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Emotion induction moderates effects of anger management style on acute pain sensitivity

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Abstract

Anger management style (AMS) is related to both acute and chronic pain intensity. Recent work suggests that an anger expressive AMS in particular may influence acute pain, and that this effect may be most pronounced during anger provocation. The present study examined whether AMS was related to subsequent pain sensitivity without regard to prior emotion induction, only when a strong negative emotion was evoked, or only when anger was provoked. Sixty-four healthy normals partook in semi-structured interviews in which they recalled and verbally described an event in which either anger, sadness, or joy was elicited. They then underwent a cold pressor pain task. Results of hierarchical multiple regressions showed that an anger expressive AMS was related *positively* to pain threshold only for participants in the anger-recall condition, and that this effect was largely accounted for by their *low* SBP reactivity during emotion induction. An anger suppressive AMS was related positively to increases in self-reported pain severity, irrespective of emotion-induction condition, and this effect was not accounted for by reactivity in any cardiovascular index. Results extend those of previous studies by illuminating the potential importance of behavioral anger expression for individuals prone to express anger in modulating their reactivity and pain sensitivity. Findings suggest that the detrimental effects of an anger expressive style on pain sensitivity may be ameliorated under conditions in which behavioral anger expression occurs. Results are discussed in terms of recent work suggesting that an expressive AMS is associated with endogenous opioid dysfunction in the absence of behavioral anger expression.

Keywords: Experimental pain; Anger management style; Emotion induction; Acute pain

1. Introduction

Dimensions of anger are related to chronic and acute pain intensity. Although high levels of trait anger and hostility appear to characterize patients with chronic pain disorders (Wade et al., 1990; Conant, 1998), much research has focused on the effects of anger management style (AMS). Both anger suppression (anger-in) and anger expression (anger-out) are correlated with chronic pain severity (Kerns et al., 1994; Bruehl et al., 2002), and anger-out, in particular, appears to influence sensitivity to acute pain induced in the laboratory (Gelkopf, 1997; Bruehl et al., 2002; Burns et al., in press).

Trait × situation models may best elucidate associations between AMS and pain sensitivity. Because the AMS construct describes how one regulates anger *when angered* (Spielberger et al., 1985), AMS effects should be most pronounced during such situations. This approach is supported by findings that anger-out consistently predicts cardiovascular reactivity when subjects are harassed, but seldom does so during general stressors (Siegman et al., 1992; Burns and Katkin, 1993). Indeed, Burns et al. (in press) found that anger-out correlated negatively with pain tolerance among subjects who performed mental arithmetic with harassment prior to pain induction, but these factors did not correlate significantly for subjects who underwent pain induction prior to mental arithmetic.

Burns et al. (in press), however, did not compare anger induction with manipulations designed to rouse other negative affects. Thus, they could not rule out the possibility that relationships between anger-out and pain sensitivity are magnified when *any* strong negative emotion is incited prior to pain induction. Findings suggest that induction of unpleasant moods increases pain sensitivity (Zelman et al.,

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1991; Meagher et al., 2001; Carter et al., 2002), but effects of anger induction and individual differences have not been addressed. The present study examined relationships between AMS and acute pain sensitivity when angry, sad or joyful emotions were evoked before pain induction. This approach allowed us to evaluate three competing models linking AMS with pain sensitivity: (a) whether AMS (anger-out, in particular) is related to pain sensitivity regardless of previous emotional arousal; (b) whether AMS is related to pain sensitivity when *any* strong negative affect is evoked; (c) whether AMS is related to pain only when anger *in particular* is aroused.

Research also addresses mechanisms by which AMS is related to pain intensity. Using an opioid-blockade paradigm, Bruehl et al. (2002) showed that high pain sensitivity shown by high anger-out subjects may be due in part to deficient opioid-mediated analgesia. Burns et al. (in press) found that DBP reactivity to harassment accounted for much of the variance linking anger-out and pain tolerance. Cardiovascular reactivity may be controlled by several neurohumoral pathways, including endogenous opioids (Morris et al., 1990; McCubbin et al., 1998; Drolet et al., 2001). Thus, consistent with Bruehl et al. (2002), harassment may have activated endogenous mechanisms that both buffered cardiovascular reactivity and reduced pain sensitivity among low anger-out participants, but not among those high in anger-out. Although the present study did not employ opioid blockade, support for the opioid-mediation hypothesis would be provided if blood pressure reactivity to the anger-induction condition partly accounted for relationships between anger-out and pain sensitivity.

2. Method

2.1. Participants

Sixty-four graduate and medical school students were recruited to participate, and were paid $30. Exclusion criteria were: (a) history of cold urticaria; (b) current cardiovascular disorder (e.g. hypertension; Raynaud's Disease); (c) current use of medications that affect cardiovascular function (e.g. beta-blockers); (d) pregnancy or a history of chronic pain (e.g. migraine); (e) inability to understand or speak English well enough to participate in interviews. Participants were instructed not to exercise vigorously (i.e. activity that involved fast walking, running, or weight-lifting that lasted 15 min or more) or consume caffeine 6 h prior to their appointments for the study. Thirty-two participants were women, and the mean age of the sample was 25.1 years (SD 3.4).

2.2. Design overview

Participants were assigned randomly to one of three conditions: anger ($n = 21$), sadness ($n = 22$) or joy ($n = 21$). There were 10 women in the Anger Condition, 11 in the Joy Condition, and 11 in the Sadness Condition. Participants experienced two tasks in a fixed order. First, they partook in semi-structured interviews in which they were asked to recall and describe aloud a recent event that evoked the emotion corresponding to their condition (i.e. an anger-provoking event for participants in the Anger Condition). Second, they underwent a cold pressor pain task in which they immersed a hand in icy water. Positive and negative affect were assessed before and after the interviews, and pain severity, threshold and tolerance were assessed during the cold pressor. Cardiovascular function was assessed continuously throughout baseline and interviews.

2.3. Apparatus

2.3.1. Cold pressor

The apparatus for the cold pressor consisted of a water-filled container separated into two compartments by a wire screen. A block of ice was kept on one side of the screen, while the other compartment contained only icy water. Participants placed their hands into this second compartment. The water was circulated with an aquarium pump to prevent it from warming near participants' hands. Water temperature was recorded before the cold pressor for each participant, and ranged from 0 to 2 °C.

2.3.2. Cardiovascular activity

Systolic and diastolic blood pressure (SBP; DBP), and heart rate (HR) were measured using a Dinamap blood pressure monitor (model #1846 SX; Johnson and Johnson Medical). A standard blood pressure cuff was placed over the brachial artery of participants' nondominant arm. Blood pressure and HR readings were obtained about every 60 s during baseline and the interviews.

2.4. Self-report measures

2.4.1. Pain severity

The McGill Pain Questionnaire – Short Form (MPQ; Melzack, 1987) was used to assess sensory (e.g. throbbing, shooting) and affective (e.g. sickening, fearful) dimensions of pain severity during the cold pressor. Participants completed the MPQ once after resting baseline (see below) and again immediately after the cold pressor (i.e. at tolerance).

2.4.2. Positive and negative affect

The Positive and Negative Affect Scale (PANAS; Watson et al., 1988) was used to tap emotional valence. The scale is designed to provide measures of two orthogonal dimensions of affect; namely, positive (PANAS-P) and negative (PANAS-N). Positive affect refers to the extent to which a person feels energetic and alert, whereas negative affect defines the degree to which a person feels distress and