REPORTS

Does Rejection Hurt? An fMRI Study of Social Exclusion

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A neuroimaging study examined the neural correlates of social exclusion and tested the hypothesis that the brain bases of social pain are similar to those of physical pain. Participants were scanned while playing a virtual ball-tossing game in which they were ultimately excluded. Paralleling results from physical pain studies, the anterior cingulate cortex (ACC) was more active during exclusion than during inclusion and correlated positively with self-reported distress. Right ventral prefrontal cortex (RVPFC) was active during exclusion and correlated negatively with self-reported distress. ACC changes mediated the RVPFC-distress correlation, suggesting that RVPFC regulates the distress of social exclusion by disrupting ACC activity.

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It is a basic feature of human experience to feel soothed in the presence of close others and to feel distressed when left behind. Many languages reflect this experience in the assignment of physical pain words ("hurt feelings") to describe experiences of social separation ([1]). However, the notion that the pain associated with losing someone is similar to the pain experienced upon physical injury seems more metaphorical than real. Nonetheless, evidence suggests that some of the same neural machinery recruited in the experience of physical pain may also be involved in the experience of pain associated with social separation or rejection ([2]). Because of the adaptive value of mammalian social bonds, the social attachment system, which keeps young near caregivers, may have piggybacked onto the physical pain system to promote survival ([3]). We conducted a functional magnetic resonance imaging (fMRI) study of social exclusion to determine whether the regions activated by social pain are similar to those found in studies of physical pain.

The anterior cingulate cortex (ACC) is believed to act as a neural "alarm system" or conflict monitor, detecting when an automatic response is inappropriate or in conflict with current goals ([4]–[6]). Not surprisingly, pain, the most primitive signal that "something is wrong," activates the ACC ([7], [8]). More specifically, dorsal ACC activity is primarily associated with the affectively distressing rather than the sensory component of pain ([7]–[9]).

Because of the importance of social bonds for the survival of most mammalian species, the social attachment system may have adopted the neural computations of the ACC, involved in pain and conflict detection processes, to promote the goal of social connectedness. Ablating the cingulate in hamster mothers disrupts maternal behavior aimed at keeping pups near (10), and ablating the cingulate in squirrel monkeys eliminates the spontaneous production of the separation cry, emitted to reestablish contact with the social group (11). In human mothers, the ACC is activated by the sound of infant cries (12). However, to date, no studies have examined whether the ACC is also activated upon social separation or social rejection in human subjects.

Right ventral prefrontal cortex (RVPFC) has been implicated in the regulation or inhibition of pain distress and negative affect (13–16). The primate homolog of VPFC has efferent connections to the region of the ACC associated with pain distress (17, 18), suggesting that RVPFC may partially regulate the ACC. Additionally, electrical stimulation of VPFC in rats diminishes pain behavior in response to painful stimulation (19). More recently in humans, heightened RVPFC activation has been associated with improvement of pain symptoms in a placebo-pain study (16).

Given that even the mildest forms of social exclusion can generate social pain (20), we investigated the neural response during two types of social exclusion: (i) explicit social exclusion (ESE), in which individuals were prevented from participating in a social activity by other players engaged in the social activity, and (ii) implicit social exclusion (ISE), in which participants, because of extenuating circumstances, were not able to join in a social activity with other players.

fMRI scans were acquired while participants played a virtual ball-tossing game ("CyberBall") with what they believed to be two other players, also in fMRI scanners, during which the players eventually excluded the participant (21). In reality, there were no other players; participants were playing with a preset computer program and were given a cover story to ensure that they believed the other players were real (22).

In the first scan (ISE), the participant watched the other "players" play CyberBall. Participants were told that, because of technical difficulties, the link to the other two scanners could not yet be made and thus, at first, they would be able to watch but not play with the other two players. This cover story was intended to allow participants to view a scene visually identical to ESE without participants believing they were being excluded. In the second scan (inclusion), participants played with the other two players. In the final scan (ESE), participants received seven throws and were then excluded when the two players stopped throwing participants the ball for the remainder of the scan (~45 throws). Afterward, participants filled out questionnaires assessing how excluded they felt and their level of social distress during the ESE scan (22).

Behavioral results indicated that participants felt ignored and excluded during ESE ($t =$ 5.33, $P < 0.05$). As predicted, group analysis of the fMRI data indicated that dorsal ACC (Fig. 1A) ($x = -8$, $y = 20$, $z = 40$) was more active during ESE than during inclusion ($t =$ 3.36, $r = 0.71$, $P < 0.005$) (23, 24). Self-reported distress was positively correlated with ACC activity in this contrast (Fig. 2A) ($x = -6$, $y = 8$, $z = 45$, $r = 0.88$, $P < 0.005$; $x = -4$, y

= 31, $z = 41$, $r = 0.75$, $P < 0.005$), suggesting that dorsal ACC activation during ESE was associated with emotional distress paralleling previous studies of physical pain (7, 8). The anterior insula ($x = 42$, $y = 16$, $z = 1$) was also active in this comparison ($t = 4.07$, $r = 0.78$, $P < 0.005$); however, it was not associated with self-reported distress.
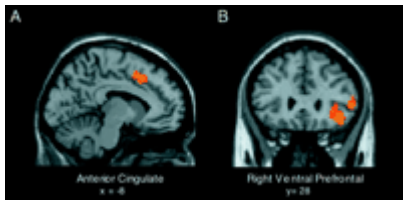

Fig. 1. (A) Increased activity in anterior cingulate cortex (ACC) during exclusion relative to inclusion. (B) Increased activity in right ventral prefrontal cortex (RVPFC) during exclusion relative to inclusion. [View Larger Version of this Image (48K GIF file)]
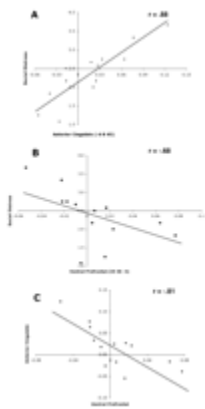

Fig. 2. Scatterplots showing the relation during exclusion, relative to inclusion, between (A) ACC activity and self-reported distress, (B) RVPFC and self-reported distress, and (C) ACC and RVPFC activity. Each point represents the data from a single participant. [View Larger Version of this Image (10K GIF file)]

Two regions of RVPFC were more active during ESE than during inclusion (Fig. 1B) ($x = 42$, $y = 27$, $z = -11$, $t = 4.26$, $r = 0.79$, $P < 0.005$; $x = 37$, $y = 50$, $z = 1$, $t = 4.96$, $r = 0.83$, $P < 0.005$). Self-reported distress was negatively correlated with RVPFC activity during ESE, relative to inclusion (Fig. 2B) ($x = 30$, $y = 34$, $z = -3$, $r = -0.68$, $P < 0.005$). Additionally, RVPFC activation ($x = 34$, $y = 36$, $z = -3$) was negatively correlated with ACC activity ($x = -6$, $y = 8$, $z = 45$) during ESE, relative to inclusion ($r = -0.81$, $P < 0.005$) (Fig. 2C), suggesting that RVPFC may play a self-regulatory role in mitigating the distressing effects of social exclusion.

ACC activity ($x = -6$, $y = 8$, $z = 45$) mediated the direct path from RVPFC ($x = 34$, $y = 36$, $z = -3$) to distress (Sobel test, $Z = 3.16$, $P < 0.005$). After controlling for ACC activity, the remaining path from RVPFC to distress was no longer significant (ß = -0.17, $P > 0.5$). This mediational model is nearly identical to the results from previous research on the self-regulation of physical pain (16).

ISE, relative to inclusion, also produced significant activation of ACC ($x = -6$, $y = 21$, $z = 41$; ($z = 41$, $t = 4.34$, $I = 0.78$, $P < 0.005$). To preserve the cover story, self-reported distress was not assessed after this condition, and thus we could not assess the relation between ACC activity during ISE and perceived distress. However, no RVPFC activity was found in this comparison, even at a $P = .05$ significance level, suggesting that the ACC registered this ISE but did not generate a self-regulatory response.

In summary, a pattern of activations very similar to those found in studies of physical pain emerged during social exclusion, providing evidence that the experience and regulation of social and physical pain share a common neuroanatomical basis. Activity in dorsal ACC, previously linked to the experience of pain distress, was associated with increased distress after social exclusion. Furthermore, activity in RVPFC, previously linked to the regulation of pain distress, was associated with diminished distress after social exclusion.

The neural correlates of social pain were also activated by the mere visual appearance of exclusion in the absence of actual exclusion. The pattern of neural activity associated with ISE and ESE provides some challenges to the way we currently understand exclusion and its consequences. Although the neural correlates of distress were observed in both ISE and ESE, the self-regulation of this distress only occurred in response to ESE. Explicit awareness of exclusion may be required before individuals can make appropriate attributions and regulate the associated distress.

Dorsal ACC activation during ESE could reflect enhanced attentional processing, previously associated with ACC activity ([4], [5]), rather than an underlying distress due to exclusion. Two pieces of evidence make this possibility unlikely. First, ACC activity was strongly correlated with perceived distress after exclusion, indicating that the ACC activity was associated with changes in participants' self-reported feeling states. Second, although inclusion is likely to require greater attentional processing than does ISE to facilitate participation in the game, there was greater ACC activity during ISE than during inclusion, indicating that the ACC activity was not fully attributable to heightened attention.

Because of the need to maintain a realistic situation in which participants would genuinely feel excluded, the study did not contain some of the controls typical of most neuroimaging studies. For instance, the conditions were always implemented in the same order so as to keep expectations consistent from one scan to the next across participants. It was especially critical that ESE came last to prevent expectations of possible exclusion from contaminating the other conditions. There was only a single ESE period to preserve ecological validity. This modification, however, diminishes, rather than increases, the likelihood of Type I errors.

This study suggests that social pain is analogous in its neurocognitive function to physical pain, alerting us when we have sustained injury to our social connections, allowing restorative measures to be taken. Understanding the underlying commonalities between physical and social pain unearths new perspectives on issues such as why

physical and social pain are affected similarly by both social support and neurochemical interventions (*2*, *3*, *25*), and why it "hurts" to lose someone we love (*1*).

References and Notes

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