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What is This?

Broken Hearts and Broken Bones: A Neural Perspective on the Similarities Between Social and Physical Pain

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Abstract

Although it is common to describe experiences of social rejection or loss with words typically reserved for physical pain, the idea that these social experiences might actually be experienced as painful seems more far-fetched. However, accumulating evidence demonstrates that social pain—the painful feelings following social rejection or loss—may rely on pain-related neural circuitry. Here, I summarize a program of research that has explored whether social pain relies on pain-related neural regions, as well as some of the expected consequences of a physical–social pain overlap. I also discuss the implications of these findings for our understanding of social pain.

"That hurt my feelings." "He broke my heart." We have all heard these kinds of statements, many of us have said them, and all of us know what they mean. There is an unavoidable feeling of pain associated with being socially rejected, excluded, or losing those closest to us. Indeed, this experience of pain following rejection or loss seems to be a nearly universal phenomenon. Individuals across the globe, using languages as diverse as Armenian and Mandarin (MacDonald & Leary, 2005), use physical-pain words to describe experiences of "social pain"—the painful feelings associated with the threat to or loss of social connection (from rejection, exclusion, death of a loved one). Nonetheless, do experiences of social rejection or loss truly cause pain? Or is the pain associated with these social experiences simply a convenient metaphor?

Accumulating research suggests that the pain of social rejection or social loss may be more than just metaphorical. Here, I review this emerging body of evidence, which has demonstrated that social pain relies on some of the same underlying neural circuitry that is associated with the unpleasant experience of physical pain (Eisenberger, 2011; Eisenberger & Lieberman, 2004). I will discuss why evolutionary pressures may have resulted in this shared circuitry and what this shared circuitry means for our understanding of social pain.

The Evolution of Social Pain

Even though social pain is described with words typically reserved for physical pain, it seems more difficult to accept the idea that social pain may actually be experienced in a manner similar to physical pain. From an evolutionary perspective, however, it makes good sense that experiences of social rejection or disconnection might actually be experienced as painful. Humans, as a mammalian species, face a very long period of immaturity, in which they rely almost completely on others (caregivers) to obtain the necessary nourishment and protection. Later in life, connection to a social group promotes survival through shared responsibilities for food acquisition, predator protection, and offspring care. Over the course of evolutionary history, the social-attachment system—which ensures social bonding and connection—may have piggybacked onto the physical-pain system, borrowing the pain signal to highlight social disconnection and motivate social reconnection (Panksepp, 1998). In other words, to the extent that being separated from a caregiver or from the social group is detrimental to survival, feeling "hurt" by this separation may have been an adaptive way to prevent it.

Building on this idea, research from animals and humans alike supports the hypothesis that physical and social pain rely on shared neurobiological and neural substrates. Specifically, both physical and social pain rely on (a) mu-opioid-related signaling, critically involved in pain processing, and (b) shared

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neural activity in regions typically associated with the unpleasant experience of physical pain.

Shared Neurobiological and Neural Substrates

In the late 1970s, Jaak Panksepp made the startling discovery that mu-opioids, neurotransmitters best known for their role in pain processing, also played a critical role in certain behaviors related to separation distress, such as infant cries in response to being separated from the mother. Mu-opioid-related drugs, such as morphine or codeine, are best known for their pain-relieving effects and are commonly prescribed for pain management. Interestingly, though, this same class of pharmaceuticals also has profound effects on social pain. Hence, across several mammalian species, morphine, which increases mu-opioid-related activity, reduces separation-distress vocalizations made by infants when separated from their mothers, whereas naloxone, which inhibits mu-opioid-related activity, increases distress vocalizations (reviewed in Panksepp, 1998). Thus, mu-opioid-related drugs, best known for their pain-relieving effects, are also critical for reducing separation distress.

In addition to shared opioid-related activity, experiences of social and physical pain also rely on shared neural substrates, specifically those associated with the distressing experience of physical pain. Although the experience of physical pain typically "feels" like one unified negative experience, pain researchers have identified two separable components underlying painful experience: (a) a sensory component, which provides information about the objective intensity of the painful stimulus and where it is coming from (e.g., on the surface of the skin vs. from the viscera) and (b) an affective component, which codes for how distressing or bothersome the painful stimulus is. Based on the significance of the affective component of pain for signaling an aversive state and motivating behaviors to reduce it, we have hypothesized that social pain relies on neural regions involved in the affective component of pain. However, given that somatic symptoms are often reported following experiences of social pain (Leary & Springer, 2001), it is possible that the sensory component of pain may contribute to social-pain experience as well.

Along these lines, the dorsal anterior cingulate cortex (dACC) and anterior insula have been shown to contribute primarily to the affective or unpleasant component of physical pain, whereas other regions like the somatosensory cortices and posterior insula have been shown to contribute to the sensory component of pain. Thus, following neurosurgery for severe chronic pain, in which surgeons remove a portion of the dACC, patients report that although they can still "feel" painful stimulation, it "no longer bothers them" (Foltz & White, 1962). Similar findings have been demonstrated following damage to the anterior insula (Berthier et al., 1988). Interestingly, damage to regions that process the sensory component of pain (somatosensory cortices) disrupts the ability to localize pain sensation but leaves the distressing experience of pain intact (Ploner, Freund, & Schnitzler, 1999).

Neuroimaging studies reveal similar findings. Participants hypnotized to selectively increase the affective component of pain without altering the sensory component showed increased activity in the dACC, but not in regions that process the sensory component of pain (Rainville, Duncan, Price, Carrier, & Bushnell, 1997). Moreover, greater self-reported pain unpleasantness routinely correlates with neural activity in both the dACC and anterior insula (Rainville, 2002).

In addition, some of these affective-pain-related regions also contribute to basic social-pain-related behaviors, such as separation-distress vocalizations in nonhuman mammals. Thus, damage to the dACC reduces these distress vocalizations upon mother–infant separation (MacLean & Newman 1988), whereas stimulating the ACC leads to the spontaneous production of these distress vocalizations but not other types of vocalizations (Robinson, 1967).

Finally, this same set of neural regions is also activated in response to social pain in humans. In the first study to explore the neural underpinnings of social exclusion (Eisenberger, Lieberman, & Williams, 2003), participants were led to believe that they would be playing an online, virtual ball-tossing game called Cyberball with two other players (who were actually computer simulated). During an initial round of the game, participants played freely with the two other players (inclusion); during another round, participants were socially excluded when the two other players stopped throwing the ball to them (Fig. 1a). Neuroimaging analyses revealed that when participants were socially excluded (vs. included), they showed greater activity in the dACC (Fig. 1b) and anterior insula, regions often associated with the distress of physical pain. Moreover, greater activity in the dACC was associated with feeling more rejected by the exclusion episode (Fig. 1c).

Subsequent studies of rejection, exclusion, and negative social evaluation have largely supported these initial findings (reviewed in Eisenberger, 2011). Moreover, research has demonstrated that, in some cases, simply viewing images that signal social rejection—without necessarily feeling socially rejected—can activate these affective-pain-related regions as well. For example, viewing rejection-themed paintings (by Edward Hopper) activated the dACC and anterior insula (Kross, Egner, Ochsner, Hirsch, & Downey, 2007), and viewing disapproving faces (through videos that were not personally relevant) led to greater dACC activity for those higher in rejection sensitivity (Burklund, Eisenberger, & Lieberman, 2007).

In addition, a recent study demonstrated that experiences of rejection can, in some cases, activate sensory-related neural regions as well (Kross, Berman, Mischel, Smith, & Wager, 2011). Thus, participants who relived an unwanted romantic relationship break-up showed greater activity in both affective-pain-related neural regions (dACC, anterior insula) and sensory-related ones (secondary somatosensory cortex, posterior insula), and these same regions were
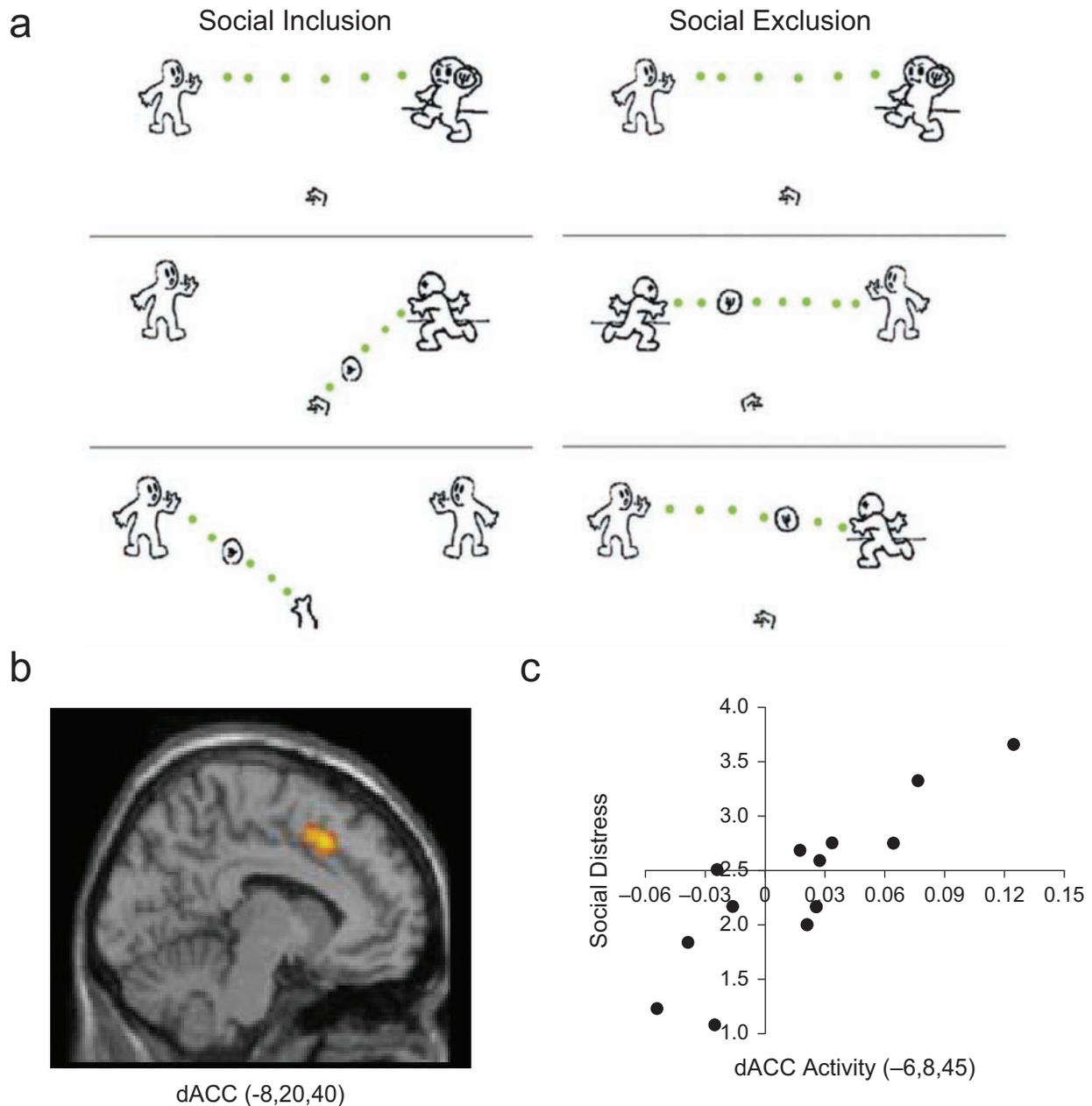
a

Social Inclusion Social Exclusion

b

dACC (-8,20,40)

c

Fig. 1. Depiction of what participants saw during the Cyberball social inclusion and exclusion tasks (a) and brain activity associated with rejection (b, c). Participants showed increased activity in the dorsal anterior cingulate cortex (dACC) during social exclusion. As shown in the plot, greater activity in the dACC was associated with greater self-reported social distress or feelings of rejection. (Adapted from "Why Rejection Hurts: The Neurocognitive Overlap Between Physical and Social Pain," by N. I. Eisenberger and M. D. Lieberman, 2004, *Trends in Cognitive Sciences, 8*, pp. 294–300. Copyright 2004 by Elsevier. Adapted with permission.

similarly activated in response to a separate physical-pain task. Additional research will be needed to further explore the role of sensory-related regions in socially painful experience.

Finally, although less is known about the neural correlates of social loss, thinking about a lost loved one activates affective-pain-related regions as well. Thus, in response to viewing images of a recently deceased loved one (vs. a stranger), participants showed greater activity in the dACC and anterior

insula (O'Connor et al., 2008). Moreover, females who lost an unborn child (vs. those who delivered a healthy baby) showed greater activity in the dACC in response to viewing images of smiling baby faces (Kersting et al., 2009).

Together, these studies suggest that varied forms of social pain—ranging from social rejection to bereavement—activate neural regions associated with the distressing emotional experience, and sometimes the sensory experience, of physical

pain. Future work will be needed to more fully explore whether these pain-related neural regions are specific to experiences resulting from the threat or experience of broken social bonds (in addition to the threat or experience of physical pain) or whether these neural regions are more generally responsive to negative affective experiences, both social and nonsocial.

Consequences of a Physical–Social Pain Overlap

One of the interesting implications of these findings is that there should be certain consequences of this shared neural circuitry. To date, we have explored two of these consequences, specifically (a) whether individuals who are more sensitive to one kind of pain are also more sensitive to the other and (b) whether factors that increase or decrease one kind of pain affect the other in a similar manner.

One of the first questions that we examined was whether individuals who were more sensitive to physical pain would also be more sensitive to social pain, an expected consequence of a physical–social pain overlap. In one study, we assessed participants' baseline sensitivity to physical pain and then explored whether this predicted their sensitivity to social exclusion. We found that individuals who were naturally more sensitive to physically painful stimulation were also more sensitive to social exclusion, reporting feeling more rejected following the Cyberball social-exclusion task (Eisenberger, Jarcho, Lieberman, & Naliboff, 2006). In a second study, we explored whether individual differences in a genetic polymorphism that relates to physical-pain sensitivity—the mu-opioid receptor gene—was also related to social-pain sensitivity (Way, Taylor, & Eisenberger, 2009). Here, we found that individuals with the version of the mu-opioid receptor gene that has been linked with increased physical-pain sensitivity reported higher levels of trait sensitivity to rejection and showed greater activity in the dACC and anterior insula in response to being socially excluded.

Another question that we have explored is whether certain factors that increase or decrease one kind of pain affect the other in a similar manner. For example, we have explored whether factors that are typically thought to decrease social pain—like social support—can also decrease physical pain and whether factors that are typically thought to decrease physical pain—like pain medication—can also decrease social pain.

To explore whether social support reduces physical pain, we asked female participants in long-term romantic relationships to rate how much pain they felt in response to a series of painful heat stimuli (delivered to their arms) as they completed a number of different tasks (Master et al., 2009). In one set of tasks, they received social support—either by holding their partners' hands or viewing pictures of their partners. In another set of control tasks, they did not receive social support; instead they either held a stranger's hand or an object or they viewed pictures of a stranger or object. When examining how each task affected pain ratings, we found that the social-support conditions (in which each participant either held her partner's hand or viewed his picture) led to reductions in pain ratings compared to the control conditions. Not surprisingly, in a neuroimaging version of this study, viewing pictures of one's partner not only reduced pain ratings but reduced pain-related brain activity as well (dACC, anterior insula; Eisenberger et al., 2011). Thus, social support, typically assumed to reduce social pain, reduces physical pain as well.

We have also explored whether certain medications typically thought to reduce physical pain, like Tylenol (generic name: acetaminophen), could also reduce social pain (DeWall et al., 2010). In a first study, participants were randomly assigned to take either a daily dose of Tylenol or a placebo for a 3-week period. In addition, during this time, they recorded their daily self-reported hurt feelings each evening. Results demonstrated that participants taking Tylenol showed a significant reduction in hurt feelings over the 3-week period, whereas participants taking placebo showed no significant change in hurt feelings. In a subsequent neuroimaging study, a separate group of participants was randomly assigned to take daily doses of Tylenol or placebo, again for a 3-week period. This time, at the end of the 3-week period, participants completed the Cyberball social-exclusion task in the fMRI scanner. Participants who had been taking Tylenol showed significantly less pain-related activity in the dACC and anterior insula in response to social exclusion than participants who had been taking the placebo (Fig. 2). Although further research will be needed to more fully understand the effects of Tylenol on socioemotional experience, this study demonstrated that Tylenol, a physical painkiller, appears to double as a social painkiller.

Conclusions

Although many of us would not hesitate to describe experiences of rejection, exclusion, or social loss as painful, it still seems difficult to imagine that these social experiences that do not physically wound us could truly lead to the same kind of pain as a broken bone or an aching stomach. However, accumulating evidence demonstrates that experiences of social and physical pain actually rely on some of the same neurobiological and neural substrates.

Of course, highlighting the shared circuitry underlying physical and social pain is not meant to suggest that these experiences are interchangeable. We know this from experience, as we can clearly differentiate between the pain of a stubbed toe and that of a social snub. Moreover, research has demonstrated clear differences between these two types of pain. For example, while individuals can relive the pain of social rejection or betrayal, they are less capable of reliving the pain of physical assault or injury (Chen, Williams, Fitness, & Newton, 2008). Still, the fact that both types of pain share overlapping neurobiological and neural substrates suggests that there are meaningful similarities in the ways in which physical and social pain are experienced.
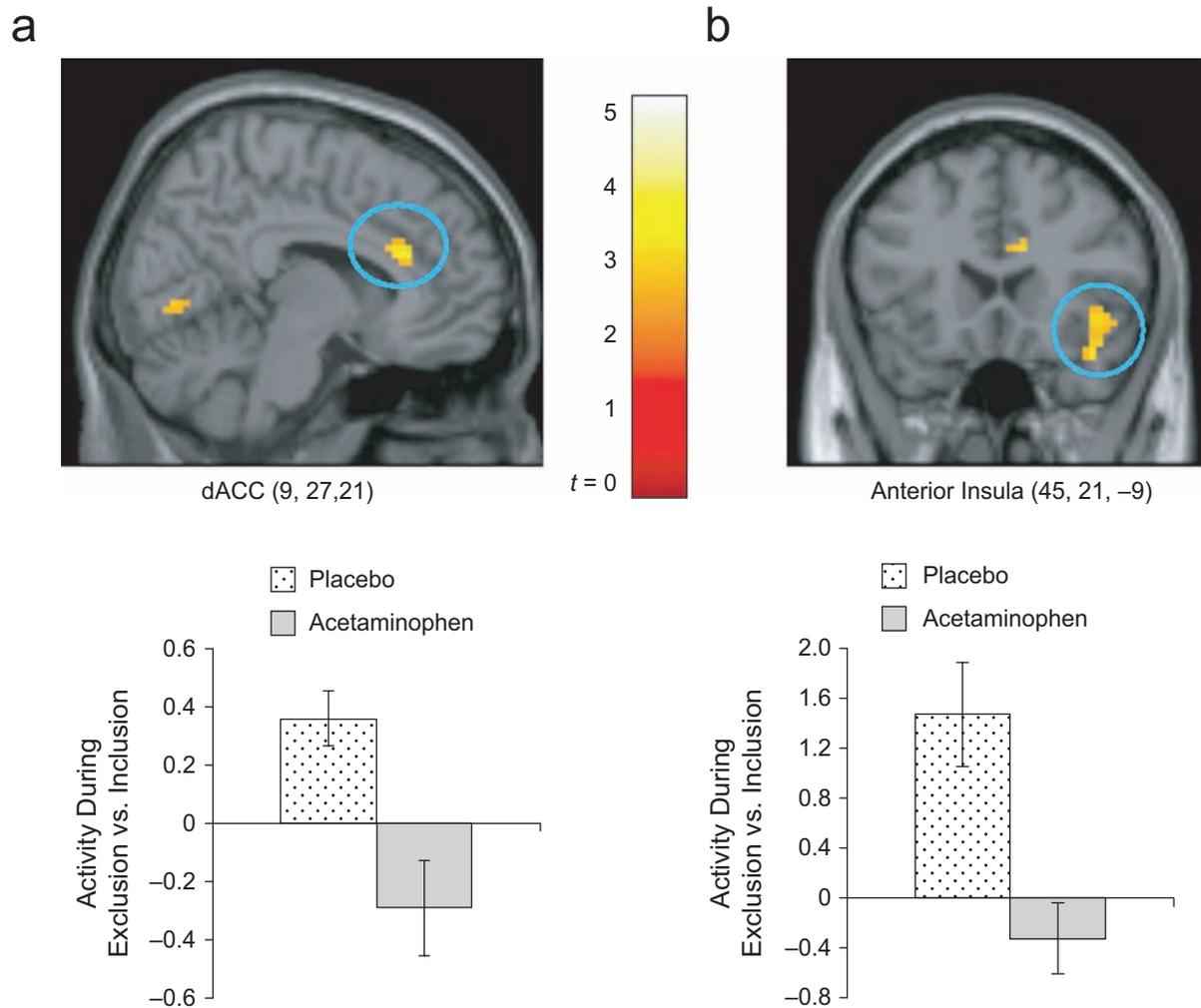
a

b

dACC (9, 27,21)

Anterior Insula (45, 21, –9)

Fig. 2. Neural activity in the (a) dorsal anterior cingulate cortex (dACC) and (b) right anterior insula during social exclusion versus inclusion for participants who took acetaminophen (Tylenol) and those who took a placebo. The brain images show neural activity during social exclusion (versus inclusion) that was greater for participants who took placebo than for those who took acetaminophen. The circled regions are those for which results are displayed in the bar graph. Reprinted from "Tylenol Reduces Social Pain: Behavioral and Neural Evidence," by C. N. DeWall, G. MacDonald, G. D. Webster, C. L. Masten, R. F. Baumeister, C. Powell, D. Combs, D. R. Schurtz, T. F. Stillman, D. M. Tice, and N. I. Eisenberger, 2010, *Psychological Science, 21*, p. 935. Copyright 2010, Association for Psychological Science. Reprinted with permission.

Given the distress and pain caused by broken social bonds, one might wonder why humans have evolved a mechanism that leads to so much suffering. It is important to keep in mind that even though social rejection or exclusion feels painful when it is occurring, these feelings serve an adaptive function. Thus, in the same way that the painful sting of a burned finger motivates us to retract from a hot object and teaches us to avoid touching it again, the pain of social rejection motivates us to avoid engaging in behaviors that might lead to social rejection. Although an exaggerated sensitivity to social pain may have negative consequences, such as an avoidance of social interactions altogether, a healthy sensitivity to social pain may be adaptive for promoting social bonds. Indeed, individuals who lack sensitivity to social pain are characterized by certain personality disorders (paranoid, schizoid, schizotypal) associated with disordered social relationships and a preference for social isolation (Wirth, Lynam, & Williams, 2010). Over the course of evolutionary history, social pain may have helped us to avoid social rejection, increasing our connection with others, our inclusion in the social group, and ultimately our chances of survival. Hence, social pain, though distressing in the moment, is an adaptation that ensures social bonding and ultimately survival.

Recommended Reading

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Acknowledgments

Declaration of Conflicting Interests

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