

Social Pain

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Abstract and Keywords

The critical importance of social connectedness to human health and well-being is well established. There is now accumulating evidence that when a threat to this connectedness (i.e., social injury) occurs, a pain-like experience—social pain—can result. The first part of this chapter presents an up-to-date summary of animal and human brain imaging studies demonstrating an overlap in biological and neural systems mediating both social and physical pain. The second part reviews the literature examining the potential implications of this overlap, including the effect of reducing physical pain on social pain, the impact of social support on physical pain sensitivity, and the effects of socially painful events on physical pain perception. The chapter concludes with an exploration of what we believe are pressing issues and questions to be addressed in future research in the expanding field of social pain.

Keywords: exclusion, ostracism, pain, rejection, social

Introduction

Being socially excluded can feel awful. Being spurned by a love interest, turned down by a desired group, or grieving the loss of a loved one are all aversive experiences and can engender feelings of sadness, frustration, shame, and anger. But there is more to it. There is an element that distinguishes these types of experiences from other types of negative experiences: Social exclusion hurts. English speakers often use terms like “broken hearted,” “hurt,” and “wounded” to describe social disconnection. Indeed, across multiple languages, as diverse as Spanish, Hebrew, Mandarin, and Inuktitut, native speakers use terms that mean “hurt” or “injury” to describe the emotional reaction to social rejection (MacDonald and Leary, 2005).

If physical injury produces physical pain, then the result of social injury can be described as social pain (MacDonald, 2009). MacDonald and Leary (2005) define social pain as “a specific emotional reaction to the perception that one is being excluded from desired re-

Social Pain

relationships or being devalued by desired relationship partners or groups. Exclusion may be a result of a number of factors, including rejection, death of a loved one, or forced separation.” (p. 202). In other words, social pain is an emotionally aversive pain-like experience that occurs in reaction to a socially disconnecting event. But does the use of the word pain merely represent a convenient metaphor or are physical and social pain related more fundamentally?

The benefits of social connection and the repercussions of disconnection to human development, health, and survival are sweeping and profound. Beginning in infancy, all mammals depend on a bond with caregivers to survive (Bowlby, 1969) and many behavioral and regulatory functions critical to survival throughout life are forged in early years through interactions with caregivers and others in the child’s social milieu (Bowlby, 1969; 1973; 1980; Schore, 1994, 2003). This social impact is by no means limited to the early years and continues throughout the lifespan. Numerous epidemiological studies have shown that socially isolated people (p. 164) are 2 to 3 times more likely to die in a given time period (Seeman, 1996; House, Landis, Umberson, 1988). People who perceive high levels of social support enjoy numerous health benefits (Uchino, 2006; Reblin & Uchino, 2008), including antibody response to flu immunization (Pressman et al., 2005), survival following myocardial infarction (Rodriguez-Artalajo et al., 2006; Schmaltz et al., 2007), and postoperative pain and recovery (Mitchinson et al., 2008). A recent review of data from the massive Framingham Heart Study showed that if a close friend becomes obese (or smokes), it increases the chance of becoming obese (or smoking) oneself by some 50%. Consider that the World Health Organization definition of health has encompassed the social sphere since 1948, stating that health is “a state of complete physical, mental and *social well-being* and not merely the absence of disease or infirmity” (*italics added*). It is perhaps because of the extensive influence of relationships on health and survival that the human need for belonging and acceptance is so strong (Baumeister & Leary, 1995). Humans evolved to depend not just on the operation of systems within the body but also on the interplay between the individual as a whole and the social systems surrounding the individual.

Given the immense and widespread impact of social factors on human health, social disconnection would pose a formidable menace. Therefore, some neurophysiological machinery may have evolved to efficiently recognize and react to threats of exclusion, as well as motivate measures aimed at preventing and repairing social injury. Because physical pain serves exactly this function in the case of physical injury, it seems that pain would make an ideal candidate for this purpose (Eisenberger & Lieberman, 2004; MacDonald & Leary, 2005; Panksepp, 1998, 2010).

This chapter investigates the notion of social pain by examining the evidence for links between the pain resulting from physical injury and the pain-like agony that can follow social exclusion. To understand why and how pain might serve as a regulator of social inclusion, it would be helpful to look more closely at the nature of pain. What is pain and what is its function?

What is Pain?

Pain is immensely powerful. It can cripple, paralyze, and instill raw fear. But it is also remarkably useful. Pain signals the presence or threat of injury and is a mechanism of self-preservation. Its considerable ability to capture attention makes it difficult to ignore, and its aversiveness strongly motivates reactions aimed at its reduction. So feared is pain that people may go to great lengths to avoid feelings of pain, even if it means also sacrificing considerable benefits, such as when individuals avoid going to the dentist although they know it is necessary (Berggren & Meynert, 1984). Although pain is unpleasant, it is difficult to imagine being able to survive without it. Individuals born without the ability to feel pain, or who feel pain sensation but experience it as nonaversive, suffer continual injuries, amputations of limbs, loss of vision, and a shortened lifespan (Nagasako, Oaklander, & Dworkin, 2003).

Pain is notoriously subjective and private and has so far proven very difficult to index with objective measures such as blood, urine, or even brain imaging tests. Even novel methods such as those that detect facial muscle activity (Craig, 1992) have been of limited use. Indeed, pain's inherently subjective and private nature can exacerbate feelings of isolation for those whose pain cannot be explained medically, potentially leading to social pain. Far from being a linear function of tissue damage, pain is a highly complex phenomenon, consisting of multiple facets, involving numerous neural circuits, as well as the endocrine and immune systems (Benarroch, 2001; Chapman, Tuckett, & Song, 2008). Pain is impacted by a plethora of contextual (e.g., beliefs, expectation), cognitive (e.g., attention), mood, chemical, memory, and genetic factors (Tracey & Mantyh, 2007), and prompts a multitude of protective (nocifensive), affective, cognitive, and behavioral responses.

A very widely cited current definition of pain states that “pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (Merskey & Bogduk, 1994, p. 210). The reference to “sensory and emotional experience” suggests that pain is regarded as having two components: sensory and affective. The sensory aspect refers to location and qualities of the sensation (e.g., throbbing or stabbing intense pain in the right big toe). The affective component refers to the emotional aspect of the experience—how unpleasant, irritating, or distressing the pain experience is. The distinctiveness of these two dimensions has been demonstrated in numerous ways. For example, each dimension can be impacted independently as a result of changes in nociceptive stimulus intensity and psychological factors (Price, Harkins, & Baker, 1987; Price, 1999). Second, hypnotic suggestions can separately target each dimension (p. 165) (Rainville, Carrier, Hofbauer, Bushnell & Duncan, 1999). Finally, pain sensation and pain unpleasantness are reliably mediated by distinct neural systems (Rainville, Duncan, Price, Carrier, & Bushnell, 1997; Tölle et al., 1999).

Although the physical sensations associated with physical pain are often quite different from those associated with social pain, the affective component may be more similar. As a result, it is the emotional aspect of a socially distressing experience to which the term social pain has been applied. MacDonald and Leary (2005) have suggested that social pain

Social Pain

is actually one form of emotional pain and that the affective responses to physical trauma are themselves a subcategory of emotional pain (MacDonald & Leary, 2005). Emotional pain can arise when some harm or loss has occurred to the individual. Just as physical pain arises from physical injury, social pain may arise from social injury. Social pain, then, has been conceptualized primarily as the emotional distress that arises in the event of social harm or loss. Although it is possible that social pain may be found to vary along sensory dimensions such as pain quality (throbbing, stabbing) and location (stomach, chest), the sensory component of social pain is currently poorly understood, and at the phenomenological level, seemingly differs from the sensory aspect of physical pain. Further research is necessary to identify the contribution of sensory elements to the experience of social pain.

Among the first lines of evidence supporting the notion of social pain there is research showing that opioids, a key player in physical pain modulation systems, also play a pivotal role in modulating social distress. It is to this research that we turn next.

Overlapping Biochemical Systems—Opioids

The opioid system is intimately and extensively involved in regulating physical pain. For example, opiate drugs such as morphine, which mimic the action of endogenous opioids, are currently considered to be among the most powerful and effective analgesic drugs available for the treatment of moderate to severe pain (Bie & Pan, 2007; Fine & Portenoy, 2004).

Not long after the discovery of the opioid system in the brain, a series of studies by Jaak Panksepp and colleagues showed that the administration of morphine calmed distress cries emitted by socially separated animals (Herman & Panksepp, 1978; Panksepp, Herman, Conner, Bishop, & Scott, 1978; Panksepp, Vilberg, Bean, Coy, & Kastin, 1978; Panksepp, Herman, Vilberg, Bishop, & DeEskinazi, 1980; but see Winslow and Insel, 1991). An opiate drug, long known as a physical pain analgesic, also dulled social pain. In one study, for example, Herman and Panksepp (1978) segregated infant guinea pigs from their mothers and placed the infants in a pen by themselves for 15 minutes each day over a period of 2 to 3 weeks. Such an involuntary separation from the mother typically provokes distress vocalizations (DVs) in young animals. When injected with the mu-opioid agonist morphine sulfate, however, the isolated guinea pigs emitted significantly fewer DVs compared with animals given a saline injection, while having no effect on overall activity. The effect was dose dependent; higher doses of opioids led to fewer DVs. These results were subsequently replicated in several studies (Herman & Panksepp, 1978, 1981; Keverne et al., 1997; Panksepp, 1998; Panksepp, Herman, Conner, Bishop, & Scott, 1978) and with different animals, including rats (Carden, Hernandez, & Hofer, 1996; Carden & Hofer, 1990b; Kehoe & Blass, 1986; Kehoe & Boylan, 1994), primates (Kalin, Shelton, & Barksdale, 1988; Keverne et al., 1989), dogs (Panksepp, Herman, Conner, Bishop, & Scott, 1978), and birds (Panksepp, Vilberg, Bean, Coy, & Kastin, 1978). Over the course of the evolutionary history of an organism, features that evolved to serve one function may

Social Pain

later be co-opted to serve another, a process known as *exaptation*. Panksepp conjectured that systems mediating both physical and social pain overlapped and since socially complex mammals appeared relatively recently in evolutionary history, the social distress system may have exapted the more evolutionarily ancient physical pain system to signal social disconnection (Panksepp, 1998).

A different line of research has found that opioids are centrally involved in the diminished pain sensitivity (hypoalgesia) observed concomitant with social isolation (Kehoe & Blass, 1986b; Naranjo & Fuentes, 1985; Puglisi-Allegra & Oliverio, 1983). For example, in one study (Puglisi-Allegra & Oliverio, 1983), mice were either separated or housed in groups of six for a period of 8 weeks. In addition, some were subjected to immobilization stress or no stress and some received morphine or saline. The socially isolated mice demonstrated a higher nociceptive threshold (lower pain sensitivity) in response to painful heat stimuli than the grouped mice. This effect was diminished with injection of naloxone (a mu-opioid antagonist), implying that the isolation-induced hypoalgesia was opioid-mediated. Most importantly, administration (p. 166) of morphine had no effect on isolated mice though it did decrease pain sensitivity in the grouped mice. Taken together, social isolation modulates physical pain sensitivity via opioid systems and the chronic activation of the opioid system (induced by long-term social isolation) may reduce opioid receptor binding (Schenk, Britt, Atalay, & Charleson, 1982). If applicable to humans, these results have important clinical implications and suggest yet another way by which social and physical pain are linked: chronic social pain may induce changes in opioid receptor binding that may compromise both an individual's endogenous physical pain modulation system operation as well as responsiveness to opiate drugs such as morphine.

In sum, the finding that opiates can calm social distress in animals, together with the finding that opioids mediate social threat-induced hypoalgesia and that chronic exposure to social threats impairs responsiveness to opiate drugs administered to relieve physical pain, provide persuasive evidence that opioids play a key role in linking both types of pain experience. A biochemical overlap implies that a neural overlap is likely but more direct evidence is required. The next two sections explore this evidence.

Overlapping Neural Systems

Our understanding of how neural systems participate in the pain experience has expanded dramatically over the past decade with the application of technologies such as positron emission tomography and functional MRI that enable the relatively noninvasive observation of responses in the intact human brain (Perl, 2007). Studies employing these imaging systems have led to the discovery of a number of anatomically distinct brain regions that are reliably related to the processing of acute physical pain (Apkarian, Bushnell, Treede, & Zubieta, 2005; Peyron, Laurent, & Garcia-Larrea, 2000; Tracey & Mantyh, 2007). These areas include bilateral secondary somatosensory cortex (SII), anterior insular cortex (AIC), anterior cingulate cortex (ACC), contralateral thalamus, the primary somatosensory cortex (S1), and the dorsolateral prefrontal cortex. These findings are corroborated by

Social Pain

results from studies employing pharmacologically-induced hypoalgesia, which have shown effects predominantly in these areas (Casey et al., 2000; Geha et al., 2007; Wagner et al., 2007; Wise et al., 2002, 2004). The numerous regions showing reliable links to acute physical pain episodes are often referred to collectively as the *pain matrix*, and reflect the rich and multidimensional nature of the pain experience.

Evidence in support of a social/physical pain neural overlap would therefore need to show that socially injurious events lead to neural responses that are similar to those occurring following physical injury. The first study examining neural responses to exclusion experiences in humans was work by Eisenberger, Lieberman, and Williams (2003). In their study, participants were asked to undergo fMRI brain scanning while playing a video ball-toss game (Cyberball) with two other players. Relative to periods of inclusion, exclusion from play midway through the game was associated with an increase in dorsal anterior cingulate cortex (dACC), insular cortex (IC), and right ventrolateral prefrontal cortex (RVPFC) activity. Higher levels of self-reported distress were associated with greater activity in the dACC and less activity in the RVPFC, but they were not related to IC activity. Meditational analyses showed that the RVPFC acts to diminish feelings of social distress through inhibition of dACC activity. A comparison condition in which participants were excluded due to a technical glitch (implicit exclusion) was associated with greater dACC activity but not with any change in RVPFC activity, perhaps because feelings of exclusion were not of sufficient magnitude to prompt self-regulatory responses. The researchers suggest that their results indicate an overlap in the neural circuitry serving both physical and social pain.

Eisenberger, Gable, and Lieberman (2007) asked participants to record how connected and accepted they felt during social interactions throughout the day over the course of 10 days. After this experience sampling period, participants engaged in a Cyberball task while being scanned with fMRI. The neural response to an exclusion experience in the scanner (exclusion from Cyberball play) was an excellent indicator of the tendency to experience social distress in daily living. Specifically, individuals who showed greater activity in the left dACC during the Cyberball game reported significantly higher levels of distress during daily social interactions (as well as greater distress in response to the Cyberball game). Greater activity in the amygdala and left periaqueductal gray also predicted daily social distress but not distress in response to the Cyberball game.

Burklund, Eisenberger, and Lieberman (2007) used fMRI to compare neural responses to brief video clips of disapproving, angry, and disgusted facial expressions in young, healthy adults. There was increased activity in the amygdala in response to exposure to all three facial expressions (compared with fixation), but disapproval faces provoked (p. 167) greater amygdala activity than anger, implying that disapproving faces can be as threatening as anger, if not more so. Contrary to their predictions, however, disapproval faces in general provoked no greater (or lesser) levels of dACC activity. The authors did find that people with higher scores on a measure of rejection sensitivity responded to disapproving (but not other) faces with greater dACC activation (but not other limbic areas),

Social Pain

as well as reduced activation in the ventromedial prefrontal cortex (VMPFC; a region that has been associated with the regulation of responses to threatening situations).

In sum, advances in imaging brain function in the living human brain have provided the opportunity to peer in on the neural activity that occurs during exposure to painful events. The research discussed in this chapter provides some preliminary evidence that there are commonalities among neural circuits involved in mediating the experience of both acute physical and social pain. If there is in fact an overlap, one implication is that any interventions targeting one type of pain should also, by some means, act to influence the other type of pain. In the next section we examine evidence that a widely used over-the-counter analgesic may also quell the pain of social injury.

Effect of Reducing Physical Pain on Social Pain

One implication of an overlap in the neural substrates of both social and physical pain is that any factor that decreases one type of pain should decrease the other type. A recent study by DeWall et al. (2010) sought to investigate whether a widely used physical pain reliever, acetaminophen, would soothe the sting of social pain. In one experiment, DeWall et al (2010) randomly assigned participants to receive either 1,000 mg/day of acetaminophen (Tylenol) or placebo over a period of 21 days. Each evening participants recorded in a diary the degree to which they experienced social pain by answering questions such as “Today, being teased hurt my feelings” and “Today, I rarely felt hurt by what other people said or did to me.” The researchers discovered that whereas individuals receiving the placebo reported no change in hurt feelings, individuals taking the acetaminophen reported a significant decline in hurt feelings over the course of the study. In a second experiment, participants were randomly assigned to consume 2,000 mg/day of acetaminophen or a placebo for 21 days, then were scanned using fMRI as they experienced social exclusion in the context of a Cyberball game. DeWall et al. found that, compared with those taking placebo, participants who had taken acetaminophen showed significantly less activity in the dACC and bilateral anterior insula during an exclusion event versus inclusion. Therefore, acetaminophen, a drug typically taken to ease physical pain, was found to calm the hurt brought on by everyday social events. Moreover, following an acute exclusion event, acetaminophen decreased neural activity in brain areas previously linked to social pain. This research has shown that interventions that typically calm physical pain have the potential to also calm the hurt of social disconnectedness. We next turn to the question of what happens to physical pain under conditions of strengthened social connectedness.

Social Support and Physical Pain Sensitivity

According to Panksepp (2010), social environments may be cause for pain but they also provide a powerful resource for coping with pain. There is evidence that feelings of social connectedness and support can have a beneficial impact on physical pain. For example,

Social Pain

social support has been found to be negatively associated with the pain of childbirth (Klaus et al., 1986), postoperative pain (Kulik & Mahler, 1989; Mitchinson et al., 2008), and clinical opiate use (Mitchinson et al., 2008). Most of this research, however, is correlational and therefore it is not possible to know whether social support eases pain or people experiencing lower levels of pain are able to garner (or at least perceive) more social support.

In one of the first experimental studies examining the effects of social support on pain, Brown, Sheffield, and Robinson (2003) asked young adults to plunge an arm into painfully cold ice water (the cold-pressor task). Participants performed this task either alone or with the companionship of a friend or stranger. Companions were instructed to provide active support (engaging in the task as much as possible), passive support (quiet, making little eye contact), or interaction support (interacting with the partner as much or little as they wished). Brown and colleagues found that recipients of active and passive support reported less physical pain than their counterparts performing the task either alone or with interaction support. The researchers suggest that the reason that the interaction support group failed to show a hypoalgesic effect similar to what was achieved under the active and passive support conditions is because negative transactions may have occurred that undermined the benefits of any positive support. The data support the conclusion (p. 168) that physically painful stimuli can be made less painful in the presence of a consistently socially supportive companion compared with the person who must endure it alone.

While Brown, Sheffield, Leary, and Robinson (2003) demonstrated the consequences of one type of socially connecting experience (the presence of a supportive companion) on physical pain processing, Master et al. (2009) extended this research by investigating the effects of two other forms of socially connecting experiences on physical pain. In their study, women in long-term romantic relationships were invited to the lab with their partners. Participants placed an arm behind an opaque curtain and this arm was then exposed to a series of thermal stimuli under several different conditions: while holding the hand of the partner (who sat behind a curtain), holding the hand of a male stranger, holding an object, viewing the partner's photo, viewing a photo of the male stranger, viewing photos of an object (chair), and viewing a fixation crosshair. Half the thermal stimulations induced low pain, whereas half induced high pain. Participants rated the unpleasantness of each stimulation. The researchers found that holding the partner's hand led to significantly lower pain ratings than holding an object or holding a stranger's hand. A similar pattern of findings emerged in the case of the photographs. Viewing a partner's photo led to significantly lower pain ratings compared with viewing photos of an object or of the stranger. Thus, being primed with photos of a loved one may be sufficient to activate associated mental representations of being loved and supported and thus reduce reactivity to pain.

In a somewhat different approach, Eisenberger, Taylor, Gable, Hilmert, and Lieberman (2007) used an experience sampling methodology to record the degree to which participants felt socially supported at several times throughout the day over a period of 10 days.

Social Pain

Subsequently, participants were placed in an fMRI scanner and played a game of Cyberball in which they were excluded from play at a certain point. Cortisol reactivity to a social stressor (speech and mental arithmetic in front of an evaluative audience) was assessed prior to the daily experience sampling. The researchers found that during the Cyberball rejection event, two brain regions were related to daily levels of social support as well as cortisol reactivity. Specifically, higher levels of reported daily social support were associated with attenuated cortisol reactivity and with lower levels of activity in the dACC and BA 8 (a subregion of the prefrontal cortex known to be associated with emotional distress) within the context of the Cyberball social rejection. They further found that this relationship between high daily social support and cortisol reactivity in the face of the social stressor, was mediated by individual differences in dACC and BA 8 reactivity. The inverse relationship between social support and neural and neuroendocrine reactivity to a stressful event suggests that stressors somehow have a diminished effect in people who experience higher levels of social support compared with those with lower levels of social support.

In sum, these studies provide converging evidence that social support curtails both the experience of pain and neural signatures known to be positively associated with pain. The next section examines another way by which social experience influences physical pain, this time when social connectedness is threatened.

Effects of Acute Social Pain on Physical Pain Experience

With human health being so extensively influenced by social relationships, social injury (e.g., loss, rejection, failure to connect) may be processed as menacing in a fashion similar to physical injury. When facing an imminent threat, the body responds by discharging a host of physiological measures aimed at facilitating survival—the so-called flight-fight-freeze (FFF) response (Bracha et al, 2004). These measures include elevating heart rate, quickening respiration, dilating pupils, interrupting digestion, and increasing sweating. The FFF response also typically involves hypoalgesia, a reduction in pain sensitivity, because pain would be counterproductive if it prevents an organism from surviving a threat. For example, prey displaying behaviors reflecting injury would be preferred by predators. Evolution, therefore, may have favored animals that expressed stress-induced hypoalgesia under threatening situations (Butler & Finn, 2009). This phenomenon of threat-induced hypoalgesia has been very well established in the literature using animal models (e.g., Amit & Galina, 1986; Butler & Finn, 2009). Endogenous hypoalgesic responses have been witnessed in mice as a result of exposure to numerous nonsocial stressors such as foot shocks (Chesher & Chan 1977), food restriction (Wideman et al, 1996), and restraint (Costa et al, 2005). Threat-induced hypoalgesia has been observed in humans upon exposure to spiders among spider-phobics (Janssen & Arntz, 1996), virtual reality game playing (p. 169) (Hoffman et al, 2001), mental arithmetic (Flor & Grüsser, 1999), and electric shock (Rhudy & Meager, 2000, 2003). For example, Rhudy and Meager (2000) randomly

Social Pain

assigned participants to three emotion-induction conditions: they provoked fear in some participants by exposing them to a painful electric shock, anxiety in others through the threat of shock, and did nothing to the third group. They found that the fear elicited by exposure to the shock caused a significant increase in pain threshold whereas the anxiety prompted by threat of shock led to an increase in pain reactivity.

For many social animals, especially humans, social defeat, loss, and exclusion may be among the most critical threats to safety of all. If so, such threats may well trigger FFF responses. Research on nonhuman animals has indeed revealed analgesic responses to a range of social stressors including social isolation (Puglisi-Allegra & Oliverio, 1983), defeat experiences (Kavaliers, 1988), and social conflict (Rodgers & Hendrie, 1983). Just as with FFF responses to physical threats, then, one response to exclusion may be the activation of endogenous pain modulation systems. Thus, we submit that the occurrence of a socially painful event presents a potent threat in and of itself, which is capable of provoking the same response that any acute threat would—the invocation of the body's stress response—with the result being the modulation of physical pain. In short, social injury should provoke a social threat-induced hypoalgesia.

Social threat has been shown to promote hypoalgesia in humans. Some participants in a study by DeWall and Baumeister (2006) were randomly assigned to receive false feedback that they would live a life marked by social isolation (friends would drift away, marriages would be short-lived). Other participants were either told that they would enjoy stable, rewarding relationships throughout life or that they would become increasingly accident prone. The group receiving the dire prediction of a life alone reported significantly higher pain thresholds and greater pain tolerance (i.e., lower pain sensitivity) relative to those not receiving this grim forecast. Although a reliable hypoalgesic effect of social disconnection was uncovered, the manipulation used in this study was arguably so severe that the results may not generalize to the kinds of experiences people are likely to encounter in daily life and therefore prompts the question, "Is such a strong shock required to produce hypoalgesia, or might reduced pain sensitivity occur as a consequence of somewhat more everyday social interactions?"

To investigate this question we asked healthy, pain-free undergraduates to work with a partner on a structured social task that entailed taking turns answering questions about themselves (Borsook & MacDonald, 2010). Though the partner posed as another participant, she was in fact an experimental confederate instructed to behave in one of two ways. For the participants randomly assigned to the negative encounter condition, the confederate's behavior was standoffish. She smiled infrequently, made little eye contact, used a closed body posture, and kept her answers brief. For the positive encounter group, the confederate showed great interest in the participant, used affirmative phrases ("I agree with you," "Absolutely true!"), smiled often and made frequent eye contact. A control group was asked to privately think about answers to the personal questions. We found that participants exposed to the negative social encounter with the confederate reported significantly lower pain intensity and unpleasantness after the exchange relative to baseline, whereas the positive exchange and no-interaction control groups presented

no change in pain ratings. The social hypoalgesic effect observed in our study is consistent with prior research (e.g., DeWall & Baumeister, 2006) yet was achieved via a considerably more naturalistic social experience, suggesting that social hypoalgesia may be a relatively common event in daily life.

Given the extensive importance of the social network to human health and survival, it seems reasonable to expect that damage to this network may very well pose a considerable threat. Acute social injury and the resulting social pain should therefore be regarded as threats, potentially provoking a physiological response, including hypoalgesia, just as would any other perceived threat. Results from studies in our own lab as well as others have support this prediction.

Why Do Both Social Disconnection and Social Support Lead to Hypoalgesia?

We saw in the last section that socially disconnecting experiences had the effect of dulling pain sensitivity in both animals and humans. But earlier we saw evidence that cues of social support (Brown, Sheffield, Leary, and Robinson, 2003; Master et al., 2009) and positive affect (Rhudy et al., 2008) also prompted hypoalgesic effects. How can such diametrically opposed experiences both lead to the same hypoalgesic outcome? Furthermore, to the extent that socially threatening experiences induce negative affect (Buckley, Winkel & Leary, 2004; Williams, Cheung, & Choi, 2000; but see DeWall & Baumeister, 2006), negative affect has been (p. 170) shown to be related to an *increase* in pain sensitivity (Rhudy & Meagher, 2000; Rhudy et al., 2008). How can all these seemingly conflicting outcomes be reconciled?

One empirically-supported theory proposed by Rhudy and colleagues (see Rhudy et al., 2008) contends that the influence of emotions on pain experience depends on both emotional *valence* (positive or negative) and *arousal* level (mild, intense). Furthermore, the exact effect of increasing levels of arousal depends on whether affective valence is positive or negative. Rhudy and colleagues point to considerable research from their own lab and others showing that moderate levels of positive affect reliably produce moderate levels of hypoalgesia, with very intense positive affect (e.g., genital stimulation) producing profound pain inhibition. This hypoalgesic effect of positive affect provides one potential explanation for the findings of studies showing pain-inhibiting consequences of social support.

By what mechanisms social support (and the concomitant positive affect) provokes hypoalgesia is not known. However, one possibility is that social support works by way of a stress-buffering process. According to a widely regarded model of the stress response (Lazarus & Folkman, 1984), the recognition of a threat instigates a process of appraisal (consciously and/or subconsciously) that takes account of an individual's coping resources such as prior exposure to the threat, assessments of one's own capabilities, and past successes or failures. It is possible that social support is taken as a resource that can

Social Pain

be brought to bear on a threat, thereby reducing its magnitude or enhancing the individual's perceived coping capacity (Cohen, Gottlieb & Underwood, 2000; Cohen & Wills, 1985). If we consider physical injury and/or the resulting pain to be the threats in the Brown, Sheffield, Leary, and Robinson (2003) and Master et al. (2009) studies, then perhaps social support diminished the perceived menace or enhanced the perceived manageability of the injury represented by the pain signal. With diminished danger, pain might abate as well. Another (related) possibility is that the positive affect generated by social support might facilitate an awareness of a broader array of creative possibilities for coping with pain. Extensive work by Fredrickson (2001, 2009) has shown that whereas negative emotions narrow one's focus to prepare the body to respond in particular ways, positive emotions can expand cognitive and behavioral repertoires, increasing the number of possible responses we can perceive. Recent evidence (Harmon-Jones & Gable, 2009) has revealed that this broadening effect of positive affect is most likely to occur under conditions of a withdrawal motivation, which presumably would be the case under conditions of painful stimuli. With a broadened perspective and enhanced creative potential, it seems likely that any problem, even pain, may seem more manageable against a backdrop of an expanded canvas of possibilities.

Whatever the mechanism, if positive affect produces hypoalgesia, how is it possible that negative affect, presumably elicited during times of social exclusion, would similarly inhibit pain? Rhudy et al. (2008) point out that in most studies, negative emotions have the effect of *facilitating* pain sensitivity, and the degree of hyperalgesia increases as negative affect increases in intensity but only up to a point. At around the point of moderate arousal, there is a threshold at which the effect of negative affect on pain reverses and nociceptive reactions become *inhibited*. In one study, for example, Rhudy and Meagher (2001) found that merely threatening people with painful shocks led to hyperalgesia, whereas exposure to shocks led to hypoalgesia. Although both groups similarly reported negative affect, the shocked group reported significantly elevated arousal compared with the threatened-only group. Rhudy and colleagues suggest that the stress-induced hypoalgesic effects observed in numerous animal and human studies occur because the stressors employed are of sufficient intensity to surpass this reversal threshold point, often because they are inescapable (which is associated with strong arousal) and are especially threatening.

The participants in the DeWall and Baumeister (2006) and Borsook and MacDonald (in press) studies both demonstrated hypoalgesia, despite an apparently wide gap in the intensity of the manipulations (life spent alone vs. an unrewarding social encounter). The valence-arousal theory implies that the reason for these outcomes is that both manipulations (even the less intense one) provoked an arousal level that exceeded the reversal threshold. Given the vital importance of social connectedness to human health, it seems possible that seemingly minimal signs of social network weakening could be processed as a formidable threat, which would elicit intense arousal. This explanation accords with the fact that in animal studies socially disconnecting stressors (isolation, conflict, etc.) almost always elicit hypoalgesia.

Social Pain

In sum, cues of a supportive social environment would signal that valuable resources are available to cope with injury, thereby provoking pain inhibition (p. 171) systems. On the other hand, by virtue of the danger associated with social disconnection, cues of social injury would signal a formidable threat, likewise provoking pain inhibition processes. Thus, it may be that hypoalgesia is an appropriate response both during times of social disconnection and social support but that it occurs for different reasons and via different mechanisms.

Future Directions

What Are the Implications of Chronic Social Pain?

Although long distinguished from acute pain only by duration, chronic physical pain is now viewed as distinguishable on a number of factors. Chronic physical pain has been shown to be associated with structural and functional changes in cerebral tissue (Apkarian et al., 2004b; May, 2008), and spontaneous pain episodes in chronic pain patients are correlated with entirely different neural activation patterns than those seen during acute pain events (Apkarian et al., 2005). For example, whereas acute pain tends to be correlated with increased activity in the ACC, IC, S1, and Thalamus, spontaneous pain episodes in chronic pain patients are associated with elevated activity in prefrontal regions of the cortex (i.e., prefrontal cortex [PFC]; Apkarian et al., 2005). In sum, long-term pain leads to a process of pain *chronification*, which entails structural and functional changes that alter the brain's response to noxious stimuli. In some cases, this chronification process is associated with the persistence of pain long after an instigating injury has healed. Indeed, it can be said that acute pain is a symptom of a condition (injury), whereas chronic pain is a condition in and of itself.

With such differences between acute and chronic pain there are grounds to expect that these differences could be influential factors guiding the dynamics between social and physical pain. First, given the presumption of a neurophysiological overlap between social and physical pains, if chronic physical pain leads to particular patterns of structural and functional changes in the brain, might chronic social pain lead to similar changes? For example, people who endure chronic feelings of painful loneliness and disconnection may exhibit neural adaptations similar to those occurring for people enduring chronic physical pain. Eisenberger et al. (2003, 2007) have found elevated activity in the dACC in response to an acute episode of exclusion. Because the transition from acute to chronic pain (i.e., chronification) is associated with a shift in activity from the classical pain matrix regions (e.g., ACC, IC, and S1) toward the PFC, would greater activity be observed in the PFC, instead of the dACC, in people suffering chronic social pain in response to, say, a task requiring them to recall/relive an episode of exclusion?

Second, if long-term social pain leads to neural changes that mirror chronic physical pain, would chronic social pain make the chronification of physical pain more likely or more rapid? Repeated exposure to stressful events has been found to alter pain responses in

Social Pain

animals (Coutinho et al., 2002; Dickinson, Leach & Flecknell, 2009; Rivat et al., 2007). For example, Rivat et al. (2007) found that while naïve rats (who had no prior exposure to the experimental stressors or resulting physiological responses) displayed stress-induced hypoalgesia, experienced rats exhibited hyperalgesia, a phenomenon known as latent pain sensitization. Coutinho et al. (2002) examined the effects of separating infant rats from their mothers for 180 minutes daily. Pain reactions were significantly higher, and stressed induced hypoalgesia significantly lower in the maternally separated rats compared to their nonisolated counterparts. Altered pain responses resulting from early exposure to stressors have also been documented in humans. For example, Taddio, Katz, Ilerich, and Koren (1997) found that circumcision in newborn infants led to increased pain responses 4 to 6 months after surgery.

Third, chronic pain patients often struggle with feelings of disconnection, being misunderstood, frustration that their condition is invisible, and not being able to express their difficulties with pain for fear that they will not be accepted. Yet, remarkably few studies have tackled how such social realities impact the lives of people with chronic pain, including the pain experience itself and the ability to function in life.

What Are the Effects of Physical Pain On Social Perceptions?

The investigation of the dynamics between social experience and physical pain has been almost entirely one way: what influence does social experience have on physical pain? It is possible, however, that the arrow of influence points the other way as well; that the presence of physical pain may alter the perception of social experiences. The impact of physical pain on a wide range of cognitive and emotional tasks has already been documented (Apkarian, Sosa, Krauss, et al., 2004a) and there is considerable evidence that people in pain (p. 172) (at least chronic pain) exhibit information biases toward pain stimuli, selectively attending to and processing pain- and illness-related stimuli (Pincus & Morley, 2001). Therefore given the crucial importance of social connections there is reason to believe that social signals may be particularly vulnerable to the influence of physical pain.

How Can Brain Imaging Be Used to Expand Knowledge of the Neural Correlates of Social Pain?

The fMRI studies by Eisenberger and colleagues (Eisenberger et al., 2003, 2007) have offered a first glimpse at the neural consequences of social exclusion. However, the features of the most commonly used experimentally induced exclusion paradigm, Cyberball, may limit the generalizability of these studies' results, suggesting that future studies should employ alternative exclusion methodologies. First, Cyberball may confound rejection with expectation violation (i.e., surprise). The arbitrary exclusion during the game may well lead to feelings of social distress, but might also induce surprise and confusion. Eisenberger et al. (2003) found increased dACC activity in response to exclusion due to an ostensible technical glitch (implicit exclusion). We suggest that there might have been a degree of expectancy violation in this implicit condition as well as in the explicit condi-

tion. Whereas in the explicit condition the surprise was being suddenly excluded for no apparent reason in the middle of a game, the implicit condition may have provoked surprise that one was not able to play a game one was expecting to play. It appears, therefore, that both exclusion conditions may have entailed some degree of *surprise*. Because dACC activation has been linked to expectancy violation (Botvinick, Cohen, & Carter, 2004), the question arises as to whether the dACC activation observed by Eisenberger et al. (2003) mainly reflects expectancy violation or social pain. Using a paradigm that separated expectancy violation from social feedback (acceptance vs. rejection), Somerville, Heatherton, and Kelley (2006) found evidence suggesting that the dorsal region of the ACC (dACC) was sensitive to expectancy violation, with increasing activity when feedback did not match expectations, whereas the ventral region was sensitive to feedback type, showing greater activity when the participant was liked versus not liked. Future studies should employ a variety of exclusion methods¹ and attempt to distinguish between the effects of social distress and other psychological states (such as expectancy violation, surprise, frustration, etc.).

Second, activity in the ACC, as well as other regions associated with the “pain matrix” such as the AIC, thalamus, and prefrontal cortex (PFC) is by no means specific to pain and occurs under a wide array of conditions other than pain (Peyron, Laurent, & Garcia-Larrea, 2000; Wager, 2005). For example, dACC activity has been linked to numerous functions including effortful thinking (of the kind that occurs when learning a new skill; Allman et al., 2001), error detection (Gehring & Knight, 2000), performance monitoring (van Veen, Holroyd, Cohen, et al., 2004), reward assessment (Knutson et al., 2000), and expectancy violation (Botvinick, Cohen, & Carter, 2004). The existence of pain cannot be assumed, therefore, by dint of merely observing activity in these regions. Combining imaging data from numerous studies employing a wide array of exclusion methods, along with improved social pain measures, will help to boost confidence that neural correlates of exclusion experiences reflect predominantly pain, rather than other phenomena.

What Is the Best Way to Operationalize Social Pain?

An important issue in the social pain literature is the measurement of social pain. Several studies (Eisenberger et al., 2003, 2006, 2007; DeWall et al., 2010) have used a brief instrument that purportedly measures social distress. Items include “I felt liked,” “I felt rejected,” “I felt invisible” and “I felt powerful.” The extent to which this instrument indexes the presence of pain, per se, is not known. The validity of this and other social pain measures has important implications for the interpretation of social pain research. For example, in the study by Eisenberger et al. (2006), sensitivity to physical pain related to sensitivity to exclusion and vice versa. But because mild to moderate negative affect prompts heightened pain sensitivity (Rhudy & Meagher, 2000, 2001, Rhudy et al., 2008), if social distress reflects general negative affect, then the positive correlation between social distress and physical pain may simply reflect the impact of negative affect on physical pain rather than any special relationship between social and physical pain. Future studies should include alternative measures that employ items relating to pain specific-

ly (“hurts,” “aches,” “stings,” “wounded,” “upset,” etc.) such as Leary and Springer’s (2001) measure of hurt feelings proneness (e.g., DeWall et al., 2010).

Conclusion

A vast and growing literature has demonstrated that social connectedness can have an immense (p. 173) impact on human health and well-being. The importance of relationships to physical integrity is perhaps the reason why we evolved such a strong drive to be accepted, and may also be the key factor that prompted the exaptation of pain to signal social injury. Social pain is the emotionally aversive pain-like experience that can attend socially excluding events (and the memory of such events) and in this chapter we have explored evidence that social and physical pain share a number of phenomenological as well as biochemical and neural features. Some of the earliest evidence of this overlap comes from a series of studies showing that opioids, a peptide long known to be intimately involved in regulating physical pain, can also effectively ease social distress and modulate social-threat induced hypoalgesia, and that chronic exposure to social distress impairs opiate efficacy in reducing physical pain. We explored findings from recent brain imaging studies showing that cortical activity in people experiencing the distress of exclusion is similar to that of people receiving nociceptive stimuli, and that people’s neural responses to an exclusion experience effectively predict the tendency to experience social distress in everyday life. We also looked at research reporting hypoalgesic responses under the opposing conditions of both social support and social threat and explored candidate mechanisms by which these responses occur. Finally, in spite of a growing body of evidence linking social and physical pain, there remain several outstanding issues that need to be addressed in future research and we identified three such issues. Being the highly social animals that we are, social exclusion feels awful. But more than that, exclusion can be as injurious to health as physical injury and as such, it can provoke a feeling that is remarkably pain-like, with consequences that parallel those of physical pain. We look forward to seeing understanding of the phenomenon of social pain and its relation to physical pain expand, ideally with increasing application in clinical settings to help people suffering from both types of pain.

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Notes:

(1.) Especially those known to produce strong responses, such as the "life alone" (Twenge, Baumeister, Tice, & Stucke, 2001) and the "no one chose you" (Nezlek et al., 1997) methods. In the "life alone" approach, participants are told that, on the basis of a personality test, it can be predicted that they will experience a life of isolation (or a life filled with rewarding relationships). In the "no one chose you" paradigm, participants are asked to select whom they would like to work with on a task, from among a group of individuals they had just met, and then are subsequently informed that no one (or everyone) chose them as partners.

Social Pain

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