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Anger regulation style, anger arousal and acute pain sensitivity: evidence for an endogenous opioid “triggering” model

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Abstract

Findings suggest that greater tendency to express anger is associated with greater sensitivity to acute pain via endogenous opioid system dysfunction, but past studies have not addressed the role of anger arousal. We used a 2×2 factorial design with Drug Condition (placebo or opioid blockade with naltrexone) crossed with Task Order (anger-induction/pain-induction or pain-induction/anger-induction), and with continuous Anger-out Subscale scores. Drug $\times$ Task Order $\times$ Anger-out Subscale interactions were tested for pain intensity during a 4-min ischemic pain task performed by 146 healthy people. A significant Drug $\times$ Task Order $\times$ Anger-out Subscale interaction was dissected to reveal different patterns of pain intensity changes during the pain task for high anger-out participants who underwent pain-induction prior to anger-induction compared to those high in anger-out in the opposite order. Namely, when angered prior to pain, high anger-out participants appeared to exhibit low pain intensity under placebo that was not shown by high anger-out participants who received naltrexone. Results hint that people with a pronounced tendency to express anger may suffer from inadequate opioid function under simple pain-induction, but may experience analgesic benefit to some extent from the opioid triggering properties of strong anger arousal.

Keywords

Trait anger-out; Anger-induction; Pain-induction; Opioid blockade

Introduction

Anger is related to both acute (Bruehl, Burns, Chung, Ward, & Johnson, 2002; Burns, Bruehl & Caceres, 2004; Burns, Kubilus & Bruehl, 2003; Janssen, Spinhoven, & Brosschot, 2001) and chronic pain intensity (Bruehl, Burns, Chung, & Quartana, 2008; Burns, Johnson, Mahoney, Devine, & Pawl, 1996; Kerns, Rosenberg, & Jacob, 1994; Wade, Price, Hamer, Schwartz, & Hart, 1990). Findings suggest that the manner in which anger is regulated—for

example, expression (anger-out) or suppression (anger-in)—is consistently related to chronic pain severity (Bruehl et al., 2002; Bruehl, Chung, Burns, & Biridepalli, 2003; Burns, Holly, Quartana, Wolff, & Bruehl, 2008; Burns et al., 1996; Burns, Quartana, & Bruehl, 2011). We have proposed that anger regulation style may be related to acute and chronic pain through definable physiological mechanisms, such as stress- or anger-induced muscle tension increases near the site of injury (Burns et al., 2008; Burns, Quartana, Gilliam, Matsuura, Nappi, & Wolfe, 2012), and deficits in endogenous opioid analgesic function (Bruehl et al., 2002, 2003).

Several studies suggest that greater propensity to express anger in particular may be associated with greater sensitivity to acute pain via endogenous opioid system dysfunction (Bruehl et al. 2002, 2003; Bruehl, Chung, Burns, & Diedrich, 2007). In a laboratory study using an opioid blockade paradigm, we found that individuals high in trait anger-out had greater acute pain sensitivity when in the placebo condition than those low in anger-out. Under opioid blockade (naloxone), participants low in anger-out exhibited significantly increased pain sensitivity compared to placebo, whereas high anger-out participants displayed no significant changes in pain sensitivity compared to placebo (Bruehl et al., 2002). Results suggest that effective endogenous opioid analgesia was manifested by low anger-out people, with those high on trait anger-out in contrast displaying evidence of deficient opioid-mediated analgesia. Furthermore, differences in endogenous opioid analgesia during a laboratory procedure were found to mediate the relationship between trait anger-out and daily chronic pain severity among chronic pain patients (Bruehl et al., 2003). Finally, in a study using a different methodology to assess endogenous opioid activity, we found that the relationship between trait anger-out and acute pain sensitivity was mediated in part by pain-induced changes in plasma beta-endorphin (Bruehl et al., 2007). Across these three studies, endogenous opioid effects were almost exclusively confined to the link between trait anger-out and pain, with largely nonsignificant effects emerging for trait anger-in.

Results of this program of research strongly suggest that the dispositional tendency to express anger physically or verbally may be related to acute and chronic pain intensity through differences in endogenous opioid function. We characterize these results as supporting a “trait model” because the link between anger-out and opioids appeared to emerge without explicit provocation of anger. What we have not evaluated so far in our studies involving trait anger management style, pain-induction, and opioid blockade is whether *the acute arousal of anger*—through interpersonal provocation—affects the relationships among these three factors. This latter “trait $\times$ situation” model stands in contrast to the trait model above.

For conceptual reasons, “trait $\times$ situation” models may be necessary to understand fully the effects of anger regulation style on a variety of dependent variables. Unlike the simple propensity to become angry, the concept of anger regulation style reflects how one handles anger when anger has been provoked (Spielberger, Johnson, Russell, Crane, Jacobs, Worden, 1985). Therefore, the effects of anger regulation style on a host of responses should be most prominent during situations in which anger is actually elicited. This trait $\times$ situation approach is supported by findings that anger regulation style is consistently predictive of

cardiovascular reactivity during situations featuring interpersonal harassment or provocation, but less commonly in the absence of such anger provocation (Burns & Katkin, 1993; Suarez, Kuhn, Schanberg, Williams, & Zimmerman, 1998).

In our prior work, we identified two possible pathways by which anger arousal could influence connections among trait anger-out, endogenous opioids and pain. In Burns et al. (2004), participants underwent either mental arithmetic with harassment followed by a cold pressor task, or underwent these tasks in reverse order. Greater trait anger-out was associated with significantly higher pain sensitivity only among participants who underwent harassment prior to pain-induction, suggesting that a more robust *hyperalgesic* effect of elevated anger-out on pain sensitivity emerged when pain was experienced following acute anger arousal. High trait anger-out people may exhibit even greater pain sensitivity when angered prior to pain-induction because of enhanced pain perception associated with simultaneous experience of strong negative affect (anger) in the absence of effective compensatory opioidergic pain modulation. These laboratory effects are supported by results from an ecological momentary assessment diary study of chronic pain patients (Bruehl, Liu, Burns, Chont, & Jamison, 2012). Here, increased momentary behavioral anger expression when the person was angered appeared to predict increased chronic pain intensity in the subsequent 4 h period. Thus, the combined effect of negative emotion and pain in the absence of optimally functioning endogenous opioid inhibitory systems may effectively magnify pain sensitivity.

In another study (Burns et al., 2003), participants were assigned to conditions in which they recalled and verbally described events that elicited anger, sadness, or joy, and then underwent a cold pressor task. High anger-out participants in the anger recall condition showed *lower* pain sensitivity during subsequent pain-induction than high anger-out participants in the other emotion recall conditions, and lower sensitivity than low anger-out participants also in the anger recall condition. These results suggest that when high anger-out individuals experience anger (in this case, by recalling an anger-inducing event), they may exhibit subsequently *diminished* pain sensitivity. One speculation regarding these findings is that the arousal of anger may provide a trigger for high anger-out people to release endogenous opioids. Whereas pain-induction by itself may not be a sufficient stimulus to trigger activation of opioid inhibitory circuits, as it appears to be for people low in anger-out, a stimulus eliciting a strong negative emotional response may be required for high anger-out individuals to exhibit optimal endogenous opioid inhibitory function.

The studies above suggesting contrasting “anger magnifies pain” and “opioid triggering” models both failed to directly evaluate involvement of endogenous opioid mechanisms in the observed findings. To address whether anger arousal is a key factor in determining links among trait anger-out, opioid dysfunction, and pain sensitivity, the present study used a pharmacological opioid blockade procedure and varied the order in which anger and pain were induced. Thus, we used a 2×2 between-subject factorial design with Drug Condition (placebo or opioid blockade with naltrexone) crossed with Task Order (anger-induction/pain-induction or pain-induction/anger-induction). Scores on the Anger-out Subscale of the Anger Expression Inventory (Spielberger et al., 1985)—as well as the Anger-in Subscale and negative affect control variables (see below)—were used as continuous variables in

analyses. The present study is based on data collected for a previous study (Burns, Bruehl, Chung, Magid, Chont, Goodlad, Gilliam, Matsuura, & Somar, 2009), in which only the effects of Drug $\times$ Task Order were examined for pain ratings recorded *at the end* of the pain stimulus. Here, we examined the degree to which anger regulation style, anger arousal, and endogenous opioids exerted effects on *changes* in pain severity (i.e., adaptation over time) during an ongoing pain stimulus.

In general, if relationships between trait anger-out and pain sensitivity depend on whether placebo or naltrexone was administered *and* on whether the individual is acutely angered, then we would expect a series of Drug $\times$ Task Order $\times$ trait anger-out interactions. For people who undergo pain-induction prior to anger-induction, pain will be unaffected by anger arousal, and so we would expect the same pattern of responses we reported in Bruehl et al. (2002). That is, higher anger-out scores will correspond with significantly higher pain ratings under placebo, but with these effects reduced or eliminated under opioid blockade because low anger out participants (who normally exhibit well-functioning opioid analgesic systems) will report similar pain intensity to high anger-out participants due to pharmacologically-blocked endogenous opioid function.

For participants who undergo anger prior to pain, we would expect dramatically different effects. If the “anger magnifies pain” hypothesis is valid, then we would expect high trait anger-out people who experience pain following anger-induction to show greater pain sensitivity than high anger-out participants who undergo pain induction first (as in Bruehl et al., in press; Burns et al., 2004), regardless of whether they received placebo or opioid blockade. In such a case, undergoing acute pain induction after becoming angered would make high anger-out people even more vulnerable to pain—perhaps due to anger-related increases in pain perception with inadequate compensatory endogenous opioid inhibition—than would pain-induction alone. In contrast, if the “opioid triggering” hypothesis is valid, then we would expect high trait anger-out participants who undergo anger-induction prior to pain *and receive placebo* to show greatly reduced pain sensitivity relative to low anger-out participants in the same set of conditions (as in Burns et al., 2003). That is, we would expect a nonsignificant or even a negative association between trait anger-out scores and pain sensitivity. Such findings would suggest that anger arousal elicits opioid release for people high in anger-out, and affects subsequent pain intensity; an effect that may diminish over time as the initial anger-evoked opioid release abates. If this effect is indeed opioid-mediated, then for people who undergo anger-induction followed by pain but *under opioid blockade*, the analgesia triggered by anger would be blocked, leaving only the detrimental hyperalgesic effects of anger arousal on pain. We would thus expect a significant positive association between trait anger-out scores and pain sensitivity in this case.

In sum, trait anger-out may interact with acute anger arousal to influence pain sensitivity in part via endogenous opioid function, but these interactive effects could appear in at least two forms. Anger arousal may magnify subsequent pain sensitivity among individuals high in anger-out due to pain enhancing perceptual effects of anger without adequate compensatory endogenous opioid inhibitory function (Bruehl et al., 2012; Burns et al., 2004). Alternatively, anger arousal may trigger opioid release selectively among high anger-out people (Burns et al., 2003). Such an effect would be supported if: (a) the relationship

between trait anger-out scores and pain sensitivity for participants receiving placebo was substantially different when anger preceded pain (inverse) compared to when pain preceded anger (positive); (b) the inverse association between anger-out and pain sensitivity following anger arousal was eliminated or even reversed when participants received naltrexone rather than placebo.

Method

Participants

Participants were 146 healthy normal volunteers (women = 67; 46 %) recruited through posted flyers, media advertisements, and e-mail announcements. Participants were paid \$75 for their participation. Exclusion criteria were: (a) history of cardiovascular disorder; (b) history of renal or hepatic disease; (c) current use of medications that affect cardio-vascular function (e.g., beta blockers); (d) history of chronic pain (e.g., low back pain, frequent headaches); (e) current alcohol or substance abuse problems; (f) a history of psychotic or bipolar disorders, or posttraumatic stress disorder; (g) a current diagnosis of major depression; and (h) pregnancy (as determined by pregnancy test conducted by investigators on all female potential participants).

This sample was comprised of 146 of the 198 individuals who participated in the larger study (described in Burns et al., 2009). Given the focus of the current study on adaptation to pain over the duration of the ischemic task, the current sample included all participants who tolerated the ischemic pain task for at least 4 min. The 4 min duration was intended to provide a sufficiently lengthy and standard exposure to the ischemic pain to increase the probability of producing detectable between-subjects factor $\times$ within-subject period effects. The cell sizes were constituted thus: Blockade + anger-induction/pain-induction = 36; Blockade + pain-induction/anger-induction = 37; Placebo + anger-induction/pain-induction = 35; Placebo + pain-induction/anger-induction = 38. The cell frequencies were not significantly different [χ^2 (1; $N = 146$) = .03; ns]. The mean age of the sample was 26.9 years ($SD = 5.3$). The sample was comprised of 5.5 % Hispanic or Latino ($n = 8$), 10.3 % Asian ($n = 15$), 6.8 % African American ($n = 10$), and 77.4 % White ($n = 113$) participants.

Design overview

A double-blind, placebo-controlled, between-subjects opioid blockade design was used. A between-subjects design rather than a within-subjects design was used due to the necessity of debriefing participants immediately following the experimental session regarding the deception involved in the harassment-based anger induction procedure (see below). This debriefing would render the anger-induction manipulation invalid upon repetition in a within-subject design. Participants were assigned randomly to one of two Drug conditions (placebo or naltrexone), and both participants and the experimenters were blind to participants' Drug condition. Participants were also assigned randomly to one of two Task Orders of a computer maze task (anger-induction through harassment) and a forearm ischemia task (pain-induction). They performed either the computer maze first and then underwent the forearm ischemia (anger-induction/pain-induction), or they underwent the forearm ischemia first and then performed the computer maze task (pain-induction/anger-

induction). Self-report measures of pain intensity were collected every 30 s during the forearm ischemia task.

Materials

Opioid antagonist—Naltrexone hydrochloride (Mallinckrodt Pharmaceuticals, Inc.) was given in the standard oral therapeutic dose (50 mg) which achieves peak blood concentrations within 60 min (Product Information, Mallinckrodt Pharmaceuticals, Inc.). Naltrexone is a nonselective opioid receptor antagonist that temporarily blocks endogenous opioid activity at all three major classes of opioid receptors. The mean elimination half-life for naltrexone and its major active metabolite (6-beta-naltrexol) are 4 and 13 h respectively, and the standard dose has been shown to block the effects of intravenously administered heroin for up to 24 h (Product Information, Mallinckrodt Pharmaceuticals, Inc.). To maintain blinding, both naltrexone and the placebo were placed in identical capsules prepared by the Vanderbilt University Investigational Pharmacy. All participants rested quietly for 60 min following administration of the drug to allow peak blockade activity to be achieved before undergoing the anger-induction and pain tasks. Given the half-life of naltrexone, it is unlikely that significant escape from opioid blockade occurred during the anger- and pain-induction procedures of the experimental session.

Hand dynamometer—Pain-induction was accomplished through an ischemic pain task (see below). This task used a hand dynamometer in order to exercise dominant forearm muscles prior to inflation of a standard manual sphygmomanometer cuff to create temporary forearm ischemia.

Questionnaires

Anger regulation style—Dispositional tendencies to express and inhibit anger were assessed with the Anger Expression Inventory (Spielberger et al., 1985). This inventory provides scales to measure anger expressive style (Anger-out Scale) and anger inhibition style (Anger-in Scale) for which Spielberger et al. (1985) report adequate internal consistency coefficients. Further, Faber and Burns (1996) found that Anger-out Scale scores predicted the degree to which anger was expressed verbally during provocation.

General negative affect—Anger regulation style may share variance with more general tendencies to experience negative affect. Any associations between anger regulation scales and criteria, therefore, may be due less to the unique influences of the anger-out and/or anger-in constructs, and more to a common substrate of the frequency with which, and magnitude to which people become anxious, depressed or angry. Trait anxiety was measured with the trait items of the Spielberger Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970). The Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) was used to assess current depressive symptoms. It is a commonly used self-report measure which has well-established psychometric properties. In the present sample, scores on the Trait Anxiety Inventory and BDI were correlated $r = .47$ ($p < .01$). The tendency to become angry was tapped with the trait section of the State-Trait Anger Scale (Trait Anger Scale; Spielberger, Jacobs, Russell, & Crane, 1983). High scorers describe

themselves as “hot-headed” and quick-tempered. This scale has been shown to have excellent psychometric characteristics.

General emotional expressiveness—Anger management style may also share variance with more general tendencies to *express* negative affect. Any associations between anger regulation scales and pain intensity may be due less to the unique influences of the anger-out and/or anger-in constructs, and more to a common substrate of the way in which people typically regulate all negative emotions. Thus, the Emotion Expressivity Scale (Kring, Smith, & Neale, 1994) was used to tap emotion regulation, broadly defined. This 17-item scale is arousal and valence nonspecific. Items include, “I do not express my emotions to other people” (reverse scored) and “I cannot always hide the way I’m feeling.” Participants rated the extent to which each item applied to them on a scale from 1 (never) to 6 (always). Possible scores range from 17 (low expressivity) to 102 (high expressivity). The Emotion Expressivity Scale is well-validated, with Cronbach’s $\alpha = .91$, and test–retest reliability $r = .90$ over a 1 month period (Kring et al., 1994).

Pain intensity ratings—An eleven-point Numeric Rating Scale (NRS; Jensen & Karoly, 1992) tapped pain intensity (0 = “None” and 10 = “Most Severe Possible”), and was completed every 30 s during the forearm ischemia task.

Anger-induction task

Anger was induced with a procedure in which participants were required to take instruction from an antagonistic confederate during performance of a computer task. It was portrayed as a collaborative task for two people (the participant and a trained study confederate presented as another study participant). The task was described as involving the participant using the computer mouse to move a computer icon as quickly as possible and with as few errors as possible from the entry to the exit of a “complex computer-generated maze.” The participant operating the mouse was unable to see the maze on the screen and had to perform the task based solely on guidance provided by the other study “participant” (the confederate) who was able to view the computer screen.

The confederate assumed an unfriendly attitude from the outset. He or she followed a semi-standardized script that included instructions to move the cursor in certain directions, exclamations about errors, derogatory comments about the participant’s ability, and comments indicating that the confederate blamed the participant for all mistakes. Trained male and female university students served as confederates. To avoid confounds involving participant-confederate gender matches, approximately equal numbers of same sex, male participant-female confederate, and female participant-male confederate matches were used. This task and the harassment manipulation were adopted from previous work (Engelbreton, Matthews & Scheier, 1989), and we have used it successfully as well (Burns et al., 2012). The maze task was 5-min in duration.

Pain-induction

Pain was induced with an ischemic pain procedure (Maurset, Skoglung, Hustveit, Klepstad, & Oye, 1992). Participants were first asked to raise their dominant forearm over their head

for 30 s followed by two min of dominant forearm muscle exercise using a hand dynamometer at 50 % of his or her maximal grip strength (as determined prior to beginning the laboratory procedures). Immediately following this, a blood pressure cuff was inflated on the participant's dominant bicep to 200 mmHg. The cuff remained inflated until subjects indicated that their maximal pain tolerance had been reached (up to a maximum of 5 min).

Procedure

Participants were screened for exclusion criteria and asked not to consume caffeine for 3 h prior to their appointments, nor use analgesics or medications potentially affecting blood pressure (e.g., pseudoephedrine) for 12 h prior to their appointments. When they arrived at the laboratory, the equipment, procedures, and function (and risks) of naltrexone were explained, and maximum grip strength was determined. Informed consent was obtained. A "cover story" for the true purpose of the study involved telling participants that the maze task would be used to assess the effects of "stress during cooperation" on pain responses, and so the maze task would be completed in conjunction with another research participant. Participants were told that they would be assigned by flip of a coin to serve either as the "guide" or the "runner." The guide would direct the other person's efforts to negotiate the maze, whereas the runner would operate the computer mouse. Participants always served as the runner. They were told that the maze task would be timed, and that their performance would be evaluated based upon how quickly the maze was completed and how many errors were made. Participants were told that they would switch roles, and perform the task again after a short rest period.

Participants were seated in a comfortable chair in an upright position throughout all experimental procedures. A 10-min resting adaptation period commenced, after which participants received the appropriate randomized drug (placebo or naltrexone), and then rested quietly for 60-min to allow peak blockade activity to be achieved.

Next, for participants in the Maze/Forearm Ischemia Task Order condition, the confederate entered the room and sat 2 m from the participant on the opposite side of a computer table. They were told not to speak. Instructions for both tasks were given, but the instructions for the maze task were emphasized and directed to both the confederate and participant. The maze task then began. After the task, the confederate left the room, and instructions for the ischemic task were then briefly reiterated and it began approximately 90 s after the computer maze was completed. During the ischemic task, participants provided pain ratings aloud every 30 s.

For participants in the Forearm Ischemia/Maze Task Order, after the 60-min drug absorption period, instructions for both tasks were given, but the instructions for the ischemic task were emphasized. The ischemic pain task was then conducted, with participants giving pain ratings aloud every 30 s. The confederate then entered the room, and instructions for the maze task were given and directed to both the confederate and participant. The maze task then began. After the task, the confederate left the room.

After completion of both tasks, participants were thoroughly debriefed (especially with regard to the computer maze and harassment), asked whether they had believed the "other

subject” was part of the study, and information regarding possible side effects of naltrexone were reviewed. They were given a “side effects” checklist, which listed 10 physical symptoms occasionally experienced by people taking naltrexone (e.g., nausea, sweating). They indicated the degree to which they were experiencing each symptom on a 1 (not at all) to 10 (the most possible) scale. Participants were kept under observation until 3 h post-drug administration to monitor for additional side effects, and were discharged after this time unless side effects required further monitoring.

Data reduction and analysis

Our primary goal was to test whether individual differences in anger-out and anger-in moderated Drug and/or Task Order effects on changes in pain intensity during the forearm ischemia task. With Anger-out Scale scores as an example, General Linear Model procedures were used to test Drug (placebo, naltrexone) $\times$ Task Order (anger-induction/pain-induction; pain-induction/anger-induction) $\times$ Anger-out Scale scores (continuous) $\times$ Period (30-, 60-, 90-, 120-, 150-, 180-, 210-, 240-s) effects on pain intensity. Significant interactions for pain intensity involving Anger-out Scale scores were pursued by testing simple interactions with the goal of isolating simple correlations between Anger-out Scale values and pain ratings at various periods. Supplementary analyses were used to further illuminate relevant effects. For example, median splits of the Anger-out Scale were used to compare high anger-out participants on pain intensity under differing conditions. General Linear Model analyses were repeated for the Anger-in Scale. Analyses were then re-run controlling for the negative affect scale scores and Emotion Expressivity Scale scores to determine whether observed effects in primary analyses were attributable to general negative affect or emotional expressivity rather than anger regulation style per se.

Results

Comparisons of Drug $\times$ Task order conditions on demographic and trait factors

The 4 groups formed by crossing Drug with Task Order were compared on demographic and trait measures. Results indicated that the groups did not differ significantly on number of men and women [for Drug groups: X^2 (df 1, $N = 146$) = .247; $p > .10$; for Task Order groups: X^2 (df 1, $N = 146$) = .718; $p > .10$], or on age of participants, Anger-out Scale, Anger-in Scale, Emotion Expressivity Scale, Beck Depression Inventory, Trait Anxiety Inventory or Trait Anger Scale scores [F 's(1,142) < 2.08 ; p 's $> .10$].

Relationships among trait factors

Correlation coefficients among Anger-out Scale, Anger-in Scale, Emotion Expressivity Scale, Beck Depression Inventory, Trait Anxiety Inventory and Trait Anger Scale scores were generated. Results indicated that there were a number of significant relationships between the trait anger regulation scales and the other factors (see Table 1). Of note, Anger-out Scale and Anger-in Scale scores showed a small but significant correlation ($r = .17$; $p < .05$), suggesting that these constructs describing different anger regulation habits do indeed represent near-orthogonal dimensions. In contrast, Anger-out Scale and Trait Anger Scale scores showed a large significant correlation ($r = .64$; $p < .01$), suggesting that these constructs describe very similar anger-related dimensions.

Drug × Task Order × Anger-out Scale × Period Effects

The Drug (placebo, naltrexone) × Task Order (anger-induction/pain-induction, pain-induction/anger-induction) × Anger-out Scale (continuous) × Period (30-, 60-, 90-, 120-, 150-, 180-, 210-, 240-s) interaction was significant [$F(7,966) = 2.11$; $p < .05$, $\eta^2 = .015$]. This complex interaction was first dissected by testing Drug × Anger-out Scale × Period simple interactions for each Task Order.

Pain/Anger Task Order

For the pain-induction/anger-induction order, the Drug × Anger-out Scale × Period was significant [$F(7,497) = 5.43$; $p < .01$, $\eta^2 = .071$]. This interaction was due to a significant Anger-out Scale × Period interaction for participants in the placebo condition [$F(7,252) = 6.71$; $p < .05$, $\eta^2 = .157$] that was nonsignificant [$F < 1$] for participants in the naltrexone condition.

To illustrate the source of these effects for the Pain/Anger Task Order, regression equations with Anger-out Scale scores regressed on pain severity values at each time point were solved for hypothetical Anger-out Scale values (+1 SD above the mean, and −1 SD below the mean). The resulting predicted pain severity values were graphed for each of the Drug conditions (see Fig. 1). Correlation coefficients were generated between Anger-out Scale scores and pain intensity values at each time point separately for each condition (see Table 2). Finally, to fully address the issue of whether participants low in anger-out show evidence of optimal endogenous opioid function, as we have shown previously (Bruehl et al., 2002), we derived median split subgroups based on Anger-out Scale scores. We then compared the pain intensity ratings of low anger-out participants in the Placebo condition with low anger-out participants in the Naltrexone condition. Results showed an overall main effect for Drug collapsed across all 8 pain ratings [$F(1,33) = 3.92$; $p < .05$; $\eta^2 = .114$], revealing that low anger-out participants in the Naltrexone condition recorded greater pain intensity ($M = 37.99$; $SD = 20.1$) than those in the Placebo condition ($M = 26.10$; $SD = 15.4$).

Taken together, the graphical depiction of values in Fig. 1, correlations between Anger-out scores and pain intensity, and the median split ANOVA indicate that low anger-out participants receiving placebo reported the lowest pain intensity; an effect nullified by administration of naltrexone suggesting that low anger-out people derive significant opioid-mediated analgesia during acute pain. Further, high anger-out participants reported similar levels of pain intensity irrespective of receiving placebo or naltrexone, suggesting deficient opioid-mediated analgesia.

Anger/Pain Task Order

For the anger-induction/pain-induction Task Order, the Drug × Anger-out Scale × Period was significant [$F(7,469) = 2.88$; $p < .01$, $\eta^2 = .041$]. Further tests revealed that the Anger-out Scale × Period interaction was significant for participants in the placebo condition [$F(7,231) = 14.80$; $p < .01$, $\eta^2 = .310$], whereas this effect was nonsignificant [$F(7,238) = 1.14$; $p > .10$, $\eta^2 = .032$] for participants in the naltrexone condition.

Again, regression equations with Anger-out Scale scores regressed on pain severity values at each time point were solved for hypothetical Anger-out Scale values (+1 SD above the mean, and -1 SD below the mean). The resulting predicted pain severity values for each condition appear in Fig. 2. In addition, correlation coefficients were generated between Anger-out Scale scores and pain intensity values at each time point separately for each condition (see Table 2). Finally, to fully address the issue of whether participants high in anger-out show evidence of endogenous opioid triggering during and following anger, we used the median split categories based on the Anger-out Scale. We compared the pain intensity ratings of high anger-out participants in the Placebo condition to those of high anger-out participants in the Naltrexone condition. Results showed an overall main effect for Drug collapsed across all 8 pain ratings [$F(1,33) = 13.03$; $p < .01$; $\eta^2 = .271$], revealing that high anger-out participants in the Naltrexone condition recorded greater pain intensity ($M = 49.71$; $SD = 19.7$) than those in the Placebo condition ($M = 30.63$; $SD = 10.9$).

Taken together, the graphical depiction of values in Fig. 2, correlations between Anger-out scores and pain intensity, and the median split ANOVA indicate that high anger-out participants receiving placebo reported relatively low pain intensity; an effect nullified by administration of naltrexone suggesting that high anger-out people derive significant opioid-mediated analgesia when anger arousal precedes acute pain.

Summary

Results suggest different patterns of effects for the two Task Orders. For participants who underwent pain-induction *prior to* harassment, associations between Anger-out Scale scores and pain intensity replicate past findings, and suggest that in the absence of acute anger arousal, high anger-out people display hyperalgesia relative to low anger-out people. For participants who underwent pain-induction following harassment and received placebo, results suggest that high anger-out people experienced the provocation in a way that virtually reversed the effect of anger-out on pain severity shown by those undergoing pain first (in absence of anger arousal). Most importantly, results further suggest that the harassment-induced analgesia shown by high anger-out participants appears to have been nullified by opioid blockade, suggesting that it was due to anger-related release of endogenous opioids.

Analyses with negative affect and general emotion expressiveness controlled

The Drug $\times$ Task Order $\times$ Anger-out Scale (continuous) $\times$ Period analyses significant above were then re-run simultaneously controlling for Emotion Expressivity Scale, Beck Depression Inventory, Trait Anxiety Inventory and Trait Anger Scale scores. The 4-way interaction remained significant [$F(7,938) = 2.01$; $p < .05$, $\eta^2 = .015$].

Drug $\times$ Task Order $\times$ Anger-in Scale $\times$ Period Effects

The Drug $\times$ Task Order $\times$ Anger-in Scale (continuous) $\times$ Period interaction was nonsignificant [$F(7,966) = 1.26$; $p > .10$, $\eta^2 = .001$]. The 3-way Drug $\times$ Anger-in Scale $\times$ Period and the Task Order $\times$ Anger-in $\times$ Period interactions were also nonsignificant [F 's < 1]. However, the Anger-in Scale $\times$ Period interaction was significant [$F(7,966) = 4.06$; $p < .01$, $\eta^2 = .029$]. To better illustrate this effect, regression equations with Anger-in Scale

scores regressed on pain severity values at each time point were solved for hypothetical Anger-in Scale values (+1 SD above the mean, and -1 SD below the mean) collapsed across Drug and Task Order conditions. The resulting predicted pain severity values appear in Fig. 3. In addition, correlation coefficients were generated between Anger-in Scale scores and pain intensity values at each time point (see Table 3).

Results imply that the source of the Anger-in Scale $\times$ Period effect was a small change in the direction of the relationship between anger-in and pain intensity over time—a negative association at first followed by a positive association later—irrespective of Drug and/or Task Order conditions.

The Anger-in Scale (continuous) $\times$ Period General Linear Model analysis was re-run simultaneously controlling for Emotion Expressivity Scale, Beck Depression Inventory, Trait Anxiety Inventory and Trait Anger Scale scores. The 2-way interaction remained significant [$F(7,938) = 3.11$; $p < .01$, $\eta^2 = .023$].

Discussion

Research indicates that anger regulation traits are associated with acute and chronic pain intensity (Bruehl et al., 2002, 2003; Burns et al., 2008, 1996, 2011). Results of our prior work strongly suggest that the dispositional tendency to express anger physically or verbally may be related to acute and chronic pain intensity through deficits in endogenous opioid analgesic function (Bruehl et al., 2002, 2003, 2007). A wide array of findings indicates that effects of anger regulation factors on concurrent and/or subsequent responses are altered during anger arousal (Burns & Katkin, 1993; Burns et al., 2003; Janssen et al., 2001; Suarez et al., 1998). However, the role of actual anger arousal in modulating links among trait anger-out, endogenous opioid function and pain sensitivity had not been directly addressed in previous studies. As discussed above, extant findings appear contradictory, with anger arousal either magnifying later pain among individuals high in anger-out (Bruehl et al., in press; Burns et al., 2004) or reducing it (Burns et al., 2003). Here, we varied the order of anger induction and pain induction among healthy people receiving either placebo or naltrexone to test two models by which relationships between anger regulation style and pain sensitivity could be affected by acute anger arousal. As suggested by our previous findings, we proposed alternatively that: (a) anger arousal may magnify subsequent pain intensity among high anger-out people because in the absence of effective opioid analgesia, the hyperalgesic effects of anger arousal on pain perceptions would dominate, or (b) anger arousal may trigger endogenous opioid release among high anger-out people, thus attenuating pain. Results generally supported the opioid triggering model for trait anger-out, with no significant Drug and Task Order effects for trait anger-in, similar to our past studies (Bruehl et al., 2002, 2003, 2007). In addition, opioid-related effects for anger-out on pain intensity remained significant even after controlling for negative affect and the tendency to express emotions in general, suggesting that findings were specific to anger regulation in particular.

Results for participants who underwent pain-induction prior to anger-induction (and thus pain intensity was not affected by anger) replicated past findings regarding relationships

between trait anger-out, opioid blockade and pain intensity (Bruehl et al., 2002). People with high anger-out reported consistently elevated pain intensity in both placebo and naltrexone conditions relative to low anger-out participants who received placebo. Absence in high anger-out participants of any pain-related effects of opioid blockade, combined with observations of elevated pain specifically among low anger-out participants under opioid blockade implicate deficient endogenous opioid analgesia in the former group. Indeed, results of a supplementary analysis indicated that low anger-out participants in the Naltrexone condition reported significantly greater pain intensity across the ischemic task than low anger-out participants in the Placebo condition. Thus, without the arousal of anger, high anger-out people fail to exhibit opioid analgesia despite experiencing acute pain sufficient to elicit optimal opioid analgesia among low anger-out people. If high anger-out people are characterized by deficient opioid function *regardless of circumstances*, then we would expect the same pattern of pain intensity effects to emerge even when anger arousal precedes pain. Levels of pain intensity were dramatically different for high anger-out people, however, when pain followed anger.

An inspection of Table 2 and Fig. 2 reveals that people high on anger-out who were angered prior to pain-induction and received placebo recorded the lowest pain intensity of any group at least until 150 s into the ischemic pain task. These results support the notion that anger arousal may have an analgesic quality among high anger-out people, and thus may reduce pain sensitivity, albeit temporarily. However, it is crucial to also note that this effect did not emerge among their high anger-out counterparts who received naltrexone. Indeed, results of a supplementary analysis indicated that high anger-out participants in the Naltrexone condition reported significantly greater pain intensity across the ischemic task than high anger-out participants in the Placebo condition. The combination of these results—low pain following anger under placebo, high pain following anger under blockade—suggest that the apparent analgesic effect of anger arousal for high anger-out people may have been partly mediated by endogenous opioids. That is, arousal of anger may have triggered opioid release selectively among high anger-out people to produce some degree of subsequent analgesia, but these effects were blocked by the opioid antagonist, naltrexone. Unlike for low anger-out people, strong negative emotion rather than exposure to painful stimulation may be needed to trigger optimal function of endogenous opioid analgesic systems in high anger-out people.

Results point to potentially important individual differences in the operation of endogenous inhibitory systems on the one hand, and pain and emotion modulation on the other. Findings that high anger-out people report greater pain intensity than low anger-out people when experiencing pain *in the absence of anger arousal*, and that this difference may be due to endogenous opioid deficits in the former suggests structural differences in inhibitory systems between these groups. However, the contrast between placebo and opioid blockade condition pain responses for high anger-out individuals who underwent pain after arousal of acute anger suggests possible functional differences in inhibitory systems between high and low anger-out people as well. Opioid triggering during anger arousal may act to modulate physiological reactivity for high anger-out people. That people reporting high anger-out have been described as displaying greater blood pressure and heart rate increases during anger provocation than low anger out people (e.g., Burns & Katkin, 1993) suggests the

possibility of even greater differences if opioid release did not occur. It may also be the case that high anger-out people experience greater anger during provocation, thus leading to the release of opioids to inhibit further reactivity. Low anger-out people may simply not reach the necessary “anger threshold” as often as high anger-out people. It should be noted, too, that the apparent analgesic effect for prior anger arousal among high anger-out people was relatively short-lived, and was fully observable only because we assessed pain intensity at 30 s intervals rather than taking an average across the epoch. The same phenomena may be true for emotional and physiological reactivity during anger and later pain.

To the extent that opioid triggering by anger arousal among individuals high in anger-out occurs, interesting clinical implications are raised. Endogenous opioids released in response to self-harm have shown the ability to reinforce this behavior in some cases (Sandman & Kemp, 2011). In parallel fashion, it is interesting to speculate whether patterns of extreme, maladaptive, and socially inappropriate expressive anger regulation (e.g., losing temper with boss by screaming, cursing, and hitting walls) might be maintained in part by the reinforcing effects of the opioids released by this anger expressive behavior. Another unusual situation regards the case of chronic pain, which is known to be linked to elevated anger and often involves chronic anger-provoking life situations (e.g., litigation, workers’ compensation cases). Psychotherapy with chronic pain patients may target overt anger expressive behaviors as something to be inhibited (for reasons of social desirability), but may thereby fail to consider the possibility that the patient may experience *reduced* pain as a result of those anger expressive behaviors. Although implications are intriguing, additional work is required to clarify how anger-related opioid triggering may contribute to both adaptive and maladaptive functioning in people high in trait anger-out.

Other results regard the uniqueness of trait anger-out effects. First, the complex interactive effects involving anger-out scores and condition remained significant with general negative affect traits controlled, suggesting that trait anger-out plays a role in affecting pain intensity that is distinct from other components of a broader negative affect construct. Second, effects also remained significant with a trait reflecting the general tendency to express emotions controlled. This finding suggests that the tendency to express anger in particular affects pain intensity beyond the effects exerted by a broader emotional expressiveness construct. Third, as in our past work, the tendency to *inhibit* the expression of anger (anger-in) did not interact with drug and Task Order condition to affect pain intensity, hinting again that only the tendency to verbally and physically *express* anger is related to pain intensity via functioning of the endogenous opioid system.

Some limitations of the study should be delineated. First, we did not use a within-subject design by which we could have derived difference scores between pain ratings under placebo and under naltrexone for each subject. The between-subjects design reduced power and reduced our ability to make strong inferences about relationships among experimental Task Order, anger-out and opioid function, but was necessary because of the harassment procedure. For ethical reasons, subjects needed to be debriefed about the true nature of the confederate’s purpose immediately following the session, which precluded a repeat of the harassment procedure during another session. Second, we used a harassment procedure to arouse anger, but did not use other procedures to invoke other emotions such as sadness or

anxiety. Thus, we do not know whether arousal of sadness or anxiety prior to pain may have endogenous opioid mediated effects on pain intensity. Anger arousal may not be a unique triggering event, and future work must consider effects of other strong negative emotions in producing short-lived, opioid-mediated analgesia. Third, although this study is unique in examining complex interaction effects of anger arousal, anger-out and opioid function, we did not test the effects of the *behaviors* that supposedly comprise the trait anger-out construct. Namely, we did not examine the effects of actual verbal or physical anger expression on the links among trait anger-out, opioid function and pain. Testing whether anger expression in the face of anger arousal also has opioid triggering properties awaits future research. Last, the sample of 146 participants was relatively small given that we examined effects of 4 conditions formed by crossing two Drug conditions with two Task Order conditions. With approximately 35 participants per cell, caution is urged in interpreting results, and replication is sorely needed.

In sum, results suggest that the tendency to regulate anger through overt verbal and/or physical expression, although conceptually and empirically related to broad trait negative affect and the tendency to generally express emotions, represents an important and unique phenomenon in the connection between emotion and pain modulation. Far from exhibiting a stable endogenous opioid deficit, people with elevated anger expressiveness reveal a more complex system of endogenous opioid function that may be responsive to anger arousal. Results hint that people with a pronounced tendency to express anger may suffer from inadequate opioid function under simple pain-induction, but may benefit to some extent from the opioid triggering properties of strong anger arousal. Turning the inferential table somewhat, such findings point to the potentially adaptive nature of anger arousal and perhaps overt anger expression for people with compromised endogenous inhibitory systems: for these people, endogenous pain modulation may occur only during strong anger. This complex relationship among anger expressiveness, anger arousal and pain modulation may present a quandary for clinical intervention. On the one hand, frequent outbursts of expressed anger may not be socially adaptive for most people, including people with chronic pain, and so may be rightfully targeted for reduction via anger management strategies. On the other hand, strong anger and perhaps its overt expression may represent a pathway by which some people gain pain relief. Future research will need to address this predicament in order to find a clinical solution that balances potential social alienation with potential endogenous analgesia.

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Fig. 1.

Pain intensity ratings over 240 s duration of ischemic pain task for participants in the Pain-Induction/Anger-Induction Task Order. Lo AOS/placebo = hypothetical Anger-out subscale score 1 SD below mean for participants receiving placebo. Hi AOS/ placebo = hypothetical Anger-out subscale score 1 SD above the mean for participants receiving placebo. Lo AOS/ naltrexone = hypothetical Anger-out subscale score 1 SD below mean for participants receiving naltrexone. Hi AOS/naltrexone = hypothetical Anger-out subscale score 1 SD above the mean for participants receiving naltrexone

Fig. 2.

Pain intensity ratings over 240 s duration of ischemic pain task for participants in the Anger-induction/Pain-induction Task Order. Lo AOS/placebo = hypothetical Anger-out subscale score 1 SD below mean for participants receiving placebo. Hi AOS/ placebo = hypothetical Anger-out subscale score 1 SD above the mean for participants receiving placebo. Lo AOS/ naltrexone = hypothetical Anger-out subscale score 1 SD below mean for participants receiving naltrexone. Hi AOS/naltrexone = hypothetical Anger-out subscale score 1 SD above the mean for participants receiving naltrexone

Fig. 3.

Pain intensity ratings over 240 s duration of ischemic pain task for all participants collapsed across Drug and Task Order. Lo AIS = hypothetical Anger-in subscale score 1 SD below mean. Hi AIS = hypothetical Anger-in subscale score 1 SD above the mean

Table 1

Correlations among trait factors

	Anger-out	Anger-in	EES	BDI	Trait anxiety	Trait anger
Anger-out	–					
Anger-in	.17	–				
EES	–.23	.45	–			
BDI	.21	.28	.05	–		
Trait anger	.64	.33	–.03	.35	–	
Trait anxiety	.08	.16	.07	.41	.20	–

Absolute value of $r = .17$ or greater is significant at $p < .05$. *EES* emotional expressivity scale, *BDI* beck depression inventory

Table 2

Correlations between anger-out scale scores and pain intensity ratings

Condition	Period (s)							
	30	60	90	120	150	180	210	240
Pain/Anger at placebo	.01	.41	.44	.51	.46	.43	.42	.40
Pain/Anger at naltrexone	.31	.13	.05	.01	.06	.01	.01	.08
Anger/Pain at placebo	-.57	-.51	-.53	-.50	-.24	-.05	.01	.01
Anger/Pain at naltrexone	-.11	-.09	-.11	-.15	-.09	.02	.05	.06

Note: absolute value of $r = .25$ or greater is significant at $p < .05$. Pain/Anger at placebo = pain-induction/anger-induction Task Order for participants receiving placebo. Pain/Anger at naltrexone = pain-induction/anger-induction Task Order for participants receiving naltrexone. Anger/Pain at placebo = anger-induction/ pain-induction Task Order for participants receiving placebo. Anger/Pain at naltrexone = anger-induction/pain-induction Task Order for participants receiving naltrexone

Table 3

Correlations between anger-in scale scores and pain intensity ratings

	Period (s)							
	30	60	90	120	150	180	210	240
Anger-in scale	-.07	-.01	.03	.06	.11	.12	.11	.11