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Suppression of anger and subsequent pain intensity and behavior among chronic low back pain patients: the role of symptom-specific physiological reactivity

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Abstract

Suppression of anger may be linked to heightened pain report and pain behavior during a subsequent painful event among chronic low back patients, but it is not clear whether these effects are partly accounted for by increased physiological reactivity during suppression. Chronic low back pain patients ($N = 58$) were assigned to Suppression or No Suppression conditions for a “cooperative” computer maze task during which a confederate harassed them. During baseline and maze task, patients' lower paraspinal and trapezius muscle tension, blood pressure and heart rate were recorded. After the maze task, patients underwent a structured pain behavior task (behaviors were videotaped and coded). Results showed that: (a) Suppression condition patients revealed greater lower paraspinal muscle tension and systolic blood pressure (SBP) increases during maze task than No Suppression patients (previously published results showed that Suppression condition patients exhibited more pain behaviors than No Suppression patients); (b) residualized lower paraspinal and SBP change scores were related significantly to pain behaviors; (c) both lower paraspinal and SBP reactivity significantly mediated the relationship between Condition and frequency of pain behaviors. Results suggest that suppression-induced lower paraspinal muscle

tension and SBP increases may link the actual suppression of anger during provocation to signs of clinically relevant pain among chronic low back pain patients.

Keywords

Anger suppression; Ironic process model; Symptom-specific reactivity; Pain behaviors

Introduction

The manner in which anger is regulated—for example, suppression (anger-in) or expression (anger-out)—is related to acute (Breuhl et al., 2002; Burns et al., 2003, 2004; Janssen et al., 2001) and chronic pain severity (Breuhl et al., 2002; Burns et al., 1996; Kerns et al., 1994). A number of investigations have focused on associations between suppressed or inhibited anger and pain (Burns et al., 1996, 2003, 2006; Kerns et al., 1994), but have relied on correlations generated between self-reported trait measures of anger-in and pain indexes as evidence of these connections. Most of these studies, therefore, have not examined how the actual *process* of suppressing anger during or following anger-provoking situations may affect acute or chronic pain intensity. To remedy this shortcoming, we have proposed and tested an ironic process model of anger suppression and pain based on Wegner's (1994) ironic process theory of mental control (Quartana & Burns, 2007).

Wegner et al., (1990, 1993), Wegner and Erber, (1992), Wegner and Gold, (1995) showed that attempts to suppress unwanted thoughts appear to have the unintended effect of making these thoughts highly accessible to consciousness. They proposed that attempts to suppress thoughts involve, first, an “operating process” that works to avoid unwanted thoughts through conscious use of distracters. However, they argue that a second process is simultaneously at work that is less conscious and deliberate. This “monitoring process” searches for mental content that signals a failure to suppress the unwanted material. Ironically, by searching for failures to rid awareness of unwanted thoughts and feelings, the cognitive accessibility of this undesired material actually increases. Findings involving a wide diversity of populations and methods support the ironic process model (e.g., Wenzlaff et al., 2001; Newman et al., 1997; Petrie et al., 1998; Tolin et al., 2002).

Our adaptation of the ironic process model for anger suppression and pain holds that suppression of anger may render anger-related thoughts and feelings highly accessible to conscious awareness because the monitoring process works to find more and more instances of failure to avoid or be rid of this content. Although suppression may subdue angry feelings and behavior initially, it may in the long run paradoxically increase the cognitive accessibility of anger, in turn leading to “contamination” (Cioffi & Holloway, 1993) of appraisals of subsequent pain with heightened feelings of irritation and annoyance. Results of our recent studies support this model (Quartana & Burns, 2007; Quartana et al., 2007; Burns et al., 2008a, b). Findings generally show that participants instructed to suppress anger-related thoughts and feelings during anger-induction revealed significantly greater pain intensity during subsequent pain-induction than participants who underwent anger-induction but were not instructed to suppress, both in healthy adults and chronic low back

pain patients. Results generally indicate that when self-reported anger following anger-induction or pain-induction is statistically controlled, differences in pain intensity are attenuated between groups instructed to suppress compared to those given a standardized “think anything” control instruction (Quartana & Burns, 2007; Burns et al., 2008a, b). These findings support the notion that initially suppressed anger can contaminate appraisals of a subsequent painful stimulus in a measurable and potentially clinically meaningful way.

To date, we have focused mostly on whether reports of angry feelings following attempts to suppress anger account for the magnitude of later reports or displays of pain. Anger regulation is also related to physiological reactivity during anger provocation (Finney et al., 2002; Burns, 1995; Lavoie et al., 2001), and such arousal may also constitute a pathway by which later pain is affected. Although much research has focused on the cardiovascular components of sympathetic and parasympathetic nervous system reactivity, recent work indicates that anger variables are also related to activation of skeletal muscles (Burns, 1997; Burns et al., 2006, 2008a, b). Flor and colleagues (Flor et al., 1985, 1991, 1992) proposed a “symptom-specificity” model of chronic pain, which holds that chronic low back pain patients can be expected to show deviant stress-induced muscular responses specific to the disorder. That is, they may reveal strong contraction of low back muscles (i.e., lower paraspinal muscles) during stress while not necessarily showing such tension increases in muscle groups distant from the pain site. Findings support symptom-specificity models among chronic low back patients (Arena et al., 1991; Burns, 1997, 2006; Burns et al., 2006, 2008a, b; Flor et al., 1985, 1991, 1992, 2002; Peters & Schmidt, 1991) and those with neck and shoulder pain (Lundberg et al., 1994, 1999).

We adapted the symptom-specificity model to help conceptualize how anger regulation—state or trait—may be related to heightened chronic pain severity (Burns et al., 2006). To the degree that anger regulation affects muscle contraction near the site of pain or injury, for chronic low back pain patients, efforts to regulate anger should influence lower paraspinal muscle reactivity more strongly than in muscle groups further from the pain site. Indeed, findings suggest that trait anger-out, anger-in and hostility interact to predict low paraspinal tension increases among chronic low back pain patients evoked during anger arousal; effects not evident for trapezius muscles (Burns, 1997; Burns et al., 2006).

In the present study, we conducted further analyses of data reported by Burns et al. (2008a, b). There, chronic low back pain patients underwent anger-induction while they were randomly assigned to either suppress thoughts and feelings about the episode and hide any signs of how they felt, or not try to suppress. All patients then underwent a structured pain behavior task (see Keefe et al., 2001), which involves engaging in everyday movements (e.g., sitting, standing, walking), and which elicits significant increases in low back pain among chronic low back pain patients (Keefe & Block, 1982; Keefe et al., 2001). We found that patients in the Suppression Condition reported significantly greater increases in anger from baseline to the maze task, significantly greater pain intensity increases from baseline to the structured pain behavior task, and revealed significantly more pain behaviors during the structured pain behavior task than those in the No Suppression Condition. Further, the difference between conditions in frequency of pain behavior was largely accounted for by the greater anger report increases of the Suppression Condition patients.

Here, we expanded our model describing ironic effects of anger suppression on pain to include symptom-specific reactivity. We propose that lower paraspinal muscle tension increases elicited during attempts to suppress anger—like the elevations in self-reported anger following suppression we have observed (Burns et al., 2008a, b)—may represent indexes of suppression-induced emotional arousal. Such increased muscle tension in the low back during attempts to suppress anger may magnify later low back pain during everyday movements. If this symptom-specific reactivity model augments our ironic process model, then we may expect that: (a) larger changes in lower paraspinal reactivity among patients trying to suppress anger will account for (i.e., mediate) the greater magnitude of pain intensity shown and/or reported by these patients; (b) links between muscle tension reactivity and pain will be specific to lower paraspinal muscles, and will not be affected by reactivity in a muscle group distant from the site of injury (i.e., trapezius muscles).

Although Flor and colleagues contended that high levels of general autonomic reactivity to stressful stimuli would not distinguish chronic low back pain patients from healthy controls, such reactivity to discrete stimuli may still predict levels of pain to subsequent painful events. On the one hand, findings indicate that resting blood pressure level is inversely related to pain sensitivity (Bruehl & Chung, 2004), and, in line with the literature on stress-induced analgesia (Martenson et al., 2009), results also suggest that blood pressure reactivity to stressful events may predict decreased pain sensitivity (Ditto et al., 1997; France & Stewart, 1995). Thus, we may expect that high blood pressure responses to our experimental manipulation will predict *lower* levels of pain report and behaviors. On the other hand, evidence suggests that the well-known inverse relationship between resting blood pressure and pain sensitivity is altered among people who have chronic pain (Bruehl & Chung, 2004; Martenson et al., 2009; Chung et al., 2008a, b; Bruehl et al., 2010). Also, there is growing evidence that the kind of stressor may affect the nature of the relationship between blood pressure reactivity and pain. Specifically, stressors that provoke irritation and/or anger seem to be linked to blood pressure-mediated *increases* in pain sensitivity (Janssen et al., 2001; Caceres & Burns, 1997). Finally, we have shown that individuals reveal greater blood pressure reactivity in the context of trying to suppress unwanted thoughts and feelings than when not suppressing (Burns et al., 2007; Quartana and Burns, in press). All told, we expected stress-induced (i.e., anger-induced) increases in blood pressure to play a role in mediating differences between chronic low back pain patients on pain report and behaviors, but inconsistent findings regarding the direction of effects—decreased versus increased sensitivity—urge caution in offering specific hypotheses.

Method

Participants

Participants were 60 chronic low back pain patients recruited through advertisements and postings at pain clinics, and they were paid \$40. The study protocol was approved by the Institutional Review Board of the first author's institution. Exclusion criteria were: (a) any current cardiovascular disorder; (b) current use of medications that affect cardiovascular function (i.e., beta blockers); (c) chronic pain stemming from malignant conditions (i.e., cancer); (d) current alcohol or substance abuse problems; (e) a history of psychotic or

bipolar disorders; (f) daily use of narcotic analgesic medication; (g) inability to understand and speak English well enough to take instructions from a confederate (see below). Inclusion criteria were: (a) musculoskeletal pain of the lower back stemming from degenerative processes, muscular or ligamentous strain, or disc herniation as determined by a physician; (b) pain duration of at least 6 mos. Due to equipment problems, the final sample was 58 chronic low back patients. Those who reported occasional use of opioid-based medication were asked not to take these substances on the morning of their appointments. Women comprised 51.7% ($n = 30$) of the sample. Additional information is in Table 1.

Design overview

The design was mixed between- x within-subjects. Two Emotion Regulation Conditions (Suppression; No Suppression) comprised the between-subjects factor, and participants were assigned randomly to these conditions. The within-subjects factor consisted of participants undergoing an anger-induction procedure (under one of the two emotion regulation conditions; see below) followed by the structured pain behavior task (see below) in a fixed order.

Measures

Recording EMG—EMG activity was recorded from left and right lower paraspinal muscles (L2–L4), and left and right trapezius muscles. Silver/silver chloride 8 mm electrodes were spaced 15 mm apart for bipolar recording (see Fridlund and Cacioppo, 1986). Sites were prepared with vigorous alcohol abrasion. Inter-electrode impedance was kept below 10 kilohms. Bioamplifiers with bandpass filters (Coulbourn Instruments) were used to record EMG. Raw electromyography (EMG) signals were amplified by a factor of 100,000. The sampling rate was 1000/sec, and signals were passed through narrow bandpass filters (100–250 Hz). Signals were integrated and “smoothed” with contour-following and cumulative integrators (Coulbourn Instruments). Per recommendations (Fridlund and Cacioppo, 1986), the time constant for integration was 100 ms. Data were collected by computer through A/D conversion using Wingraph software.

Recording cardiovascular indexes Systolic (SBP) and diastolic blood pressure (DBP) and heart rate (HR) were measured with a Dinamap 1846 SX oscillometric BP monitor (Johnson & Johnson Medical Inc.). Readings were obtained every 60-s. Data were collected by computer through A/D conversion using Wingraph software.

Pain intensity checklist Eleven-point Numeric Rating Scales (NRS; Jensen and Karoly, 1992) tapped self-reported pain intensity (0 = None, and 10 = Most Severe Possible).

Anger-induction task

Anger was induced by having a confederate give instructions to a participant during a demanding task. The task was described as a cooperative task for 2 people, the object of which was for one person to move a computer cursor from the entry to the exit of a computer-generated maze. This person, however, was not able to see the maze, but instead moved a computer mouse across a white pad— representing the boundary of the maze— according to instructions issued by the other person—in every case, the confederate—who

was able to see the computer screen. Thus, one person told the other to move the mouse in certain directions and distances in an effort to exit the maze. The participant was always assigned to move the mouse and the confederate always instructed. The participant was also told that they and the other person “need to act as a team to do well,” that “errors” resulted from bumping into maze walls or reversing direction of the cursor, and that he or she cannot speak to the other person (confederate). They were also told that after the participant has completed the second part of the experiment, he or she would have a turn as the instructor and the other person (confederate) would move the mouse.

The confederate assumed an unfriendly attitude from the moment they walked into the room. During the task, the confederate followed a semi-standard script that included instructions to move the cursor in certain directions, reversing directions, exclamations about errors, derogatory comments about the participant's ability, and comments indicating that the confederate blamed the participant for all mistakes. This task and the harassment manipulation were adopted from Engebretson et al. (1989), and we have used it previously to arouse anger in particular (Burns et al., 2009). Male and female experimenters served as confederates. To avoid confounds involving participant-confederate gender matches, equal numbers of same sex, male participant-female confederate, and female participant-male confederate matches were used.

Emotion regulation manipulations

Suppression—In this condition, participants were told to suppress thoughts about their feelings during the maze task and not to show any behavioral signs that showed how they felt. Full details of the procedure are provided in Burns et al. (2008a, b). In brief, patients were asked to give their “best effort on the maze task,” were told that working with a partner “can bring up a lot of thoughts and feelings,” and that they were supposed to suppress what they were thinking and feeling about the task and to hide how they felt at all times. It was emphasized that participants could not speak to the other person.

No suppression—In this condition, participants were told to think anything they wanted and to reveal their feelings if they wanted, but without speaking to the confederate or standing. In brief, they were asked to give their best effort on the task, were told that working with a partner “can bring up a lot of thoughts and feelings,” and that they could “deal with your thoughts and feelings in any way” they chose and that they should “feel free to think about and/or to show your feelings at any time.” Again, it was emphasized that they could not speak to the other person.

Structured pain behavior task

A structured pain behavior task (Keefe and Block, 1982; Keefe et al., 2001) was used as a naturalistic pain-induction manipulation that allows assessment of both self-reported pain intensity and observable pain behaviors (e.g., grimacing). This task involves sitting, standing, walking, reclining, and bending to lift a light-weight object (see below); everyday activities that typically produce mild pain in chronic low back pain patients.

The procedure, variables and data reduction for the structured pain behavior task are described in detail in Burns et al. (2008a, b), and followed the procedures described in Keefe and Block (1982). In brief, participants engaged in 1- and 2-min sitting and standing periods, and two 1-min reclining and walking periods. We added a separate bending and lifting sequence, in which participants picked up a pencil (placed at their feet) from the floor, stood erect, and then replaced it on the floor. The order of positions and activities was varied randomly across participants. The tester spoke to participants only to request activity changes. The 11-min session was videotaped.

Behaviors coded were guarding, bracing, rubbing, grimacing, and sighing. Keefe and Block (1982) reported excellent inter-rater agreement in coding these behaviors, ranging from 93 to 99%. Test–retest reliability of intervals up to 6-mos was also adequate (Keefe et al., 2001). For our data, three graduate students in clinical psychology coded behaviors. Videotapes were prepared for interval recording following the procedure of Keefe et al. (2001). Two raters coded all 58 videotapes separately. Inter-rater reliabilities ranged from $r = .88$ for “bracing” to $r = .96$ for “grimacing.” We used the total frequency of *combined* behaviors in analyses to comprise an index of “pain behaviors.”

Procedure

Participants were screened for exclusion criteria and asked not to consume caffeine, alcohol or nicotine during the 6 h prior to their appointments. When they arrived at the laboratory, procedures and risks were explained, including information about the “other participant” (i.e., confederate) “who will arrive shortly.” All participants reported compliance with the opioid-based medication, caffeine, alcohol and nicotine restrictions. Informed consent was obtained. Participants sat upright. The blood pressure cuff and electrodes were attached, and the participant sat quietly for 10 min while resting EMG, SBP, DBP, and HR readings were taken. After 10-min, the confederate entered and sat 2 m from the participant on the opposite side of a computer table. They were told not to speak. Another 5-min resting period proceeded, after which the participant completed a pain NRS while the confederate did likewise as part of the ruse. Instructions for the maze task and emotion regulation instructions, depending on condition, were given. After the 5-min maze task, the confederate left the room, the electrodes were disconnected and the blood pressure cuff was removed. The participant was then brought to an adjoining room, instructions for the structured pain behavior task were given, and this task began approximately 2.5 min after the maze task ended. After the 11-min structured pain behavior task, the participant completed a pain NRS. They were debriefed, especially with regard to the deception of the confederate. All participants were asked whether they believed the confederate was indeed another patient and was also, like them, a participant in the study. All indicated that they believed the confederate was, in fact, another participant.

Data reduction and analyses—For lower paraspinal and trapezius EMG, readings from left and right sites were summed and averaged. Baseline values for EMG, SBP, DBP and HR were defined as the mean of readings taken during the last 3 min of the 10-min resting period. Maze task values for EMG, SBP, DBP and HR were defined as the mean of readings taken during the task.

Potential mediation pathways were examined by which Emotion Regulation Condition may affect pain indexes indirectly through physiological reactivity during harassment, guided by recommendations of Baron and Kenny (1986). As described above and reported in Burns et al. (2008a, b), we found that Suppression Condition participants reported significantly greater pain severity increases and showed more pain behaviors during the structured pain behavior task than No Suppression participants. These results indicate that the IV (Condition) was related significantly the DVs (pain report and behavior), and thus that the total effects of Emotion Regulation Condition on pain indexes were significant—a precondition for testing mediation.

Here, we conducted a series of 2 Emotion Regulation Condition (Suppression; No Suppression) $\times$ Period (baseline; maze task) mixed design ANOVAs to first determine whether the maze task with harassing confederate produced significant EMG and cardiovascular reactivity, and second to examine whether Condition was related significantly to the proposed physiological reactivity mediators—another precondition for testing mediation. In cases where the Condition was related significantly to a potential mediator (physiological reactivity index), we examined whether residualized change scores for the reactivity index were related significantly to the pain intensity and behavior DVs—yet another precondition for mediation.

In cases where these preconditions were met, we then applied a bootstrapping technique to test the indirect mediation pathway (MacKinnon et al., 2002; Preacher and Hayes, 2008; Shrout and Bolger, 2002). Specifically, we obtained 95% bootstrapped confidence intervals for indirect effects of Condition on pain indexes. Bootstrapping is a nonparametric approach to effect-size estimation and hypothesis testing that does not make assumptions about the form of variable distributions within a given model (e.g., normal versus skewed). Bootstrapping has been recommended as a means of circumventing the power problem introduced by asymmetries and other forms of non-normality in the sampling distribution of $a * b$ (Preacher and Hayes, 2008). In the current study, $a * b$ represents the indirect effect of Emotion Regulation Condition on pain intensity and behaviors, and is defined as the product of the condition to physiological reactivity path (a) and the physiological reactivity to pain index path (b), or $a * b$.

The bootstrapping approach is completed by taking a large number of samples of size n (where n is the original sample size) from the data, sampling with replacement (i.e., an observation that appears only once in the original data can appear multiple times in a bootstrapped dataset), and computing the indirect effect, $a * b$, in each sample. *The point estimate of $a * b$* is the mean $a * b$ calculated over all the bootstrap samples, and the estimated standard error is the standard deviation of the $a * b$ estimates. For confidence interval estimation, we took 1,000 bootstrap samples. To create the 95% confidence interval, the elements of the vector of 1,000 estimates of $a * b$ are sorted from low to high. The lower limit of the confidence interval is the 25th score and the upper limit is the 976th score in this distribution. Bootstrapped confidence intervals were used to test the significance of indirect effects, which appears to be a more appropriate method of indirect effect testing than normal-theory tests (e.g., Sobel's test), particularly for studies with relatively small samples and non-normally distributed variables (MacKinnon et al., 2002; Preacher & Hayes, 2008;

Shrout & Bolger, 2002). More specifically, a bias-corrected bootstrapped confidence interval was used because it has been shown that Type I error rates are lower and statistical power higher for this method than the series of regression analyses (i.e., causal steps approach) recommended by Baron and Kenny (1986).

Results

Physiological changes from baseline to maze task

Lower Paraspinal and trapezius muscles—An Emotion Regulation Condition (Suppression, No Suppression) $\times$ Period (baseline, maze task) mixed design ANOVA was performed for lower paraspinal EMG values. The 2-way interaction was significant [$F(1,56) = 4.02$; $P < .05$; $\eta^2 = .067$]. See Table 2 for means and SDs. Within-subject simple effects analyses showed that both the Suppression [$F(1,28) = 40.50$; $P < .001$] and No Suppression Conditions [$F(1,28) = 46.52$; $P < .001$] provoked significant increases in lower paraspinal muscle tension, and an ANCOVA—controlling for baseline lower paraspinal values—revealed that lower paraspinal values during the maze task were greater for participants in the Suppression than in the No Suppression Condition [$F(1,55) = 3.96$; $P < .05$; $\eta^2 = .067$]. Although both conditions produced significant increases in lower paraspinal muscle tension, participants attempting to suppress anger revealed greater lower paraspinal tension than those not attempting to suppress anger. Thus, Condition exerted a significant effect on this potential mediator.

An Emotion Regulation Condition $\times$ Period (baseline, maze task) mixed design ANOVA was performed for trapezius EMG values. The 2-way interaction was nonsignificant [$F(1,56) < 1$]. However, the Period main effect was significant [$F(1,56) = 81.44$; $P < .001$], and so, participants, irrespective of Condition, showed significant trapezius muscle tension increases during the maze task. See Table 2 for means and SDs. Thus, Condition did not have a significant effect on trapezius muscle tension changes, and so this reactivity index was not pursued further as a potential mediator.

SBP, DBP, and HR—The Emotion Regulation Condition $\times$ Period mixed design ANOVA for SBP was significant [$F(1,56) = 4.14$; $P < .05$; $\eta^2 = .069$]. See Table 2 for means and SDs. Within-subject simple effects analyses showed that both the Suppression [$F(1,28) = 19.55$; $P < .001$] and No Suppression Conditions [$F(1,28) = 8.99$; $P < .01$] elicited significant SBP increases. An ANCOVA controlling for baseline SBP values, however, showed that SBP values during the maze task were greater for those in the Suppression than in the No Suppression Condition [$F(1,55) = 3.95$; $P < .05$; $\eta^2 = .067$]. Although both conditions produced significant SBP increases from baseline, participants attempting to suppress anger revealed greater SBP than those not attempting to suppress anger. Condition therefore had a significant effect on this potential mediator.

The Emotion Regulation Condition $\times$ Period mixed design ANOVA for DBP [$F(1,56) = 3.43$; $P < .07$; $\eta^2 = .058$] and HR [$F(1,56) < 1$] were nonsignificant. The Period main effects for DBP [$F(1,56) = 18.82$; $P < .001$] and HR [$F(1,56) = 32.99$; $P < .001$] were, however, significant. See Table 2 for means and SDs. As was the case for trapezius reactivity, participants showed significant DBP and HR increases during the maze task regardless of

Emotion Regulation Condition. As Condition did not significantly affect DBP and HR reactivity, these factors were not considered in further mediation analyses.

Summary

Participants in both conditions showed significant increases from baseline to the maze task across all physiological indexes. Findings for lower paraspinal and SBP reactivity were noteworthy, however, in that participants in the Suppression Condition revealed greater lower paraspinal tension and SBP increases than those in the No Suppression Condition. Thus, Emotion Regulation Condition was related significantly to the 2 DVs [pain intensity ratings and behaviors, per prior results (Burns et al., 2008a, b)] and to potential physiological mediators (lower paraspinal and SBP reactivity).

Tests of lower paraspinal and SBP reactivity as potential mediators of the links between

Emotion regulation condition and pain indexes—Residualized change scores for lower paraspinal muscle tension, SBP and pain intensity ratings were computed by regressing baseline values on maze task values. Zero-order correlations were generated among these scores and frequency of pain behaviors. Results showed that lower paraspinal ($r = .35$; $P < .01$) and SBP ($r = .45$; $P < .01$) change scores (i.e., reactivity) were related significantly to pain behaviors, but not to pain intensity ratings (r 's $< .19$; P 's $> .10$). Given that a mediator must be at least be significantly related to a DV, mediation analyses were not pursued for pain intensity ratings.

Analyses proceeded to test whether lower paraspinal reactivity mediated the relationship between Emotion Regulation Condition and pain behaviors. See Fig. 1. Recall that Condition was related significantly to frequency of pain behaviors—as reported in Burns et al. (2008a, b)—which converted to a total effect of $r = .29$ ($P < .05$). The path from lower paraspinal reactivity to pain behaviors—with Condition controlled—was significant ($b = .30$, $P < .05$), as was the path from Condition to lower paraspinal muscle reactivity ($r = .26$, $P < .05$). The direct effect of Condition on pain behaviors was not significant ($b = .21$, $P > .10$) with lower paraspinal reactivity in the model. Most critically, the bootstrapped mean for the $a * b$ path (i.e., indirect effect) was $b = .08$ and the 95% confidence interval did not include zero ($b = .04$ to $b = .12$). This finding suggests that the total effect of Emotion Regulation Condition on pain behaviors featured a significant indirect effect via anger-induced lower paraspinal muscle tension increases.

With SBP reactivity as the potential mediator (see Fig. 2), the path from SBP reactivity to pain behaviors—with Condition controlled—was significant ($b = .40$, $P < .05$). Also, the path from Emotion Regulation Condition to SBP reactivity was also significant ($r = .26$, $P < .05$). The direct effect of Condition on pain behaviors was not significant ($b = .19$, $P > .10$) with SBP reactivity in the model. Finally, the bootstrapped mean for the $a * b$ path (i.e., indirect effect) was $b = .10$ and the 95% confidence interval did not include zero ($b = .05$ to $b = .15$). Similar to lower paraspinal reactivity, this finding suggests that the total effect of Emotion Regulation Condition on pain behaviors featured a significant indirect effect through the effect of anger-induced SBP increases.

Taken together, results suggest that the greater number of pain behaviors exhibited by chronic low back pain patients following attempts to suppress anger than those shown by patients who did not try to suppress could be at least partly explained by the greater magnitude of anger-induced symptom-specific physiological reactivity (lower paraspinal muscle tension) and SBP increases revealed by the former.

Discussion

We tested an ironic process model of anger suppression and chronic pain with regard to the role of symptom-specific reactivity in fostering delayed effects on pain severity among chronic low back pain patients. Our focal hypothesis was that increased tension of muscles in the lower back (i.e., lower paraspinal muscles) during attempts to suppress anger would represent a generative mechanism whereby anger suppression would lead to subsequent low back pain during everyday movements, whereas tension increases in muscles far from the lower back (i.e., trapezius) would not. The role of anger-induced cardiovascular reactivity in affecting later pain was also examined. Results supported the mediating roles of lower paraspinal and SBP reactivity, such that greater lower paraspinal muscle tension and SBP increases shown by patients who attempted to suppress anger, as opposed to those who did not, partly explained the greater number of pain behaviors they subsequently exhibited during a task mimicking everyday activities.

An important finding was that symptom-specific lower paraspinal reactivity, induced during interpersonal harassment, appeared to predict later pain intensity during a naturalistic pain task among chronic low back pain patients. Although we have reported that lab-induced lower paraspinal reactivity was correlated significantly with self-reports of daily chronic pain severity (Burns, 2006), to our knowledge this is the first report of low back muscle tension increases predicting lab-induced pain behaviors among a sample of people with chronic low back pain. These findings add much support to the general notion that muscle tension changes can negatively affect acute flareups and background levels of pain among people with conditions that produce persistent pain (Lundberg et al., 1994, 1999). As we have reported previously (Burns, 2006; Burns et al., 2008a, b), trapezius muscle tension also increased significantly during anger-induction. However, trapezius reactivity was not related significantly to later pain intensity, and thus in the absence of significant effects for a muscle group distant from the site of pain and/or injury, a symptom-specific approach is supported. That is, only tension in muscles near the site of the pain problem for chronic low back pain patients—lower paraspinal muscles—predicted subsequent pain behaviors.

Nonetheless, it is important to note that lower paraspinal tension increased on average by .47 microvolts. Although the pain behaviors were in response to everyday activities, it is not immediately clear what the “real-world” impact of this level of muscle tension may be. On one level, results do indeed indicate that a .47 microvolt increase in lower paraspinal muscle tension preceded and correlated with subsequent pain behaviors, suggesting that such tension increases played some kind of causal role. On another level, the muscle tension increases exhibited by chronic low back patients could represent just one component of a more comprehensive anger arousal experience. Indeed, as reported in Burns et al. (2008a, b), self-reported anger following the maze task also predicted subsequent frequency of pain

behaviors. An apparently small lower paraspinal muscle tension increase as part of more general anger response could thus predict a portion of pain behaviors.

Beyond this “main effect” for lower paraspinal reactivity, a key finding was that lower paraspinal reactivity appeared to mediate the effects of anger suppression on later pain behaviors. Although harassment elicited significant increases in lower paraspinal muscle tension across emotion regulation conditions, attempts to suppress thoughts and to hide feelings produced somewhat greater reactivity. These findings are consistent with work by Gross and colleagues regarding the physiological effects of expressive suppression (54), and our work showing that inhibiting anger expression in particular led to slower lower paraspinal recovery to baseline following provocation than expressing anger (Burns et al., 2008a, b). Mediation tests in the present study suggest that the greater lower paraspinal tension reactivity shown by participants attempting to suppress anger were partly responsible for the greater pain behaviors these people showed later. One way to interpret these findings is that attempts to suppress thoughts, feelings and behaviors of anger may have caused greater tension in muscles of the lower back than simply feeling angry, which in turn generated greater low back pain intensity, reflected in this case in more frequent pain behavior during everyday activities. When considered together with the null effects for trapezius reactivity, results indicate that suppressing anger may not only induce muscle tension in general, but may aggravate a particular clinical vulnerability factor which in turn elevates pain. As our prior work suggests, anger states may be bad enough for chronic low back pain patients (Burns, 2006), but deliberately suppressing anger-related thoughts and behavior may have even greater detrimental effects on subsequent well-being.

We also found that anger-induced SBP reactivity predicted frequency of subsequent pain behaviors. The literature regarding associations between blood pressure reactivity to stress and pain sensitivity as it applies to our results is characterized by at least two key issues. First, there is reason to believe that associations between stress-induced blood pressure responses may be related to pain sensitivity differently among healthy people compared to chronic pain patients. In healthy individuals, blood pressure levels and pain sensitivity appear to be inversely related (37). Although mechanisms underlying this inverse association are not fully understood, it has been proposed that blood pressure response to stress triggers baroreceptors, which in turn activate endogenous descending pain inhibitory systems (Bruehl and Chung, 2004; Zamir and Maixner, 1986). Evidence suggests, however, that there is an absence, or even a reversal of this pattern among chronic pain patients (e.g., Maixner et al., 1997), including CLBP patients (Chung et al., 2008a, b). In addition, baroreceptor sensitivity is diminished coincident with increased pain sensitivity and exaggerated temporal summation of pain in chronic low back pain (Chung et al., 2008a, b). Thus, despite evidence of stress-induced analgesia (Martenson et al., 2009), stress-induced blood pressure reactivity in systems compromised by persistent pain of long duration may produce quite different relationships. Second, it may also be the case that different stressors have distinct impacts on pain responses. Whereas evidence indicates that some lab stressors elicit reduced pain sensitivity mediated by blood pressure increases (Ditto et al., 1997; France and Stewart, 1995), stressors that provoke irritation and anger may uniquely elicit blood pressure mediated increases in pain sensitivity (Janssen et al., 2001; Caceres and Burns, 1997). Our results support this notion that anger-induced increases in SBP in particular may have

detrimental effects on subsequent pain sensitivity, especially among those suffering from chronic pain. Further research is certainly needed to uncover the specific mechanisms that link arousal of discrete emotions (e.g., approach versus avoidance) with subsequent pain sensitivity.

Similar to lower paraspinal reactivity, SBP reactivity also mediated the effect of Emotion Regulation Condition on pain behaviors such that patients in the suppression condition showed greater SBP responses to anger provocation, which in turn predicted more frequent pain behaviors. As we and others have shown, suppression of thoughts and behavioral expression during stressful events appears to enhance physiological reactivity in general (Quartana & Burns, 2007; Wegner et al., 1990; Gross & Levenson, 1993). To the extent it does—manifested as muscle tension or cardiovascular function or both—such regulation during anger may carry risks for later aggravated pain in chronic low back pain patients.

Our findings expand what is known about the rather ambiguous relationship between anger inhibition and chronic pain. As we have argued (Quartana & Burns, 2007; Burns et al., 2007, 2008), many conclusions regarding the effects of anger inhibition on chronic pain intensity stem from results of *correlational* studies examining links between trait anger-in scales and indexes of—usually—average daily pain. By combining an ironic process model of anger suppression with experimental laboratory methods, the present study contributes to establishing a causal connection between the actual process of suppressing anger and pain intensity, and further implicates definable physiological mechanisms. Please note that the pain-induction task used here—the structured pain behavior task—appears to induce low back pain among chronic low back pain patients (Keefe et al., 2001). Present results therefore imply that initial attempts to suppress anger may lead to significant aggravation of *clinically-relevant* pain stemming from a chronic condition because of amplified muscle tension and blood pressure increases during suppressed anger. That is, suppressing anger during provocation may have influenced chronic low back pain patients' subsequent display of grimacing, bracing, and guarding during activity – behaviors reflecting discomfort and suffering – at least in part due to increased tension in muscles near the site of injury.

The present study has limitations. First, we did not use a condition to arouse a negative emotion other than anger. In Quartana and Burns (2007), we had distinct anxiety- and anger-induction conditions crossed with experiential *or* expressive suppression manipulations. This design allowed us to make explicit comparisons between the effects of anxiety suppression and anger suppression manipulations on pain intensity. Because participants in the two anger suppression conditions reported the greatest subsequent pain intensity, we decided to include only anger-induction in this study. Despite prior results and present findings that lower paraspinal and SBP reactivity during the maze task accounted for group differences in pain behaviors, we still cannot definitively conclude that effects on pain behaviors during movements found in everyday life were specific to the suppression of anger. Second, the effect size for differences between suppression and no suppression conditions on pain behaviors was somewhat modest ($r = .29$). Indeed, on average the suppression participants showed only 3.5 more pain behaviors over an 11-min period than their no suppression counterparts (see Burns et al., 2008a, b). Thus, the clinical or “real-world” significance of this suppression-induced difference in (clinically-relevant) pain

behaviors must be interpreted cautiously. Third, our suppression manipulation combined suppression of thoughts and suppression of behaviors. Although Quartana and Burns (2007) did not find significant differences on later pain intensity between “experiential” (thoughts and feelings) and “expressive suppression” (behavior), their study awaits replication. Also, it may be the case that thought/feeling suppression (a la Wegner) and expressive suppression (a la Gross) may have distinct effects on physiological parameters, and so exert effects on later pain intensity through different mechanisms. For instance, inhibition of behavioral expression of anger may be the key ingredient in elevating lower paraspinal muscle tension, and so this single element of suppression may exert the most pronounced ironic effects on later pain intensity. Exploring these issues are topics for further research.

Results of numerous studies show that the regulation of strong negative emotions influences physical health. Emotion suppression has historically been viewed as a problematic and pathogenic manner of regulating emotions, and recent empirical results appear to support these claims (Consedine et al., 2002; John & Gross, 2004). Our findings here extend previous work on an ironic process model of anger suppression and pain (Quartana & Burns, 2007) to reveal that the connection between initial attempts to suppress anger during provocation and subsequent aggravation of clinical pain among chronic low back pain patients may occur partly through anger-induced physiological arousal, most particularly in a parameter reflecting a clinical vulnerability (i.e., low back muscles). Beyond the laboratory, such results may indicate the possibility of a vicious cycle involving suppression-induced muscle tension reactivity leading to more pain during later activities leading to sustained muscle tension as a response to the pain, and so on. Research indicates that deliberate efforts to disinhibit previously suppressed emotions around traumatic or stressful events appear to have beneficial effects on patients with chronic pain conditions (Broderick et al., 2004; Kelley et al., 1997). Results of work uncovering effects of the process of emotional suppression on physical pain and the physiological mechanisms that enhance later pain intensity may guide the development and use of therapeutic techniques designed to preempt patient pain by halting inhibition and applying relaxation tactics before a vicious cycle begins in earnest.

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Fig. 1. Mediation of relationship between emotion regulation condition and pain behavior via lower paraspinal reactivity

Fig. 2. Mediation of relationship between emotion regulation condition and pain behavior via SBP reactivity

Table 1**Descriptive data (*N* = 58)**

Variables	Statistics			
	<i>M</i>	<i>SD</i>	%	<i>n</i>
Age (years)	39.2	9.7		
At least 12-years of education			90.0%	52
Ethnicity				
Caucasian			67.2%	39
Hispanic			10.3%	6
African American			15.5%	9
Asian			1.7%	1
Native American			5.2	3
Pain duration (mos)	48.4	45.2		
Opioid-based			20.7%	12
Nonsteroidal			32.8%	19
Anti-inflammatory				
Muscle relaxants			34.5%	20
Antidepressants			8.6%	5

Table 2
Mean (SD) lower paraspinal, trapezius, SBP, DBP, and HR values by condition and period

Dependent variable and condition	Period	
	Baseline	Maze Task
Lower Paraspinal		
No suppression	.64 (.2)	1.00 (.4)
Suppression	.66 (.3)	1.24 (.6)
Trapezius		
No suppression	.68 (.3)	1.99 (1.0)
Suppression	.67 (.2)	2.16 (1.3)
SBP		
No suppression	123.05 (18.8)	127.67 (21.8)
Suppression	125.55 (23.7)	135.55 (27.0)
DBP		
No suppression	72.30 (7.5)	74.55 (9.3)
Suppression	74.23 (11.5)	79.26 (12.9)
HR		
No suppression	67.66 (9.3)	72.35 (12.4)
Suppression	68.38 (6.2)	73.87 (8.2)