



Does physical pain augment anxious attachment?

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ABSTRACT

This study tested the hypothesis derived from social pain theory (MacDonald & Leary, 2005) that pain affect serves as a signal of perceived social exclusion. Participants ranging in experience of persistent physical pain completed measures of pain affect, anxious and avoidant attachment, anxiety, and depression. Higher levels of pain affect were found to relate to higher levels of anxious, but not avoidant, attachment. Further, anxious attachment partially mediated the relation between pain affect and emotional distress. These data support the conclusion that one reason individuals with persistent pain experience anxiety and depression is because of heightened concerns over rejection. The data also support the conclusion that anxious attachment is more strongly related to the fight-flight-freezing system than avoidant attachment.

KEY WORDS: adult attachment • pain • social exclusion

Attachment theory (Bowlby, 1973) argues that a biological system that strongly motivates infants and caregivers to remain close should be evolutionarily functional in that it would increase the likelihood of successful transmission of genetic material to future generations for both parties. Many attachment theorists propose that this system remains functional throughout adulthood, helping adults regulate closeness across a variety of interpersonal relationships (e.g., Hazan & Shaver, 1987). However, little is known about the physiological mechanisms that may underlie adult attachment. Social pain theory (MacDonald & Leary, 2005) suggests that pain

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affect, or the emotional unpleasantness that often accompanies physical injury, may serve as a signal of social exclusion and thus contribute to attachment regulation. One consequence of the multipurpose nature of pain affect is that pain that results from physical injury may heighten perceptions of social exclusion, and thus over time may contribute to more anxious attachment. The present study tests this hypothesis by examining attachment across individuals who vary widely in their experience of persistent pain (pain clinic patients and community members). We test whether higher levels of pain are related to more anxious attachment, and whether these rejection concerns brought on by pain can partially account for the relation between pain and emotional distress.

Early research on childhood attachment suggested three distinct styles of attachment between infants and caregivers: Secure, avoidant, and anxious-ambivalent (Ainsworth, Blehar, Waters, & Wall, 1978). Attachment theorists proposed that these early experiences with parental acceptance and rejection create relatively stable mental models of self and others (Hazan & Shaver, 1987). The *model of self* provides a guide to the extent to which the self is worthy of affection, and the *model of other* informs the individual about the extent to which others can be expected to provide support. Anxious attachment (or a model of self as unworthy of love) in adulthood is marked by heightened anxiety in response to separation and conflict in close relationships, hypervigilance to negative cues from others, increased rejection sensitivity, and preoccupation with relationships in general, when compared with secure attachment (Feeney, 1999). Avoidant attachment (or a model of others as unreliable) is marked by discomfort with closeness to others, difficulty with intimacy, emotional unavailability, and a reluctance to rely on the assistance of close others (Feeney, 1999).

In order to identify possible physiological mechanisms underlying these attachment dimensions, we believe it may be useful to consider them in terms of social avoidance and approach motivations. This way, attachment theory could be brought into line with some of the more comprehensive theories of the base motivations that aid in behavior regulation across a wide range of mammalian species. In particular, Gray and McNaughton (2000) formulated a model of behavior regulation that argues that three main physiological systems interact to produce base motivations (see also McNaughton & Corr, 2004). The first is the fight-flight-freeze system (FFFS) that responds to aversive stimuli, producing higher degrees of *fear* as the stimuli become increasingly threatening and providing motivation for avoidance and escape. Importantly, research with nonhuman animals has shown the FFFS to be activated by factors such as physical pain and social defeat (Gray & McNaughton, 2000). The second is the behavioral approach system (BAS) which responds to appetitive stimuli, producing what Corr (2005) has termed *anticipatory pleasure*, and providing motivation for approach behavior. Finally, the behavioral inhibition system (BIS) serves mainly to resolve competing approach and avoidance motivations (however, the model suggests that the BIS might also serve to resolve approach-approach and avoid-avoid conflicts). The theory states that the

existence of such goal conflict produces *anxiety*, motivating an assessment of risk that Corr (2005) described as being 'experienced as worry and rumination, and a feeling of possible danger or loss' (p. 233).

Thus, the question becomes how the attachment dimensions match up with approach and avoidance motivations, and consequently the physiological systems described in Gray and McNaughton's (2000) model. In our view, the dimension of anxious attachment, or the model of self, appears to represent sensitivity to the key danger of close relationships – rejection. That is, anxious attachment represents perceptions of the degree of threat in a relationship or the *fear* that one will be hurt by a close other, and thus may be most strongly tied to the FFFS. On the other hand, avoidant attachment, or the model of other, appears to represent sensitivity to the key reward of close relationships – intimacy. That is, avoidant attachment represents perceptions of the degree to which one will be benefited by closeness or the *anticipatory pleasure* from interaction with a close other, and thus may be most strongly tied to the BAS. This line of reasoning becomes more clear when the attachment prototypes used in categorical measures of attachment are considered (e.g., Bartholomew & Horowitz, 1991). Secure individuals (positive model of self and others), who are comfortable with intimacy, would be expected to experience BAS activation in association with close relationships without competing FFFS activation, resulting in *anticipatory pleasure* in relation to close others. Anxious-ambivalent individuals (negative model of self, positive model of others), who desire intimacy but fear rejection, would be expected to experience both BAS and FFFS activation in association with close relationships, which would trigger BIS activity leading to *anxiety*, risk assessment, worry, and rumination. Dismissing avoidant individuals (positive model of self, negative model of others), who downplay the importance of relationships and value self-reliance, would be expected to experience little activation of either the BAS or FFFS in association with close relationships, resulting in low levels of fear but also low levels of motivation to pursue intimacy (i.e., indifference). Finally, fearful avoidant individuals (negative model of self, negative model of others), who have strong rejection concerns and are not emotionally expressive, would be expected to experience FFFS activation in association with close relationships without competing BAS activation resulting in *fear* in relation to close others.

This analysis suggests that physiological mechanisms underlying anxious attachment should be associated with the FFFS. Carver and White (1994) developed the BIS/BAS scales in an attempt to assess self-reports of chronic tendencies towards avoidance and approach, respectively. The BIS items center on reports of fear and anxiety, suggesting this scale may tap chronic FFFS activation, at least in part. The BIS scale has been shown to be strongly and positively related to anxious attachment, but unrelated to avoidant attachment (Meyer, Olivier, & Roth, 2005). The BAS items centre on reports of the anticipation of pleasure, and this scale (consisting of three subscales) has been shown to be negatively related to avoidant attachment, but unrelated to anxious attachment (Meyer et al., 2005). Although such

self-reports must be considered a distal measure of the activation of these physiological systems, these data suggest that anxious attachment is more strongly related to danger sensitivity than reward sensitivity.

Social pain theory (MacDonald & Leary, 2005) provides insight into at least one physiological mechanism that may contribute to the danger sensitivity of individuals high in anxious attachment. This theory argues that feelings of pain, which are known to activate the FFFS (Gray & McNaughton, 2000), may also serve as a warning signal of social exclusion. Thus, pain may be one physiological contributor to the dimension of anxious attachment. More specifically, physical pain consists of two separate components (Price, 2000). The first is pain sensation, or the information about physical trauma that is collected by the body's specialized pain receptors. The second is pain affect, or the feelings of unpleasantness that frequently accompany pain sensation. Social pain theory argues that perceptions of rejection trigger feelings of pain affect. The theory suggests that, over evolutionary history, animals who developed increasing interdependence required mechanisms to motivate social cohesion. Because new evolutionary developments often arise from previously existing physiological mechanisms, social regulation mechanisms would likely have developed from earlier behavior-regulation systems. Pain may have provided a particularly useful signal for regulating social behavior for at least two reasons. First, pain would have provided a sense of aversiveness that, when associated with rejection, would promote learning that would lead social animals to avoid conditions that foster such exclusion. Second, because of pain's links with the FFFS, feelings of pain would motivate timely behavior to escape or avoid the rejection threat, in much the same way that physical pain produces quick, defensive reactions.

Recent evidence argues for a link between social exclusion and feelings of pain. In a review, MacDonald and Leary (2005) showed that responses to both physical pain and social exclusion are mediated by several common brain and neuroendocrine mechanisms across social animals. These include the periaqueductal gray and anterior cingulate cortex (ACC) brain areas as well as the opioid and oxytocin neuroendocrine systems. For example, in a functional magnetic resonance imaging study, Eisenberger, Lieberman, and Williams (2003) demonstrated activation of the ACC in response to experimentally induced social exclusion that the authors described as highly similar to activation seen in response to physical pain. Indeed, experimentally induced social exclusion has been shown to lead to physiological responses similar to those caused by physical pain such as increased blood pressure and cortisol (Stroud, Tanofsky-Kraff, Wilfley, & Salovey, 2000), disruptions in higher-order cognitive function (Baumeister, Twenge, & Nuss, 2002), potentiation of the startle response (Downey, Mougios, Ayduk, London, & Shoda, 2004), and aggression (Twenge, Baumeister, Tice, & Stucke, 2001).

If one role of feelings of pain is to serve as a signal of social exclusion, then pain from any source has the potential to contribute to perceptions of rejection. That is, pain affect from physical injury that is experienced in

conjunction with social interaction may lead an individual to feel more rejected in that interaction. Individuals experiencing persistent pain, then, may begin to feel more chronically rejected as pain continually pervades their interactions, leading to more anxious attachment. In this way, persistent pain may lead to particularly high levels of emotional distress such as anxiety and depression. Not only would pain heighten negative emotionality by activating the FFFS directly, but it would also do so indirectly by promoting anxious attachment. That is, individuals living with persistent pain may experience increased negative emotionality, in part because their constant pain affect provides an unrelenting signal of social threat. Research has demonstrated that persistent pain is related to higher levels of both anxiety (Weisberg & Keefe, 1999) and depression (Fishbain, Cutler, Rosomoff, & Rosomoff, 1997). Further, anxiety and depression have been consistently linked with anxious, as well as avoidant, attachment (e.g., Mickelson, Kessler, & Shaver, 1997). Simpson and Rholes (2004), in a review of the attachment and depression literature, argued that anxious attachment is a stronger predictor of depression than avoidant attachment. Overall, these data are consistent with the suggestion that the link between persistent pain and negative emotionality may be partially mediated by anxious attachment.

However, because pain is more strongly linked to the FFFS than the BAS, persistent pain would not be expected to contribute to the dimension of avoidant attachment, or the model of other. Thus, we would not expect avoidant attachment to mediate the pain–distress link, despite the associations between avoidant attachment, anxiety, and depression. This may seem surprising, given the notion from attachment theory that stressors such as pain activate the attachment system to draw the individual closer to helpful others (Bowlby, 1973). In our view, the key distinction here is between acute and persistent pain. In the short term, pain may well promote social approach in an attempt to alleviate suffering. However, as pain persists and social approach does not bring relief, the perceived rewards of promoting closeness should decrease. That is, if others are not able to help with the pain then the value of approaching these others diminishes. Indeed, research has shown that those experiencing persistent pain become more introverted over time (Phillips & Gatchel, 2000), reporting increased social anxiety and avoidance of social situations (Sharp & Harvey, 2001).

To our knowledge, no research has directly examined links between reports of physical pain affect and the attachment dimensions. One study that did examine a pain–attachment link showed that chronic pain patients who were higher in anxious attachment experienced higher levels of emotional distress in response to their pain (Mikulincer & Florian, 1998). However, we propose that it may be more accurate to think of anxious attachment, at least in part, as a result of persistent pain. Thus, anxious attachment may play the role of mediator, rather than moderator, of the link between persistent pain and emotional distress. That is, pain sufferers may experience distress, in part because their pain leads to heightened

concern over rejection from close others. In the current study, we tested this hypothesis by asking individuals ranging in their experience of persistent physical pain (pain clinic patients and community members) to report on attachment and emotion. We hypothesized that reports of physical pain affect would predict both anxious attachment and emotional distress (i.e., anxiety and depression) but not avoidant attachment. Further, we expected that anxious attachment would partially mediate the relation between physical pain affect and emotional distress. That is, the experience of pain affect should lead to more anxiety and depression partly due to increased concerns over rejection.

Method

Participants

A total of 156 individuals participated in the current study (82 females, 73 males, 1 gender not listed). Sixty-four participants who were being treated for persistent pain were recruited from a multidisciplinary pain center located at the Royal Brisbane and Women's Hospital (34 females, 29 males, 1 gender not listed, mean age = 54.69 years). Some of these participants were recruited for the study prior to a regular group information session with a resident psychologist. Individuals expressing interest in participating were provided with a written information sheet and consent form. In addition, each individual was supplied with a questionnaire battery that was collected the following week. Pain center participants were also recruited via a mailing of an identical information sheet, consent form, and assessment battery to those who had received treatment during the preceding 6-month period. A further 92 respondents were recruited from a community shopping center (48 females, 44 males, mean age = 37.70 years). Community participants received identical verbal and written information as those approached from the multidisciplinary pain center. These participants were requested to return the completed questionnaire battery on the day they received it. No remuneration was offered in exchange for participation in either setting.

Procedure

All participants completed questionnaires that were collated in alternate forms to account for effects attributable to order.

Measures

Physical pain affect. Pain affect was measured via two items tapping tolerance for, and suffering as a result of, pain. First, participants were asked to rate the pain they felt that day on a 101-point scale (0 = 'no pain' to 100 = 'pain so severe you could not tolerate it for more than 1 minute'). Second, participants were asked to describe their current pain-related suffering on a 6-point scale (0 = 'no suffering' to 5 = 'intolerable suffering'). The two items were converted to z-scores, then averaged to form an overall pain affect score, Cronbach's $\alpha = .88$.

Attachment Style Questionnaire (ASQ). The ASQ (Feeney, Noller, & Hanrahan, 1994) is a 40-item fixed-choice questionnaire that measures the

fundamental dimensions underlying adult attachment. The first dimension, anxious attachment, is measured with 13 items that tap the extent to which people hold a negative model of self (e.g., 'I wonder why people would want to know me'), Cronbach's $\alpha = .83$. The second dimension, avoidant attachment, is measured with 16 items that tap the extent to which individuals hold a negative model of others (e.g., 'I prefer to depend on myself rather than other people'), Cronbach's $\alpha = .80$. Answers to ASQ items were given on a 6-point scale (1 = 'totally disagree' to 6 = 'totally agree').

Depression and anxiety. These constructs were measured via the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995) that measure subclinical and clinical levels of depression and anxiety. Participants were asked to indicate the extent to which 28 behavioral descriptors applied to them over the past week using a 4-point scale (0 = 'did not apply to me at all' to 3 = 'applied to me very much, or most of the time'). Statements pertaining to each scale are: 'I felt that life wasn't worthwhile' (depression scale), and 'I felt scared without any good reason' (anxiety scale). Scoring involves summing each scale to produce scores for depression, Cronbach's $\alpha = .96$, and anxiety, Cronbach's $\alpha = .91$.

Demographic information. A brief instrument for recording the demographic information of participants assessed age, gender, marital status (married vs. not married), and level of achieved education (below grade 10, grade 10–11, grade 12, tertiary – attempted, tertiary – completed).

Results

We conducted mediation analyses to test whether insecure attachment (i.e., avoidant and anxious attachment) would mediate any relation between pain affect and emotional distress (i.e., anxiety and depression). Analyses were conducted on the 135 cases with complete data. In order to be maximally certain that effects could be attributable to pain affect, a number of steps were taken. First, initial tests were conducted to assess any demographic differences between the two samples (i.e., pain center and community). These analyses revealed that the pain center sample was significantly older, $t(133) = 5.95, p < .001$, and had completed significantly fewer years of formal education, $t(133) = 5.81, p < .001$, than the community sample. Thus, age and education were controlled in all analyses. Further, to account for group differences not captured by our demographic measures (e.g., method of recruitment), recruitment group was dummy-coded (0 = community, 1 = pain center) and also controlled in all analyses. This may be considered a conservative analytic strategy as some of the variance from the pain affect variable should be accounted for by the recruitment group variable. However, this strategy allows for the most confidence that effects of the pain affect variable are attributable to feelings of pain and not other differences between the two groups. Finally, we accounted for any influence of gender by controlling for this variable as well (0 = male, 1 = female) (see Table 1 for zero-order correlations between all variables).

According to Baron and Kenny (1986), establishing a variable as a mediator between the relation of two other variables requires a series of regression

TABLE 1
Zero-order correlation matrix

	Pain	AXA	AVA	DEP	ANX	Group	Age	Sex
AXA	.184*							
AVA	.035	.389***						
DEP	.430***	.574***	.297***					
ANX	.375***	.469***	.112	.824***				
Group	.744***	.062	.102	.340***	.306***			
Age	.328***	-.214*	-.041	-.014	.029	.459***		
Sex	.027	.118	-.082	.019	.111	-.018	-.011	
EDU	-.418***	-.172*	-.035	-.282**	-.294**	-.450***	-.320***	.022

Note. AXA = anxious attachment; AVA = avoidant attachment; DEP = depression; ANX = anxiety; EDU = education.

* $p < .05$; ** $p < .01$; *** $p < .001$.

analyses. First, after accounting for the control variables by entering them simultaneously in the first step of the regression analysis, we must establish pain affect as significantly related to attachment. Second, again accounting for controls, we must establish pain affect as significantly related to the emotion in question. The third analysis tests whether attachment predicts the emotion in question independently of pain affect. The final analysis tests whether controlling for attachment significantly weakens the relation between pain affect and defensiveness. In order to test this final criterion, we employed Sobel's (1982) test to determine the statistical significance of the applied mediating variable (Baron & Kenny, 1986). In order to examine the unique effects of each attachment dimension, anxious attachment was controlled when examining avoidant attachment and vice-versa.

The only significant predictor of avoidant attachment was anxious attachment, $\beta = .422, p < .001$. Avoidant attachment was unrelated to recruitment group, age, gender, or education. Controlling for these factors, avoidant attachment was not significantly related to pain affect, $\beta = -.202, p = .100$. Thus, avoidant attachment could not mediate any relation between pain affect and emotional distress.

Anxious attachment was significantly predicted by all the control variables except recruitment group. Higher levels of anxious attachment were reported by participants who were older, $\beta = -.298, p = .001$, female, $\beta = .152, p = .046$, less formally educated, $\beta = -.231, p = .008$, and higher in avoidant attachment, $\beta = .375, p < .001$. Central to our hypotheses, higher levels of anxious attachment were significantly related to higher levels of pain affect, $\beta = .286, p = .012$.

Higher levels of anxiety were significantly related to being in the pain center sample, $\beta = .275, p = .006$, and lower levels of education, $\beta = -.223, p = .016$. Anxiety was marginally related to age, $\beta = -.163, p = .077$, and unrelated to gender and avoidant attachment. Controlling for these variables, higher levels of anxiety were related to higher levels of pain affect, $\beta = .288, p = .018$. Thus, we examined whether anxious attachment would mediate the relation between pain affect and anxiety. Controlling for pain affect, anxious attachment significantly predicted anxiety, $\beta = .428, p < .001$. Sobel's (1982) test indicated a partial correlation between pain affect and anxiety that was significantly smaller than the zero-order correlation, $z = 2.27, p = .023$. The relation between pain affect and anxiety was reduced to nonsignificance, $\beta = .165, p = .147$, suggesting that

anxious attachment fully mediated the relation between pain affect and anxiety (see Figure 1).

Depression was significantly related to all the control variables except gender. Higher levels of depression were reported by participants who were in the pain center sample, $\beta = .326, p = .001$, younger, $\beta = -.215, p = .015$, less formally educated, $\beta = -.196, p = .025$, and higher in avoidant attachment, $\beta = .252, p = .001$. Controlling for these variables, higher levels of depression were related to higher levels of pain affect, $\beta = .382, p = .001$. Thus, we examined whether anxious attachment would mediate the relation between pain affect and depression. Controlling for pain affect, anxious attachment significantly predicted depression, $\beta = .466, p < .001$. Sobel's (1982) test indicated a partial correlation between pain affect and depression that was significantly smaller than the zero-order correlation, $z = 2.35, p = .019$. However, the relation between pain affect and depression remained significant, $\beta = .249, p = .016$, suggesting that anxious attachment partially mediated the relation between pain affect and depression (see Figure 2).

FIGURE 1
Mediation analyses for anxiety.

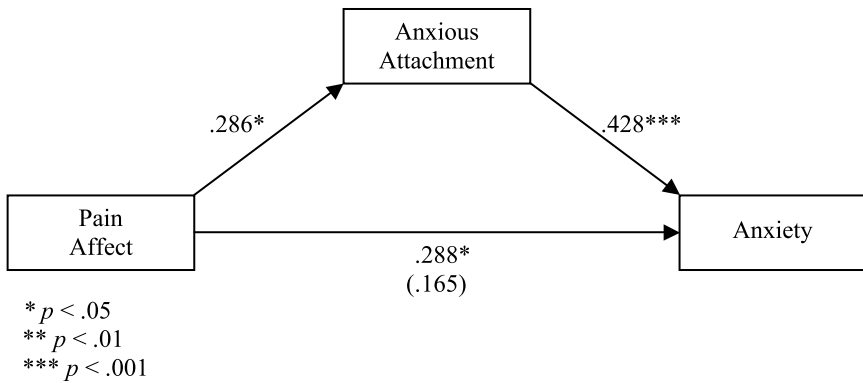
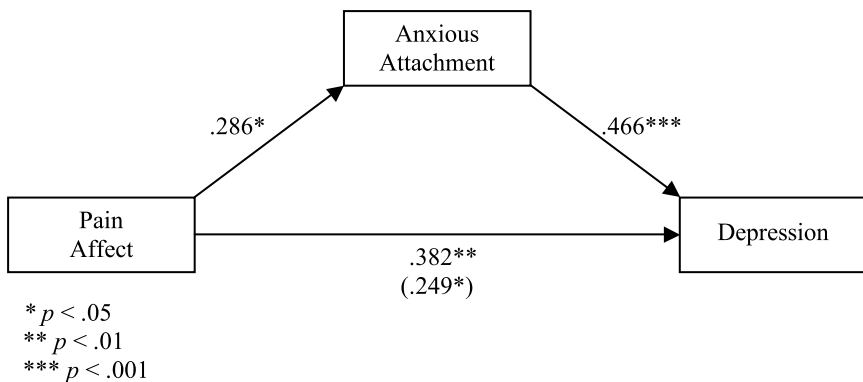


FIGURE 2
Mediation analyses for depression.



Discussion

The data supported the hypothesis that anxious attachment would partially mediate the relation between pain affect and emotional distress. First, as predicted, higher levels of pain affect were related to higher levels of anxious attachment. That is, individuals who reported more suffering from and less tolerance of physical pain also reported more concern over being rejected by others. This result is consistent with the notion that pain affect serves as a signal of social exclusion, so that when pain results from a physical injury one outcome may be increased perceptions of rejection. In turn, anxious attachment fully mediated the relation between pain affect and anxiety, and partially mediated the relation between pain affect and depression. Although full mediation was found in the case of anxiety, these data should not be taken to support anxious attachment as the sole cause of anxiety in response to pain. Part of our analytic strategy was the decision to control for recruitment group (pain center vs. community), and this variable accounted for some of the variance in pain affect. Supplementary analyses (available on request) revealed that, when not controlling for recruitment group, anxious attachment was a significant, partial mediator of anxiety. Indeed, the theoretical justification and research evidence for pain being directly tied to FFFS mechanisms (Gray & McNaughton, 2000) is too strong to suppose this relation can be explained solely in terms of rejection concerns. Thus, we believe the best interpretation of our data is that higher levels of pain affect were related to more anxiety and depression both directly and as a result of more anxious attachment.

Also as predicted, avoidant attachment was not significantly related to reports of pain affect. Avoidant attachment has been previously found to be unrelated to the BIS scale, but significantly related to the BAS scale (Meyer et al., 2005). The lack of association between avoidant attachment and pain affect (as well as the lack of a significant correlation with the BIS scale) argues against avoidant attachment as an FFFS mechanism. Instead, the association between avoidant attachment and the BAS suggests that avoidant attachment may involve the regulation of appetitive or approach motivation (promotion of closeness). On the other hand, the relations of anxious attachment with pain affect, as well as the BIS but not the BAS scale (Meyer et al., 2005), support the notion that anxious attachment involves the regulation of defensive or avoidance motivation (prevention of exclusion). By focusing on anxious attachment as a form of avoidance motivation, we narrow the range of physiological mechanisms that may underlie such fear of rejection to those associated with the FFFS. Our data suggest that pain affect may be one such mechanism. Indeed, some features of anxious attachment are strikingly similar to features of response to physical pain. For example, Shaver and Mikulincer (2002) describe anxiously attached individuals as chronically focused on negative emotions and unable to detach from psychological pain. Similarly, in a review of the physical pain and attention literature, Eccleston and Crombez (1999) argue that physical pain places strong demands on attention, particularly among

those who expect pain cues to be present. It seems reasonable to suggest that the strong focus on feelings of pain from interpersonal and physical sources among individuals who anxiously expect harm in these environments may be founded on a general sensitivity to danger, or higher FFFS activation.

The results of this study also provide insight into the nature of emotional response to physical pain. Our results suggest that anxiety and depression in response to persistent pain may represent, in part, a hypersensitivity to interpersonal devaluation. In turn, the heightened sense of social threat evoked by persistently high pain affect may lead to more defensive responding in social situations. One such defensive response is aggression. Aggression has been documented as occurring frequently among pain patients in healthcare settings (Fishbain, Cutler, Rosomoff, & Steele-Rosomoff, 2000). In 1992, the U.S. Bureau of Labor Statistics reported that healthcare recipients perpetrated 45% of nonfatal workplace assaults, and was 'the group most responsible for incidents of workplace violence' (cited in Fishbain et al., 2000, p. 141). Some authors suggest that aggression in health settings can be explained by frustration at delayed service provision, displeasure at the physicians' inability to cure, or dissatisfaction at being discharged (Fishbain et al., 2000). Although these factors offer some explanation of aggression in healthcare settings, they do not seem to account for the high prevalence of aggression in these settings as opposed to other public or community services that may provoke equivalent frustration. We suggest that the signal of interpersonal devaluation promoted by pain affect may partially account for the high incidence of aggression in healthcare settings. In particular, our results suggest that medical conditions that involve pain have the potential to increase perceptions of rejection, a known precursor of aggression (Twenge et al., 2001). Indeed, when the sort of frustrations with healthcare service noted earlier are combined with an increased tendency to interpret interpersonal cues as rejecting, emotional and physical hostility may be especially potentiated. More generally, consideration may need to be given to the extent to which emotional response to persistent pain is the result of a change in perceived social conditions, rather than a reaction to one's physical health *per se*.

Of course, some caution should be taken in interpreting the results. First, the correlational nature of the study limits the conclusions that can be drawn. Although we argue that the experience of pain increases anxious attachment, other causal interpretations could be argued including an unmeasured third variable. For example, individuals high in neuroticism or negative affectivity may be more likely to report higher levels of both pain affect and anxious attachment. Indeed, it may be helpful in future research to control for neuroticism to examine whether the relation between pain and anxious attachment is more than a function of general negative emotionality. However, even if the pain-anxious attachment link was attributable to neuroticism, this would still be consistent with the notion of anxious attachment as chiefly avoidance motivation. For example, neuroticism is strongly correlated with the BIS scale, and weakly, if at all, correlated

with the BAS scale (e.g., Gomez & Gomez, 2005). Thus, the link between pain and anxious attachment would still represent an expression of sensitivity to danger.

Another potential interpretation of the results is that higher levels of anxious attachment may cause increased perceptions of physical pain. In fact, such an interpretation would be entirely consistent with social pain theory, which argues that pain affect is a multipurpose mechanism, informing individuals about both physical and social injury. That is, if perceived rejection or devaluation by close others accompanies physical injury, social pain may heighten the experience of felt physical pain. Indeed, individuals who experience any type of persistent pain frequently feel misunderstood and invalidated by others who do not appreciate the full reality of their pain, including medical professionals (e.g., Chapman, 1991). Ultimately, then, the relation between physical pain and anxious attachment is likely to be more reciprocal than we have portrayed here. However, given that many participants in our study were experiencing identifiable physical ailments, it seemed wisest to interpret that data with physical pain as the starting point. In any event, ours is the first study to demonstrate a link between higher levels of pain affect and higher levels of anxious attachment. Future experimental and longitudinal research would help to clarify the causal links between these variables.

A further limitation of the current study is the extent to which the measures of depression and anxiety can be considered to be indicative of activity in the FFFS. Research has consistently supported the conclusion that both depression and anxiety are more strongly correlated with the BIS scale than the BAS scale (e.g., Campbell-Sills, Liverant, & Brown, 2004). Although items from the depression and anxiety scales generally represent negative emotionality, the specific items appear to be consistent with both emotional activity generated by the FFFS (i.e., fear) and the BIS (i.e., anxiety). Indeed, this mixture of fear and anxiety items is a problem with the BIS scale itself, and there is no scale that provides a pure measure of the FFFS. In a sense, BIS activity can be considered a marker of FFFS activity, given that the BIS is active in response to a combination of FFFS and BAS activity. Thus, a scale that combines fear and anxiety questions can still provide information about fear system activity. However, it is unlikely that any self-report scale will provide a clean indication of the activation of physiological defense. Thus, it seems important in future research to combine measures of self-reports of emotion with physiological indicators of defense system activation such as skin conductance (Dawson, Schell, & Filion, 1995).

As this study represents the first attempt to link the dimension of anxious attachment directly to perceptions of pain, it is far from the final word on the subject. Along with future research involving experimental and longitudinal designs, further investigation of the link between the attachment dimensions and basic defense systems, measured both with the BIS/BAS scales and physiological indicators, seems warranted. For example, it seems reasonable to ask whether other known activators of FFFS mechanisms

(e.g., high levels of carbon dioxide) would also relate to perceptions of rejection. Research strategies examining the link between activators of the BAS and the attachment dimensions could shed light on the basic motivational nature of avoidant attachment. The present data point most clearly to the role of anxious attachment in emotional response to physical pain, and suggest a future focus on the social threat implications of pain. In general, we propose that considering the links between the attachment dimensions and more basic approach and avoidance regulation systems has the potential to aid in understanding the physiological underpinnings of adult attachment.

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