter, we will present evidence for the four corollary hypotheses derived from pain overlap theory.

### HYPOTHESIS #1: PHYSICAL AND SOCIAL PAIN SHARE A COMMON PHENOMENOLOGICAL AND NEURAL BASIS

The first hypothesis proposes that physical and social pain share a common phenomenological and neural basis. We have already mentioned one reason to believe that these two types of pain share a common phenomenological experience; they share a common vocabulary. In the English language, the same words are used to describe instances of both physical and social injury. Thus, we can have a *broken* bone or a *broken* heart; we can feel the pain of a *stomachache* or of *heartache*; and we can be *hurt* by a dog's *bite* or by another's *biting* remark. Indeed, the use of pain words to describe episodes of physical and social pain is a phenomenon common to many different languages (MacDonald & Shaw, this volume). However, linguistic evidence alone does not substantiate the claim that physical pain and social pain share the same underlying phenomenology. A "broken heart" could simply be a figure of speech and might not actually be experienced as physically painful. Showing that the same neural regions are involved in the experience of both physical and social pain provides more substantial evidence that these two types of pain share a common phenomenological experience.

In this section, we will 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 the affective distress associated with the physical-social pain overlap. Though there are undoubtedly several other neural structures involved in this overlap, such as the insula and periaqueductal gray, we will focus primarily on the role that the dACC plays in the affective component of physical and social pain.

### *Physical Pain and the dACC*

For nearly a century, it has been known that the dACC plays a role in the experience of physical pain. Since the mid-1930s, neurosurgeons have used cingulotomy, a circumscribed lesioning of the dACC, for the treatment of intractable chronic pain disorders (Davis et al., 1994). Following cingulotomy for chronic pain, patients report still being able to feel the intensity of pain but that the pain itself no longer bothers them (Foltz & White, 1968).<sup>2</sup>

Pain researchers have subsequently subdivided painful experience into two components: the intensity and the unpleasantness of painful experience (Price, 2000; Rainville et al., 1997). Rating the *intensity* of pain can be likened to rating the loudness of the volume on a radio; whereas the *unpleasantness* of pain can be likened to rating the extent to which the volume on the radio is perceived as bothersome. While the dACC has been shown to be involved in the perceived unpleasantness of physical pain, other neural regions such as the somatosensory cortex and posterior insula have been shown to be involved in processing the sensory-discriminative aspects of pain (Peyron, Laurent, & Garcia-Larrea, 2000). Pain disorders involving damage to somatosensory areas leave patients unable to identify where the pain is coming from or how intense it is but still able to experience the distress associated with having the pain in the first place (Nagasako, Oaklander, & Dworkin, 2003).

The first neuroimaging study linking pain distress to dACC activity used hypnotic suggestion to alter the perceived unpleasantness of painful stimulation without changing the perceived intensity (Rainville et al., 1997). Using positron emission tomography (PET), it was observed that dACC activity corresponded to changes in the perceived unpleasantness of painful stimuli whereas the activity of primary somatosensory cortex, typically associated with the perceived intensity of painful stimulation, remained unaltered. Since then, several neuroimaging studies have linked the increasing unpleasantness of painful stimulation with dACC activation (Peyron et al., 2000). In a similar manner, pain-sensitive individuals, who report more pain unpleasantness to less intense pain, show significantly more dACC activity to painful stimulation than do less pain-sensitive individuals (Coghill, McHaffie, & Yen, 2003).

### *Social Pain and the dACC*

**Social Pain in Animals.** The cingulate gyrus has no distinctive counterpart in the reptilian brain, appearing for the first time, phylogenetically, in mammalian species (MacLean, 1985a, 1993).<sup>3</sup> Several behavioral characteristics accompany the evolution of mammals as well. These newly emerged characteristics, differentiating mammals from their reptilian ancestors, include audiovocal communication for maintaining maternal-offspring contact and the nursing of young along with maternal care.<sup>4</sup> As the cingulate appeared on the evolutionary scene

at the same time as these characteristics, it may be a contributor to these uniquely mammalian behaviors.

One of these uniquely mammalian behaviors is the production of distress vocalizations, which are considered to be the most primitive and basic mammalian vocalization with the original purpose of maintaining mother-infant contact (MacLean, 1985a). Typically, infants emit distress vocalizations when separated from their caregivers and the sound of these vocalizations elicits distress in the mother, motivating her to retrieve her young. Consistent with the idea that distress vocalizations evolved in the context of parent-child relations, reptilian newborns, which receive no parenting as they are born almost completely mature, do not produce distress vocalizations (MacLean, 1985b).

To establish that the cingulate gyrus plays a causal role in the distress of social distance and the production of distress vocalizations, lesions to the cingulate gyrus should lead to: (a) fewer distress vocalizations when socially separated, and (b) fewer attempts at maintaining social closeness if social distance is no longer distressing. Consistent with this causal role, ablation of the dACC in squirrel monkeys leads to decreased distress vocalizations (Kirzinger & Jurgens, 1982; MacLean & Newman, 1988). Additionally, cingulate lesions in macaques lead to decreases in affiliative behavior, as indicated by a reduction in the amount of time spent in social interactions or in proximity with other macaques (Hadland et al., 2003). This drop-off in social affiliation may be the result of a reduced need for social closeness because social distance is no longer experienced as aversive.

Additionally, if the dACC is one of the primary neural regions involved in the production of distress vocalizations, localized stimulation of the dACC should elicit distress vocalizations while stimulation of other language areas should not. To this end, electrical stimulation of the dACC leads to the spontaneous production of distress vocalizations in rhesus monkeys (Robinson, 1967; Smith, 1945); whereas, stimulation of the area corresponding to Broca's area in monkeys and in apes, an area known to be involved in speech production, elicits movement of the vocal chords but no distress vocalizations (Leyton & Sherrington, 1917; Ploog, 1981).

The cingulate gyrus also plays a role in caregiver responses to infant distress vocalizations. Ablation of the cingulate gyrus in adult female rats results in deficits in maternal behavior, including the nursing and retrieval of pups (Stamm, 1955). Following cingulate ablation in females, rat mothers become less responsive to the distress vocalizations of their pups. In one study, the survival rate of rat pups with cingulate-lesioned mothers was only 12%, compared to a 95% survival rate in rat pups with sham-lesioned mothers (Stamm, 1955).

**Social Pain in Humans.** Much less is known about the neural correlates of social pain in humans. However, if the dACC is involved in social pain in other mammals, it is reasonable to suggest that it is involved in human social pain as well. If this is true, lesioning the dACC in humans should have social consequences,

making individuals less sensitive to social pain and potentially less interested in social affiliation as social closeness is no longer relieving. An early study noted that lesioning the dACC for chronic pain or anxiety disorders had social consequences. Following cingulotomy, patients became less socially inhibited, less shy, and less socially sensitive (Tow & Whitty, 1953). In other words, these patients became less socially concerned and more socially uninhibited.

Another frequent consequence of cingulotomy is akinetic mutism, in which patients temporarily do not initiate vocalization based on a lack of desire rather than a lack of ability (Laplane et al., 1977). While highly speculative, the disinclination to initiate vocalization may reflect a reduction in the concern for social connection. Destroying the portion of the cingulate associated with detecting social separation and vocalizing to reestablish connection, may result in the temporary absence of self-initiated vocalizations.

Perhaps the most direct evidence for the role of the dACC in human social pain comes from a neuroimaging study investigating the neural correlates of one type of social pain: social exclusion (Eisenberger, Lieberman, & Williams, 2003). In this study, participants were led to believe that they would be playing a virtual ball-tossing game with two other players over the Internet while in the fMRI scanner. In reality, there were no other players; rather, the computer images of the other players were preprogrammed to include the participant during one round of the ball-tossing game and to exclude the participant in another round of the game by not throwing the ball to the participant. Upon being excluded from the game, compared to when being included, participants showed increased activity in dACC, the region most often associated with the affective distress of physical pain. In addition, the amount of activity in this area correlated strongly ( $r = .88$ ) with the amount of self-reported social distress participants felt during the exclusion episode. Thus, mirroring the animal research, the dACC seems to play a role in the distress associated with perceived social distance in humans as well.

## HYPOTHESIS #2: PHYSICAL AND SOCIAL PAIN RELY ON THE SAME COMPUTATIONAL MECHANISMS

Because the dACC is involved in the experience of both physical and social pain, it is plausible that the underlying computational processes of the dACC are relevant in the processing of both types of pain. Understanding the computations underlying this shared neural circuitry is important for building a more complete model of the physical-social pain overlap.

Cohen and colleagues have shown that the dACC acts as a conflict or discrepancy monitor, detecting when an automatic habitual response is contextually inappropriate or conflicts with current goals (Botvinick et al., 2001). One simple example of conflict, often used to elicit dACC activation, is the Stroop task in which the automatic word-reading response conflicts with the goal of color nam-



ing (e.g., name the ink color of the word R-E-D printed in blue ink; MacDonald et al., 2000).

A number of other studies suggest that the dACC may be sensitive to goal conflicts and unexpected events more generally (Weissman et al., 2003), detecting discrepancies between automatic responses and current goals, between actual and expected events, and between new stimuli and pre-existing representations that do not map onto each other. When the dACC detects these discrepancies, the prefrontal cortex is notified of the problem so that it can exert executive control (Miller & Cohen, 2001) by overriding automatic processes.

Though much research supports this account of the dACC as a discrepancy detector, it is not clear how this function relates to physical or social pain processes. On the one hand, the dACC has been characterized as a discrepancy detector, producing activity to simple response conflicts such as those evidenced in the Stroop task (Botvinick et al., 2001). On the other hand, the dACC has been characterized as a distress center, producing activity to instances of both physical and social pain (Eisenberger et al., 2003; Rainville et al., 1997). How can these two characterizations of dACC function be reconciled?

If one conceptualizes the dACC as a neural alarm system (Eisenberger & Lieberman, 2004), the connection between discrepancy detection and the experience of physical and social pain quickly makes more sense. Most real world alarm systems (e.g., fire alarms) have two components. The first component is the sound of the alarm bell, the part of the alarm that signals that there is a problem, interrupts ongoing activity, and directs attention at solving the problem. This part of the alarm may be analogous to the experience of physical or social pain, which is also distressing, attention-getting, and disruptive. The second component of the alarm is the machinery that detects when something has gone wrong or has strayed from a desired set point (e.g., too much smoke in the room). In essence, this part of the alarm system detects discrepancies from some standard, initiates the sounding of the bell, and may be analogous to the discrepancy detection function of the dACC. Rather than discrepancy detection and distress being two competing accounts of dACC functioning, the analogy to an alarm system suggests that they may actually be two sides of the same coin: the two complementary processes of a neural alarm system. Based on this account, this multi-purpose alarm should be triggered once the underlying machinery has detected either physical damage, a discrepancy from the healthy state of the body, or social distance, a discrepancy from the desired state of social connection.

Though it seems reasonable that the dACC might act as a neural alarm system, detecting discrepancy and producing the subsequent feelings of distress, these two properties of dACC activity have yet to be linked. Typically, studies of the dACC as a discrepancy detector do not assess phenomenological distress and studies of the dACC's involvement in distress do not assess discrepancy detection. In order to examine whether these two properties of dACC function are two sides of the same coin, we examined two hypotheses: (1) whether individuals who tend to be

distressed more often are more sensitive to discrepancy as evidenced by increased dACC activation during a discrepancy detection task, and (2) whether activating discrepancy detection processes heightens an individual's sensitivity to distress.

To test the first hypothesis, we examined whether individuals high in neuroticism, those who tend to experience distress more often, are more sensitive to discrepancy detection, as evidenced by more dACC activity to a simple discrepancy detection task (Eisenberger, Lieberman, & Satpute, in press). Because neuroticism is often defined as the tendency to experience negative affect (Costa & McCrae, 1980; Eysenck, 1967), neurotics should show a greater sensitivity to discrepancy, if discrepancy detection and distress go hand-in-hand.

In this study (Eisenberger et al., in press), participants were scanned while performing an "oddball" task, a simple discrepancy detection task in which a sequence of letters is presented, one at a time, on a computer screen. In this task, 80% of the letters are the letter "X", but participants are instructed to press a button only when they see a letter other than X. Because the base-rate expectation of seeing an X is 80%, seeing other letters violates this expectation and leads to activation of the dACC (Braver et al., 2001; Weissman et al., 2003).

It was found that heightened dACC reactivity to the oddball trials, relative to non-oddball trials, was significantly correlated with higher levels of self-reported neuroticism ( $r = .76$ ). In other words, individuals higher in neuroticism showed more dACC reactivity to this simple discrepancy detection task, implying that heightened levels of distress and a more sensitive alarm system go hand-in-hand.

The second study investigated whether increasing the activity of the alarm system's discrepancy detector would simultaneously make distress-related cognitions more accessible, particularly in neurotics who may have a more sensitive alarm system to begin with. In this study (Eisenberger & Gable, 2004), participants were exposed to either a normal Stroop task involving discrepancy detection or a modified Stroop task containing no discrepancy (neutral task), in which individuals were simply asked to name the color of different shapes. Following the manipulation of discrepancy detection processes, participants completed a lexical decision task in which reaction times to different categories of words were assessed. Faster reaction times to a certain class of words were presumed to be indicative of cognitions that were more readily accessible. Participants were exposed to five categories of words including: (1) social rejection words ("abandonment," "rejection"); (2) negative trait words ("lazy," "dullness") to control for the negativity of the social rejection words without the social relationship component; (3) social comfort words ("love," "support"); (4) positive trait words ("charming," "clever") to control for the positivity of the social comfort words without the social relationship component; and (5) non-words ("tlinking," "works").

In general, participants did not become more sensitive to social rejection words following discrepancy. However, individuals higher in neuroticism did. It was found that after controlling for reaction times to negative trait words, individuals higher in neuroticism were significantly faster to social rejection words following

the discrepancy detection task compared to the neutral task. There were no between-group differences in reaction times to social comfort words after controlling for positive trait words, and there were no neuroticism by condition interactions. Thus, inducing minimal discrepancy detection processes made neurotics more sensitive to social rejection, suggesting that activating this system can make certain individuals more sensitive to distressing cues such as those indicating social pain. This study, along with the one described before it, provides evidence for the notion that discrepancy detection and distress are two complementary processes that underlie the functioning of the dACC.

### HYPOTHESIS #3: INDUCING OR REGULATING ONE TYPE OF PAIN SIMILARLY INFLUENCES THE OTHER

In this section, we will provide evidence showing that enhancing one type of pain or its predictors should heighten an individual's sensitivity to the other type of pain (pain potentiation effects). Alternatively, reducing one type of pain or its predictors should diminish an individual's sensitivity to the other type of pain (pain regulation effects).

#### *Pain Potentiation Effects*

Whereas it seems quite intuitive that physical harm produces physical pain and that social harm produces social pain, the notion that experiencing or enhancing sensitivity to one type of pain might potentiate one's sensitivity to the other type of pain is far from obvious. However, there is at least some evidence supporting this hypothesis. Correlational accounts suggest that the experience of one kind of pain directly correlates with a heightened sensitivity to the other. For example, Bowlby noted that when children feel physical pain, they become much more sensitive to the whereabouts of their caregiver and experience distress more frequently and easily upon noting distance from the caregiver (Bowlby, 1969). Similarly, compared to 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 et al., 2003).

To date, no studies have experimentally manipulated physical pain to investigate the consequences for social pain or have manipulated social pain to investigate the consequences for physical pain. However, several studies have investigated the effects of failure on the experience of physical pain. If the perceived consequence of failing is that one would not be accepted or liked by others, failure could trigger feelings of social pain. This might occur for individuals who have been told that they have failed at something that they consider important for their social identity or for their acceptance or inclusion in a certain group.

In line with this, college-age participants who were informed that they performed far below average on a college entrance exam, reported heightened pain ratings to a cold-pressor task (van den Hout et al., 2000). For the college undergraduates who participated in this study, intelligence is likely to be a characteristic that is valued by themselves and their families. Failing on an academic test could signify that their family or other important social relationship members would disapprove or reject them, thus eliciting social pain and a corresponding sensitivity to physical pain. In a similar study, college-age participants who were given computerized feedback indicating that they had performed poorly on a reading comprehension task, also reported higher pain ratings to a cold-pressor task (Levine, Krass, & Padawer, 1993).

### *Pain Regulation Effects*

Diminishing one type of pain or diminishing an individual's sensitivity to one type of pain has been shown to reduce an individual's sensitivity to the other type of pain as well. A great deal of correlational research has shown that individuals with more social support experience less cancer pain (Zaza & Baine, 2002), take less pain medication, are less likely to suffer from chest pain following coronary artery bypass surgery (King et al., 1993; Kulik & Mahler, 1989), report less labor pain, and are less likely to use epidural anesthesia during childbirth (Kennell et al., 1991). Thus the perception or presence of social support, presumably indicative of a lesser likelihood of social harm, appears to regulate physical pain in several different health domains.

Experimental evidence has demonstrated similar effects. Animal research has shown that the presence of another animal lessens the distressing experience of painful stimulation (Epley, 1974). For example, electric shock punishment was less effective in training rats that were tested in groups than rats that were tested alone (Rasmussen, 1939), suggesting that the shocks were less aversive and thus a less effective training device when the rats were in a social group. In addition, a rat's immobility due to electric shocks was reduced by the presence of a companion rat (Davitz & Mason, 1955). Finally, baby goats displayed fewer emotional reactions to electric shock when their mother was present than when she was absent (Liddell, 1954).

Human research has shown similar effects as well (Epley, 1974). The presence of companions has been shown to reduce the amount of self-reported fear associated with electric shocks (Amoroso & Walters, 1969; Buck & Parke, 1972) and to increase participant's tolerance of intense electric shock, suggesting that painful stimulation is experienced as less painful when in the presence of a companion (Seidman et al., 1957). More recently it has been shown that participants in the presence of either a friend or a supportive stranger reported less pain to a cold-pressor task than when alone (Brown et al., 2003). In short, experimentally manipulating the presence of supportive others can reduce pain sensitivity.

More evidence for pain regulation effects comes from drug studies. Opiate-based drugs, known to reduce physical pain, have also been shown to reduce separation distress vocalizations, elicited by infant mammals when separated from their caregivers or the social group (Nelson & Panksepp, 1998; Panksepp, 1998). In fact, one of the surest ways to increase a rat's consumption of opiates (especially for female rats) is through social isolation (Alexander, Coombs, & Hadaway, 1978), as the increased consumption of opiates seems to regulate the animal's experience of isolation distress.

Antidepressant medications or selective serotonin reuptake inhibitors (SSRIs) also have similar effects on both physical and social pain. Antidepressants, typically prescribed for treating anxiety and depression, often related to or resulting from social pain, are effective in alleviating physical pain as well (Nemoto et al., 2003; Singh, Jain, & Kulkarni, 2001). In fact, antidepressants are now regularly prescribed to treat chronic pain conditions.

#### HYPOTHESIS #4: TRAIT DIFFERENCES RELATING TO ONE TYPE OF PAIN RELATE TO THE OTHER TYPE AS WELL.

The last hypothesis that will be considered in this chapter is that trait differences related to the sensitivity to one type of pain should also relate to the sensitivity to the other type of pain. Because neuroticism has been shown to link to both aspects of the alarm, a heightened sensitivity to discrepancy (Eisenberger et al., 2004) and heightened distress (Costa & McCrae, 1980; Eisenberger & Gable, 2004), neuroticism may well represent a trait amplification of this alarm system. As such, neuroticism should be associated with a greater sensitivity to and a more distressing experience of both physical and social pain.

Some observational evidence already exists that suggests this might be the case. Beck noted that the two of the most frequent types of anxious thoughts that neurotic individuals had, revolved around the possibility of physical harm (being attacked, being in a car accident) and the possibility of social harm (being rejected, ostracized; Beck, Laude, & Bohnert, 1974). In addition, Twenge (2000) has shown that increases in the levels of neuroticism and anxiety in the United States, over the past 40 years, directly correspond with increases in indicators of social distance (divorce rates) and increases in the prevalence of physical dangers (crime rates). In the following section, we will review evidence suggesting that neuroticism is associated with a heightened sensitivity to both physical and social pain.

#### *Neuroticism and Physical Pain Sensitivity*

Neuroticism is frequently associated with the tendency to be hypersensitive to physical symptoms, such as pain or discomfort, and to be distressed by these symptoms more often (Watson & Pennebaker, 1989). Epidemiological studies

report that 50% of patients seeking treatment for medically unexplained physical symptoms, often visceral or somatic pain, are either anxious or depressed (Katon, Sullivan, & Walker, 2001). Neuroticism, along with similar constructs such as trait negative affect and trait anxiety, has also been shown to be associated with lower pain thresholds (Bisgaard et al., 2001; Pauli, Wiedemann, Nickola, 1999; Phillips & Gatchel, 2000; Shiomi, 1978; Wade & Price, 2000) and higher pain unpleasantness ratings (Wade et al., 1992). In addition, neuroticism predicts greater levels of postoperative pain following cholecystectomy (Bisgaard et al., 2001), is associated with higher pain severity ratings to chest pain symptoms (Costa et al., 1985), and is associated with higher levels of psychological distress due to pain in individuals with low back pain (BenDebba, Torgerson, & Long, 1997).

### *Neuroticism and Social Pain Sensitivity*

Perhaps less intuitive than the neuroticism-physical pain link, is the hypothesis that neuroticism is associated with a heightened sensitivity to social pain as well. Though some have made the claim that the experience of anxiety is fundamentally a fear of social rejection or exclusion (Baumeister, 1991; Baumeister & Tice, 1990), most do not intrinsically equate neuroticism or trait anxiety with a specific sensitivity to social rejection. However, studies suggest that neuroticism is at least partly associated with a heightened sensitivity to the possibility or actuality of social pain.

Several studies have shown that neuroticism correlates well with measures assessing sensitivities to social pain, such as measures of rejection sensitivity or interpersonal sensitivity. Rejection sensitivity is defined as the tendency to expect rejection and is assessed by questions such as "How concerned or anxious would you be over whether or not this person would want to go out with you?" (Downey & Feldman, 1996; see also Romero-Canyas & Downey, this volume). Interpersonal sensitivity is defined as the tendency to react with excessive sensitivity to the interpersonal behavior of others or the perceived or actual negative evaluation by others and is assessed by statements such as "I worry about what others think of me" (Boyce & Parker, 1989). Recent studies have demonstrated that self-reported neuroticism is correlated positively with rejection sensitivity ( $r = .36$ ; Downey & Feldman, 1996) and with interpersonal sensitivity ( $r = .48$  to  $r = .61$ ; Boyce & Parker, 1989; Gillespie, et al., 2001; Luty et al., 2002; Smith & Zautra, 2002). Self-reported neuroticism also correlates highly with self-reported generalized social anxiety ( $r = .58$ ; Norton et al., 1997). Similarly, substance abusers who are high in neuroticism are more likely to relapse specifically from episodes of social rejection ( $r = .47$ ; McCormick et al., 1998). Lastly, not only are neurotics more sensitive to the possibility of social rejection but they can also experience greater and longer-lasting episodes of grief following the actual loss of close others (Bailey, 2001; Ogrodniczuk et al., 2003).

## CONCLUSION

There is something in staying close to men and women, and looking on them, and in the contact and odor of them, that pleases the soul well . . .

Walt Whitman, "I Sing the Body Electric," 1855

We began this chapter with a quote from Aristotle, who suggested that no individual would want to live without social connections. We now end this chapter with a quote by Walt Whitman, written nearly 2,000 years later, indicating a similar idea—that part of what makes life worth living is being close to others. Indeed, if asked to pinpoint the best and worst experiences of life, most of us would pick those experiences involving the making and breaking of social bonds. For most, no occasion could be happier than a marriage or the birth of a child, and none could be more painful than the loss of the ones we love. Increasingly, evidence is pointing to the importance of social connections not only for our happiness and well-being but for our survival as well. Through the studies reviewed here, we are beginning to appreciate that the need for social connection is so essential to survival, at least in mammalian species, that being left out or disconnected from the social group is processed by the brain in a manner similar to physical pain. Just as physical pain has evolved to alert us that something has gone wrong with our bodies, social pain is a similarly potent signal that alerts us when something has gone wrong with our social connections to others, an equally important threat to the survival of our species.

In this chapter we have reviewed pain overlap theory, which advances the notion that physical and social pain rely on parts of the same underlying system for their operation. We have also provided evidence for several hypotheses that can be derived from this theory. We have shown that the dACC acts as one of the neural substrates of the physical-social pain overlap and that it is involved in both the detection of physical and social danger and in the alarming experience that follows. We have shown that potentiating or regulating one of these forms of pain influences the other form of pain in a congruent manner. Lastly, we have provided some evidence to suggest that neuroticism is associated with a heightened sensitivity to indicators of both types of pain. These are not the only implications that can be derived from this theory. Other hypotheses that remain to be explored include whether physical and social pain have similar behavioral consequences, result in similar health outcomes, or share other common neural structures or neurotransmitters not reviewed here. Continuing to explore the underlying commonalities between physical and social pain may provide us with new ways of treating physical pain and new techniques for managing social pain. Perhaps most importantly, understanding this overlap may provide us with answers to two of our most fundamental questions: why it hurts to lose those we love and why being close to others "pleases the soul well."

## NOTES

1. Although we will focus more specifically on the common neural structures underlying physical and social pain, we recognize that the shared opiate substrates are an important part of the physical-social pain overlap and will refer to them in a general manner throughout the chapter.
2. Although we can use the behavioral consequences of cingulotomy to inform our working knowledge of the phenomenology of ACC activation, it should be kept in mind that cingulotomies are only performed in the most extreme and severe cases of pain or anxiety. Thus, one should use caution when extrapolating from cingulotomy patients to the general population as the functioning of this neural region may be different for healthy individuals.
3. In non-primates, the cingulate gyrus is the primary unit of analysis; whereas in primates and humans, anterior and posterior sectors of the gyrus are treated separately.
4. The evolution of play is also a uniquely mammalian behavior; however, a complete discussion of the evolution of play behavior will not be discussed here (for a full review and discussion of play, see MacLean, 1985a or Panksepp, 1998, Chapter 15).

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