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Anger Arousal and Behavioral Anger Regulation in Everyday Life among Patients with Chronic Low Back Pain: Relationships to Patient Pain and Function

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Abstract

Objective—To determine the degree to which patient anger arousal and behavioral anger regulation (expression, inhibition) occurring in the course of daily life was related to patient pain and function as rated by patients and their spouses.

Method—Married couples ($N = 105$) (one spouse with chronic low back pain) completed electronic daily diaries, with assessments 5 times/day for 14 days. Patients completed items on

their own state anger, behavioral anger expression and inhibition, and pain-related factors. Spouses completed items on their observations of patient pain-related factors. Hierarchical linear modeling was used to test concurrent and lagged relationships.

Results—Patient-reported increases in state anger were related to their reports of concurrent increases in pain and pain interference and to spouse reports of patient pain and pain behavior. Patient-reported increases in behavioral anger expression were related to lagged increases in pain intensity and interference and decreases in function. Most of these relationships remained significant with state anger controlled. Patient-reported increases in behavioral anger inhibition were related to concurrent increases in pain interference and decreases in function, which also remained significant with state anger controlled. Patient-reported increases in state anger were related to lagged increases in spouse reports of patient pain intensity and pain behaviors.

Conclusions—Results indicate that in patients with chronic pain, anger arousal and both behavioral anger expression and inhibition in everyday life are related to elevated pain intensity and decreased function as reported by patients. Spouse ratings show some degree of concordance with patient reports.

Keywords

Anger arousal; behavioral anger expression and inhibition; pain and function; patient and spouse ratings; electronic daily diary; lagged associations

For people with chronic pain, anger-related factors are significantly associated with pain intensity, mood and physical function such that greater anger is related to poorer adjustment (Janssen, Spinhoven, & Brosschot, 2001; Kerns, Rosenberg, & Jacob, 1994; Nicholson, Gramling, Ong, & Buenevar, 2003; Wade, Price, Hamer, Schwartz & Hart, 1990). Evidence suggests that how anger is regulated – expression, inhibition – may have greater consequences for chronic pain patients' pain and function than their level of state anger (Bruehl, Chung, & Burns, 2006; Bruehl, Burns, Chung, Ward, & Johnson, 2002; Burns et al., 2008).

Anger expression may be related to pain and function along at least two pathways. First, it has been proposed that people who tend to verbally or physically express anger and who exhibit high pain sensitivity may be characterized by deficits in endogenous inhibitory mechanisms (Bruehl et al., 2002). Findings support this model and indicate that chronic pain patients and healthy people alike who are high in trait anger-expressiveness show evidence of deficient endogenous opioid function, thus leaving them with elevated pain sensitivity (Bruehl et al., 2002). Second, a symptom-specific reactivity model (Flor, Turk & Birbaumer, 1985) has been adapted for anger and chronic pain, and holds that anger arousal may lead to increases in muscle tension near the site of injury, and thereby increase pain (Burns, Bruehl, & Quartana, 2006). In the case of chronic low back pain (CLBP) patients, the relevant muscles would be in the low back (e.g., lower paraspinal [LP] muscles). Results support this model, and indicate that anger arousal among CLBP patients produces greater increases in LP muscle tension than sadness (Burns, 2006), and that CLBP patients with elevated trait anger-expressiveness showed greater increases in LP muscle tension during anger arousal than those low in trait anger-expressiveness (Burns et al., 2006).

Anger inhibition may be related to pain and function also along at least two pathways. First, a thought suppression model (Wegner, 1994) was adapted for anger inhibition and pain, and holds that inhibiting anger has the ironic consequence of amplifying and maintaining anger which in turn exerts delayed negative effects on responses to later events (Burns, Quartana, & Bruehl, 2008). Thus, inhibiting anger can worsen later pain perception as ironically sustained thoughts of anger contaminate the experience of pain. Results support this model in that chronic pain patients and healthy people who inhibited anger during anger arousal showed greater increases in anger during anger-induction and reported greater anger and pain intensity during subsequent acute pain-induction than those who did not inhibit anger during anger arousal (Burns, 2008; Quartana & Burns 2007). Second, the symptom-specific reactivity model may also apply to anger inhibition. Findings showed that CLBP patients evinced greater increases in LP muscle tension while inhibiting anger during anger arousal than those not inhibiting, and these increases were shown to predict the frequency of observable pain behaviors during subsequent pain induction (Burns, Quartana, Gilliam, Matsuura, Nappi, & Wolfe, 2012).

These conceptual models support hypotheses that *both* anger expression and inhibition can lead to increased pain sensitivity and intensity, and may do so via distinct pathways. Findings from numerous cross-sectional questionnaire and lab-based studies support these contentions (Bruehl et al., 2006; Burns et al., 2008). However, the questionnaire studies based on traits tell us little about the effects of *actual anger regulation behaviors* on pain and functioning, and although some lab-based studies did manipulate anger arousal, anger regulation behavior and pain in controlled conditions, ecological validity is limited by the artificial nature of the procedures. Thus, we know little about relationships between what people actually do in everyday life to regulate anger and their pain and function.

Results of two recent daily diary studies help address this shortcoming. In the first study (van Middendorp, Lumley, Moerbeek, Jacobs, Bijlsma, & Geenen, 2010), results suggested that behavioral anger expression during an anger-provoking event during the day was related to elevated end-of-day pain. They also reported that state anger was related to higher end-of-day pain. This design did *not* include true lagged analyses, so it is not clear whether anger predicted pain or vice versa. In the second study (Bruehl, Liu, Burns, Chont, & Jamison, 2012), results of lagged analyses suggested that behavioral anger expression during one period was related to elevated pain in the subsequent period, providing the first evidence of a longitudinal effect. Their assessment was limited, however, only to anger expression and patient rated pain intensity, and, moreover, did not include state anger.

In the present study, we used electronic diary methods to replicate and extend prior work, and evaluate the degree to which patient feelings of anger and behavioral anger regulation – *both* expression and inhibition – occurring in the course of daily life was related to *both* patient pain and function as rated by patients. In addition, these data were collected as part of a larger study that also included spouse ratings of patient pain and function (Burns et al., in press). Specifically, patients with chronic low back pain (CLBP) and their spouses completed electronic diary entries 5 times/day for 14 days using Personal Data Assistants (PDAs). On one level, if anger arousal and/or behavioral anger regulation, as it occurs in everyday life, are related to pain and function, then concurrent analyses would show

relationships between increases in state anger, anger expression and/or inhibition and fluctuations in pain and function. Further, if anger arousal and/or behavioral anger regulation actually *influence* pain and function, then *lagged* analyses would show that initial increases in state anger, anger expression and/or inhibition predict later fluctuations in pain and function, particularly in the case of anger inhibition given prior evidence of delayed effects on pain (Burns et al. 2008) (i.e., anger → pain). Alternatively, it may be the case, as suggested by results of Feldman, Downey and Schiffer-Neitz (1999), that initial pain intensity could predispose someone to experience later anger and the need to regulate it, and thus initial increases in pain would predict later fluctuations in anger arousal, anger expression and/or inhibition (i.e., pain → anger). On another level, if increases in anger arousal, anger expression and/or inhibition are indeed related to changes in patient pain and function, these increases should be related to demonstrable pain and function changes that can be observed by reporters other than the patient. We hypothesized that patient-reported increases in their anger arousal, behavioral anger expression and/or inhibition would be related to *spouse* ratings of observable patient behavior. Finally, the need to regulate anger presupposes the presence of anger arousal. If patient behavioral anger regulation per se is a key phenomenon related to current and later pain and function, then anger regulation factors should exert unique effects beyond those attributable to anger arousal alone. To address this issue, we controlled for either concurrent state anger or lagged state anger where relevant. Note that because anger arousal is a core and necessary constituent of the phenomenon “anger regulation,” statistically controlling for state anger represents a conservative and stringent approach.

Method

Participants

One hundred and twenty-one married couples were recruited through referrals from staff at the pain clinics of Rush University Medical Center in Chicago, IL, Duke University Medical Center in Durham, NC, Memorial Hospital in South Bend, IN, and through advertisements in local newspapers and flyers provided at various health care agencies. Each participant received \$150. The protocol was approved by the Institutional Review Boards at Rush University Medical Center, Duke University Medical Center, and University of Notre Dame.

Patient inclusion criteria were: a) pain of the lower back stemming from degenerative disk disease, spinal stenosis, or disk herniation (radiculopathy subcategory), or muscular or ligamentous strain (chronic myofascial pain subcategory); b) pain duration of at least 6 months with an average intensity of at least 3/10 (0 = “no pain”, 10 = “worst pain possible”); and c) age between 18 and 70 years. The inclusion criterion for spouses was age between 18 and 70 years.

Exclusion criteria for both patients and spouses were: a) current alcohol or substance abuse problems, or meeting criteria for alcohol or substance abuse or dependence (within the past 12 months); b) a history of, or current psychotic or bipolar disorders; c) inability to understand English well enough to complete questionnaires; d) acute suicidality; and e) meeting criteria for obsessive-compulsive disorder or posttraumatic stress disorder within the past 2 years. A further exclusion criterion for patients was if their pain complaint was

due to malignant conditions (e.g., cancer, rheumatoid arthritis), migraine or tension headache, fibromyalgia, or complex regional pain syndrome. A further exclusion criterion for spouses was if they reported currently suffering from a condition that caused episodes of acute pain (i.e., migraine headaches) or reported a history of chronic pain within the past 12 months.

Inclusion and exclusion criteria were assessed by a detailed medical and psychosocial history, including administration of the Mood Disorder, Psychotic Screening, and Substance Use Disorders modules of the Structured Clinical Interview for DSM-IV Axis I Disorders – Non-Patient Edition (SCID-IV/NP; First, Spitzer, Gibbon, & Williams, 1996).

Of the 121 couples recruited, eight couples declined to participate in the diary portion of the study, three couples withdrew before completing 14 days of data collection, four couples lost data due to PDA malfunctions, and one couple's data were lost due to failure to upload it from the PDA at an appropriate time. Thus, the final sample was 105 couples. Women patients comprised 48.6% of the sample ($n = 51$). Demographic characteristics of couples not included in this investigation did not differ significantly from those who were included. See Table 1.

Electronic Diary

The PDA program signaled participants to complete five assessments each day, starting at 8:50 am and occurring every three hours until 8:50 pm. Frequent assessments helped minimize retrospective bias in ratings (Stone & Shiffman, 1994). Daily diary data obtained in this manner appear to suffer little from reactivity effects that may be linked to monitoring (Cruise, Broderick, Porter, Keall, & Stone, 1996; Jamison, Raymond, Levine, Slawsby, Nedeljkovic, & Katz, 2001). Variability in ratings within the day is also captured by this method (Peters, Sorbi, Kruise, Kerssens, Verhaak & Bensing, 2000; Stone, Broderick, Porter, & Kaell, 1997). Previous studies support reliability, validity, and compliance with electronic diary methods when used to assess pain, affect, and behavior (Cruise et al, 1996; Jamison et al., 2001; Peters et al, 2000; Stone & Shiffman, 1994). Electronic diaries with time-stamped entries also allow accurate assessment of when ratings were made, something that cannot be done with paper diary methods (Jamison et al., 2001). Finally, the PDA software we used allowed us to include branching algorithms. We used the branching algorithms to direct participants to questions about anger regulation if a state anger threshold was crossed (see below), and to assess whether spouses observed patients in the past three hours. If spouses reported observing patients, they were directed to questions asking about patient behavior, but if they did not observe the patient, they were not asked these questions. Both patients and spouses completed electronic diary measures for 14 consecutive days. We used the Experience Sampling Program (ESP; Barrett & Feldman, 2000) on handheld Palm® Zire 22 PDAs, running the Palm OS platform.

Measures

State anger—At each assessment, patients rated the extent to which they felt annoyed, irritable, and angry during the past 3 hours. Responses were made on 9-point scales with anchors at 0 (*not at all*), 2 (*somewhat*), 4 (*much*), 6 (*very much*), and 8 (*extremely*). Because

the inter-correlations among these three items was $r = .63$ or greater and the alpha coefficient of internal consistency for the three-items was $\alpha = .97$, a State Anger variable was computed by summing and averaging them.

Behavioral anger expression and inhibition—If any of the three items on the state anger scale were rated a “2” or greater, the diary software activated a branch algorithm through which participants were asked about behavioral anger expression and inhibition at that assessment. The items and responses were as follows:

When you felt irritated, annoyed or angry during the past 3-hours, to what degree did you do the following?

1. I spoke or shouted about my anger or annoyance
2. I did physical things like gesture, pound the table, slam doors, throw things, etc.
3. I kept my anger or annoyance to myself
4. I hid from others how angry or annoyed I was

These responses were rated on the same 9-point Likert scale. A behavioral anger expression variable was computed by summing the first two items ($r = .69$, $p < .01$), and a behavioral anger inhibition variable was computed by summing the last two items ($r = .90$, $p < .01$).

Patient-reported pain-related variables—At each assessment, patients also rated “how intense was your pain,” “to what degree did your pain interfere with you being physically active,” and “how much did you rest (sit, lie down) because of your pain” during the past 3 hours. Responses were made on 9-point scales with anchors at 0 (*not at all*), 2 (*somewhat*), 4 (*much*), 6 (*very much*), and 8 (*extremely*).

Spouse-observed patient pain-related variables—At each assessment, spouses were asked, “Did you observe your spouse during the past 3 hours?” If they responded, “yes,” then the diary software activated a branching algorithm through which spouses were asked about various aspects of patients’ pain. Here, we analyzed only items that reflected observable patient behavior. The items “how much pain did he/she appear to be in,” “how physically active was he/she,” and “how many ‘pain behaviors’ (complaining, grimacing, etc) did you hear or see?” were included. Responses for the pain intensity and physical activity item were made on a 9-point scale with anchors as described above, and the pain behavior item used anchors, 0 (*none*), 2 (*a few*), 4 (*some*), 6 (*many*), and 8 (*very many*).

Procedure

Patients and spouses who inquired about participation underwent screening procedures over the phone. Eligible patients and spouses attended an initial session during which they signed consent forms to participate, and completed questionnaires. Patients and spouses were instructed to carry the PDAs with them throughout the day for 14 consecutive days. Research assistants described and defined terms contained in the diary items for participants, and provided them with printed instructions as well. For instance, the term “pain behaviors” was explicitly defined for spouses. Participants were also given printed versions of these

instructions for later reference and were asked to phone the research assistants with any problems or questions.

Starting at 8:50 am, and then again every three hours until 8:50 pm, participants were prompted by the PDA alarm to complete assessments. Participants had 15 minutes following the alert to respond to the PDA and diary items. After the alarm, the PDA would emit a signal every 30-seconds until participants responded. Participants were given the option to tap the screen to dismiss the alarms and delay the signal for up to 15 minutes. If participants did not respond within 15 minutes of the original prompt, the time period was coded as missing data. Data for each assessment session was time stamped. After 14 days of data collection, participants returned the PDA, data were downloaded, and participants were debriefed.

Data Preparation

All item responses submitted past the 15-minute response interval were discarded. After deleting these responses, out of 7350 possible total responses, there were 80.1 – 87.1% complete data for the items in the diary. This amount of complete data is in the range typically found in other electronic diary studies involving pain patients (Shiffman, Stone, & Hufford, 2008).

Data Analyses

All analyses were conducted in SPSS version 20 (IBM, 2011). Descriptive statistics were used to characterize the study variables. Our main analyses focused on cross-lagged relationships between behavioral anger regulation and pain over time. For these analyses, hierarchical linear modeling was used. Predictor variables were centered using person mean centering. As a consequence of this centering algorithm, parameters represent a person's deviance from his or her own average over the course of the study. Parameters, therefore, represent within-subjects effects. Autocorrelation of the dependent variable (DV) over time was accounted for by controlling for DV values measured three hours prior. State Anger was entered into the model to account assess unique impacts of Behavioral Anger Expression and Behavioral Anger Inhibition over and above the impact of anger arousal. The equations also adjusted for time since the start of the study to account for patient reactivity, and to account for the unequal spacing of time points due to the nighttime lag between 8:50 pm and 8:50 am measurements. Random effects were included for intercept and time to account for individual differences at baseline and over the course of the study. We thus modeled statistical causality to the extent that change in predictor variables preceded, and was correlated with change in subsequent dependent variables while also ruling out potential extraneous variables (Duckworth, Tsukiyama & Maya, 2010).

Two general models were computed. The first model was computed to estimate concurrent effects and the second to estimate lagged effects. Concurrent models were those in which either patient behavioral anger expression or inhibition (IV) was related to patient pain-related variables (e.g., pain intensity) at the same time while controlling for prior values of the pain-related factor. A representative level-1 model for concurrent effects (i.e., all

variables are measured at the same time point, aside from prior measurement of the dependent variable) is:

$$\begin{aligned} \text{Pain Intensity}_{ij} = & \pi_{0ij} + \pi_{1ij}(\text{Behavioral Anger Expression}) \\ & + \pi_{2ij}(\text{State Anger}) + \pi_{3ij}(\text{Pain Intensity at } t \\ & - 1) + \pi_{4ij}(\text{Time}) + r_{ij}, \end{aligned}$$

where i represents the “ t ”th time point and j represents the “ j ”th person. Time is measured in hours since the start of the study and is centered at 0 so that the intercept, π_{0i} , represents patient pain intensity at the first time point of the study. Behavioral Anger Expression is the person’s deviation in behavioral anger expression from his or her behavioral anger expression scores across the entire study. The DV, Pain Intensity, is the person’s present pain intensity. Pain Intensity at $t-1$ represents the score of the DV at the prior time point (viz., three hours earlier).

The general lagged effects model was the same as the concurrent effects model except that all of the predictor variables (IV’s) were lagged, or measured at the prior time point, three hours earlier. Lagged models tested whether either patient behavioral anger expression or inhibition predicted patient pain-related factors (e.g., pain intensity) three hours later while controlling for prior values of the pain-related variables. Thus, the general lagged level-1 model of behavioral anger expression and pain intensity is:

$$\begin{aligned} \text{Pain Intensity}_{ij} = & \pi_{0ij} + \pi_{1ij}(\text{Behavioral Anger Expression at } t \\ & - 1) + \pi_{2ij}(\text{State Anger at } t \\ & - 1) + \pi_{3ij}(\text{Pain Intensity at } t \\ & - 1) + \pi_{4ij}(\text{Time}) + r_{ij}, \end{aligned}$$

We focused on relatively short, 3 hour lags rather than the longer lags often found in extant literature, such as evening of Day 1 to evening of Day 2. A short assessment window allowed us to capture acute effects of immediate patient anger regulation that might be lost over longer periods. Also, regarding spouse ratings of patient pain and function, spouses reported observing patients during 51% of the 3-hour intervals. Spouses reported observing some sign that the patient was in pain or distress during 73% of these intervals.

Effect sizes were estimated using Cohen’s f^2 , which provides an estimate of variance accounted for by the independent variable of interest (Cohen, 1988).

Results

Descriptive Analyses

Means and SDs of patient-reported and spouse-reported variables appear in Table 2. Zero order correlations averaged across each of the 70 time points for each person are shown in Table 3. The aggregate concurrent correlations revealed that patient reports of pain and function corresponded to a considerable degree with spouse observations of patient pain and function.

To address issues of validity, correlation coefficients were generated among aggregated state anger, behavioral anger expression, and behavioral anger inhibition scores. State anger scores were related significantly to both behavioral anger expression ($r = .63, p < .01$) and inhibition ($r = .38, p < .01$). Aggregated behavioral anger expression and inhibition scores were not significantly associated ($r = .13, p = .21$).

Scores on the Trait Anger-out Subscale of Spielberger's Anger Expression Inventory (Spielberger & Reheiser, 2004) were not significantly correlated with mean levels of behavioral anger expression ($r = .14, p = .15$), nor with the frequency of behavioral anger expression ($r = .17, p = .08$). Trait Anger-out Subscale scores were significantly associated with greater variability in behavioral anger expression shown by a significant correlation between Trait Anger-out scores and the within-subject standard deviation of behavioral anger expression ($r = .26, p < .01$). Trait Anger-out Subscale scores were also significantly related to each subject's highest state behavioral anger expression score ($r = .28, p < .01$). Results provide some support that the diary behavioral anger expression items were tapping the propensity to express anger.

Scores on the Trait Anger-in subscale (Spielberger & Reheiser, 2004) were significantly associated with mean levels of behavioral anger inhibition ($r = .36, p < .01$), and frequency of behavioral anger inhibition ($r = .22, p < .05$). Trait Anger-in subscale scores were also correlated significantly with greater variability in behavioral anger inhibition, shown by a significant correlation between Trait Anger-in scores and the within subject standard deviation of behavioral anger inhibition ($r = .31, p < .01$), and with each subject's highest state behavioral anger inhibition score ($r = .35, p < .01$).

Patient Variables

Pain Intensity—For concurrent models, patient-reported behavioral anger expression was not significantly associated with pain intensity at the same time point, $B = .02, SE = .02, t = 1.24, p = .21$, controlling for prior pain intensity. Behavioral anger inhibition was also not significantly associated with pain intensity, $B = .02, SE = .01, t = 1.71, p = .09$. However, patient-reported state anger was significantly associated with pain intensity at the same time, $B = .08, SE = .01, t = 16.31, p < .01, f^2 = .05$. For lagged models, greater patient behavioral anger expression was significantly associated with greater patient-reported pain intensity in the three hours following the expression, $B = .04, SE = .01, t = 2.83, p < .01, f^2 = .03$. This relationship remained significant after adjusting for lagged state anger (i.e., anger level at the time of behavioral anger expression), $B = .05, SE = .02, t = 3.00, p < .01, f^2 = .03$. Lagged patient-reported state anger itself was not significantly associated with pain intensity three hours later, $B = .00, SE = .01, t = .86, p = .39$. Behavioral anger inhibition was not significantly associated with pain intensity three hours later, $B = .01, SE = .01, t = 1.89, p = .06$.

Pain Interference—Behavioral anger expression as reported by the patient was not significantly associated with self-reported pain interference at the same time controlling for prior pain interference values, $B = .02, SE = .02, t = 1.18, p = .24$. Greater behavioral anger inhibition was significantly associated with greater self-report pain interference at the same

time, $B = .05$, $SE = .01$, $t = 5.14$, $p < .01$, and this association remained significant after adjusting for current state anger, $B = .05$, $SE = .01$, $t = 5.02$, $p < .01$, $f^2 = .009$. Patient-reported state anger was also significantly associated with pain interference at the same time, $B = .09$, $SE = .01$, $t = 14.67$, $p < .01$, $f^2 = .037$. For lagged analyses, greater behavioral anger expression was significantly associated with greater self-reported pain interference three hours later, $B = .04$, $SE = .02$, $t = 2.15$, $p < .05$, and this relationship remained significant after accounting for lagged state anger, $B = .05$, $SE = .02$, $t = 2.22$, $p < .05$, $f^2 = .002$. Greater behavioral anger inhibition was also significantly associated with greater self-reported pain interference three hours later, $B = .03$, $SE = .01$, $t = 2.22$, $p < .05$, and this association remained significant after adjusting for lagged state anger, $B = .03$, $SE = .01$, $t = 2.22$, $p < .05$, $f^2 = .002$. However, lagged state anger was itself not significantly associated with pain interference three hours later, $B = .00$, $SE = .01$, $t = .09$, $p = .93$.

Rest Because of Pain (Downtime)—Concurrent tests showed that patient-reported behavioral anger expression was not significantly associated with self-reported downtime at the same time, $B = .01$, $SE = .02$, $t = .30$, $p = .77$. Greater behavioral anger inhibition was significantly associated with greater downtime, $B = .03$, $SE = .01$, $t = 2.77$, $p < .01$, and this association remained significant after adjusting for state anger, $B = .03$, $SE = .01$, $t = 2.76$, $p < .01$, $f^2 = .003$. However, patient-reported state anger was not significantly associated with self-reported downtime at the same time, $B = .01$, $SE = .01$, $t = 1.46$, $p = .15$. Lagged tests showed that greater behavioral anger expression was significantly associated with greater self-reported downtime three hours later, $B = .05$, $SE = .02$, $t = 2.21$, $p < .05$, but the relationship was not significant after accounting for lagged state anger, $B = .03$, $SE = .02$, $t = 1.36$, $p = .17$, $f^2 = .001$. The lagged effect for behavioral anger inhibition on later downtime was not significant, $B = -.01$, $SE = .01$, $t = -1.10$, $p = .27$. Lagged patient-reported state anger was also significantly associated with self-reported downtime three hours later, $B = .02$, $SE = .01$, $t = 2.96$, $p < .01$, $f^2 = .003$.

Spouse-Observed Variables

Pain Intensity of Patient Observed by the Spouse

Greater patient-reported behavioral anger expression was significantly associated with greater patient pain intensity as seen by the spouse at the same time, $B = .07$, $SE = .03$, $t = 1.99$, $p < .05$, but the relationship was not significant after adjusting for patient state anger, $B = .05$, $SE = .04$, $t = 1.46$, $p = .15$. Patient-reported behavioral anger inhibition was not significantly associated with concurrent patient pain intensity as judged by the spouse $B = .01$, $SE = .02$, $t = .95$, $p = .35$. State anger was also significantly associated with greater patient pain intensity as seen by the spouse at the same time, $B = .03$, $SE = .01$, $t = 3.78$, $p < .01$, $f^2 = .007$. In lagged analyses, greater patient-reported behavioral anger expression was significantly associated with greater patient pain intensity as seen by the spouse three hours after expression, $B = .06$, $SE = .03$, $t = 2.41$, $p < .05$, $f^2 = .006$. This relationship was no longer significant after adjusting for lagged state anger, $B = .04$, $SE = .03$, $t = 1.58$, $p = .11$. Indeed, lagged state anger was itself significantly associated with greater patient pain intensity as seen by the spouse three hours later, $B = .03$, $SE = .01$, $t = 3.48$, $p < .01$, $f^2 = .006$. Behavioral

anger inhibition reported by the patient was not significantly associated with pain intensity as seen by the patient's spouse three hours later, $B = .02$, $SE = .02$, $t = 1.27$, $p = .20$.

Patient Pain Behaviors Observed by the Spouse

Greater patient-reported behavioral anger expression was significantly associated with a greater frequency of patient pain behaviors observed by the spouse at the same time, $B = .01$, $SE = .01$, $t = 2.26$, $p < .05$, and the relationship remained significant after adjusting for current state anger, $B = .06$, $SE = .03$, $t = 2.07$, $p = .05$, $f^2 = .007$. Greater behavioral anger inhibition was not significantly associated with the frequency of pain behaviors observed by the spouse, $B = .01$, $SE = .02$, $t = .72$, $p = .47$. State anger was also significantly associated with a higher frequency of pain behavior as seen by the spouse at the same time period, $B = .04$, $SE = .01$, $t = 4.42$, $p < .01$, $f^2 = .006$. Patient-reported behavioral anger expression was not significantly associated with the frequency of pain behaviors observed by the spouse three hours later, $B = .00$, $SE = .00$, $t = -.55$, $p = .58$, nor was lagged behavioral anger inhibition, $B = .00$, $SE = .00$, $t = -.11$, $p = .92$. However, lagged state anger was significantly associated with a higher frequency of pain behavior as seen by the spouse three hours later, $B = .03$, $SE = .01$, $t = 3.33$, $p < .01$, $f^2 = .005$.

Patient Activity Level Observed by the Spouse

Behavioral anger expression reported by the patient was not significantly associated with patient physical activity level as observed by the spouse at the same time, $B = -.03$, $SE = .03$, $t = -.81$, $p = .42$, nor was behavioral anger inhibition, $B = -.01$, $SE = .01$, $t = -.83$, $p = .41$, and state anger, $B = .01$, $SE = .01$, $t = -.71$, $p = .48$. Lagged behavioral anger expression was not significantly associated with later patient physical activity level observed by the spouse, $B = .01$, $SE = .03$, $t = -.51$, $p = .61$. However, greater patient behavioral anger inhibition was significantly associated with less patient physical activity observed by the spouse three hours later, $B = -.04$, $SE = .02$, $t = -2.77$, $p < .01$. This relationship remained significant after adjusting for lagged state anger, $B = -.04$, $SE = .02$, $t = -2.77$, $p < .01$, $f^2 = .008$. Lagged state anger itself, however, was not significantly associated with patient physical activity observed by the spouse three hours later $B = .00$, $SE = -.01$, $t = -.40$, $p = .69$.

Tests of Inverse Pathways

Models were reversed so that patient-reported and spouse-observed pain-related variables were used as IVs to predict patient-reported behavioral anger expression and inhibition three hours later. No lagged effects were significant, suggesting that pain and function do not affect subsequent increases in anger regulation. Models were also reversed so that pain-related variables were used as IVs to predict patient reported state anger. Only a single effect was significant. Namely, the lagged effect for patient reported downtime was significantly associated with state anger levels three hours later $B = .06$, $SE = .02$, $t = 2.61$, $p < .01$, $f^2 = .001$.

Discussion

Findings from cross-sectional questionnaire, laboratory, and treatment outcome studies (Bruehl et al., 2006; Burns, Johnson, Devine, Mahoney, & Pawl, 1998; Burns et al., 2008) document relationships between anger-related factors and pain and function for people with chronic pain. Comparatively little is known, however, about whether anger arousal and/or anger regulation strategies in *everyday life* are related to pain and function. Two daily diary studies provide preliminary evidence that anger arousal and behavioral anger expression are related to subsequent pain intensity, and the present study sought to replicate and expand these findings. Analyses focused on longitudinal effects whereby anger arousal and/or anger regulation could affect subsequent pain and function or vice versa. We also focused on isolating unique effects of anger regulation apart from those of state anger. Finally, spouse ratings of patient pain and function gave the advantage of having someone report on patient factors other than the patient.

Increases in patient anger arousal (above their own means) were related concurrently to patient-reported increases in pain intensity and pain interference and to *spouse-reported* increases in patient pain intensity and pain behaviors. These relationships emerged irrespective of how patients regulated their anger at the time, and point to the potential deleterious effects simply becoming angry has on the moment-to-moment pain and function of people with chronic pain. Unique here is that increases in patient pain intensity and pain behaviors were even observed by another person – the patient's spouse. The importance of anger arousal is magnified by significant lagged effects wherein increases in patient anger arousal (above their own mean) at, for example, 9:00 am predicted increases in patient-reported downtime and spouse-reported increases in patient pain intensity and pain behaviors at 12:00 pm. Thus, anger arousal may be related not only to pain and function at the moment anger increases, but may also exert delayed detrimental effects on pain and function; again, effects that may be evident to another person.

Regarding the regulation of anger, present findings are consistent with those of Bruehl et al. (2012) in that lagged analyses in the present study revealed that increases in patient behavioral anger expression (above their own mean) were related to increases in pain intensity three hours later. Extending previous work, we also show for the first time that behavioral anger expression was related to increases in interference due to pain and downtime three hours later. That is, for example, patient behavioral anger expression at 9:00 am predicted patient pain severity, interference and downtime at 12:00 pm. Of note, behavioral anger expression was not related significantly to any patient-reported factors at the same time as the expression episode. Testing the inverse pathways (i.e., pain → anger) did not produce a single significant effect, suggesting that previous changes in pain and function did not increase the likelihood of later anger expression. Taken together, null findings for concurrent effects and inverse pathways coupled with significant lagged effects in the direction of anger → pain suggest that patient regulation of anger via overt expression adversely influences a range of factors reflecting patient well-being, but does so, unlike initial arousal of anger, primarily *after* the episode has occurred.

The lagged effects for behavioral anger expression remained largely significant when effects of increased state anger (above one's own mean) at the time of the anger expression episode were statistically controlled. That is, for example, patient behavioral anger expression at 9:00 am predicted patient pain severity and interference at 12:00 pm even after controlling for degree of anger arousal at 9:00 am. Controlling lagged state anger allowed us to identify unique effects of expressing anger on pain and function beyond the effects exerted by anger arousal; analyses unique to this report. If the behaviors associated with regulating anger via overt expression are at least partly the culprits, then it can be expected that these behaviors would reveal unique effects. This we showed for pain intensity and pain interference, but not for downtime. The effect for downtime became nonsignificant when controlling for lagged state anger levels. However, it should be noted that controlling for state anger represented a conservative and stringent approach. Given that anger arousal is a core and necessary constituent of the construct or phenomenon "anger regulation," and that a certain level of anger arousal was needed to trigger the anger regulation questions on the PDA, findings that a formerly significant relationship between anger expression and a pain-related factor would become nonsignificant are not surprising. Consider the significant lagged effect of state anger on later downtime described above. One way to interpret the result that behavioral anger expression did not exert a significant unique effect on later downtime is that the magnitude of anger experienced during the behavioral anger expression episode and the anger expression itself worked in conjunction to increase later downtime.

Consistent with findings for state anger, patient-reported increases in behavioral anger expression were also related to *spouse* observations of increases in patient pain intensity and frequency of pain behaviors at the same time as the anger expression episode. Thus, patient-reported anger expression at 12:00 pm was related to spouse judgments that patients were experiencing increased pain intensity and were showing more pain behaviors also at 12:00 pm. Of note, a significant lagged relationship indicated that patient-reported anger expression at, for example, 9:00 am was related to spouse judgments that patients had increased pain intensity at 12:00 pm. The concurrent link between patient-reported anger expression and spouse-reported patient pain behaviors remained significant after controlling for state anger. Patient behavioral anger expression did not, however, have significant unique concurrent or lagged relationships with pain intensity when current and lagged state anger were statistically controlled. As above, consider the significant concurrent and lagged effects of patient state anger on spouse-reported patient pain intensity and pain behaviors. Taken together, these could be taken to mean that the magnitude of patient anger experienced during the behavioral anger expression episode and the anger expression itself worked in conjunction to affect what the spouse saw.

Behavioral anger inhibition (above patient's own average) was related significantly to increased pain interference and downtime at the same time that anger was being inhibited. The lagged effect for pain interference was also significant, indicating that behavioral anger inhibition was related to greater pain interference three hours later. These relationships remained significant after concurrent and lagged state anger levels were controlled, suggesting that the anger inhibition behaviors are related to adverse patient factors apart from the simple arousal of anger. That is, behavioral anger inhibition at 12:00 pm was

related to greater pain interference and downtime at 12:00 pm while controlling for state anger at 12:00 pm. Also, patient-reported anger inhibition at, for example, 9:00 am was related to increased pain interference at 12:00 pm while controlling for state anger at 9:00 am. The concurrent effects suggest that anger inhibition was related to worsening patient pain and function at the time anger is inhibited. However, any causal inference is greatly tempered in concurrent analyses, and so it appears just as likely that increases in pain interference and downtime could increase the likelihood that anger is inhibited. Still, these results, especially the significant lagged effect on pain interference, are consistent with experimental work which showed that anger suppression manipulated in the lab predicted greater pain intensity and even observable pain behaviors during subsequent acute pain induction (Burns et al., 2008). It should be noted that, like behavioral anger expression, results show relationships to patient function beyond just pain intensity. Here, behavioral anger inhibition was related to reports of pain-related interference to perform everyday activities and to downtime. Results add to what we know about the possible deleterious effects of inhibiting anger on the pain and function of chronic low back pain patients.

Significant relationships between patient-reported behavioral inhibition and spouse observations of patient pain and function were limited to a single lagged effect for physical activity level. That is, patient-reported anger inhibition at, for example, 9:00 am was related to spouse judgments that patients were *less* physically active at 12:00 pm, even while controlling for patient reported state anger at 9:00 am. These results coupled with results for anger expression suggest a relatively low correspondence between relationships between patient reported anger regulation and pain and function and relationships between patient anger regulation and spouse judgments of patient pain and function. Consider that the most concordance between patient and spouse ratings exists between relationships of patient reported *anger arousal* and concurrent and lagged pain and function. Moreover, relationships between patient reported behavioral anger expression and concurrent and lagged spouse judgments of patient pain intensity were rendered nonsignificant by controlling for state anger. Results imply that the manner in which anger is regulated adds to the effects on *patient* perceptions of adverse changes in pain and function, but that simple increases in patient anger arousal may drive much of the effects on patient pain and function that can be observed by *another person*. Of note, and relevant to the purpose of examining spouse ratings, these findings are unique and strengthen claims that anger arousal and its regulation contribute negatively to patient pain and function because anger-related changes in patient pain and function were evident to an external observer.

Some limitations need be delineated. First, we did not examine potential mechanisms through which anger arousal and anger regulation may exert detrimental effects on patient pain and function. As described above, a number of laboratory studies have begun to document mechanisms by which anger arousal, and anger expression and inhibition may influence pain and function among chronic pain sufferers. The theoretical models postulated by these investigators and their findings guided us to hypothesize that anger arousal and both anger expression and inhibition would be related to increases in pain and decreases in function. But explicit tests of changes in endogenous opioid function and/or low back muscle tension using ambulatory monitoring in conjunction with electronic diary

assessments must await future research. Second, we used only two items to assess behavioral anger expression and two items to assess behavioral anger inhibition. These items were adapted from items from Spielberger's widely used and validated trait measures of anger-out and anger-in (Spielberger & Reheiser, 2004), and we reported (above) initial psychometric data for them. Still, these data represent preliminary steps to assess reliability and validity, and caution should be taken when interpreting findings. Third, the three hour lags in assessments may have been too long to capture all of the effects of anger regulation on later pain and function. More frequent assessments, although perhaps burdensome to participants, may be needed to flesh out these relationships. Conversely, we confined analyses to three hour lags, as opposed to, say, six or nine hour lags, which we chose in order to focus on events linked relatively closely in time and to limit the number of analyses. Examination of longer lags may, again, be needed to fully understand these relationships. Finally, the magnitudes of the effect size coefficients we report suggest that the effects we found were small. On one level, effect size estimation in hierarchical linear modeling continues to evolve, and guidelines on how to interpret these effects emerging out of many within-person observations have not been established. On another level, these small effects occurring frequently throughout the day may be critical. While strong episode of anger arousal and/or vehement expression thereof may exert larger effects, less emotionally charged and short-lived episodes of patient anger may represent oft-repeated small "injuries," and may be of great importance in slowly eroding patient function.

In sum, results replicate past findings and extend knowledge of the effects of anger-related factors on the pain and function of those suffering chronic low back pain, and indicate that anger arousal and behavioral anger expression and inhibition in everyday life are related to elevated pain intensity, pain interference and downtime as reported by patients. Supporting the validity of these findings, spouse judgments of patient pain intensity and pain behavior during and following anger arousal and anger regulation episodes showed some concordance with reports of patients. Results from cross-sectional questionnaire, lab-based, treatment outcome and now daily diary studies all point to the potential clinical relevance of altering problematic anger-related factors in the service of improving patient pain and function. Although outcome research on anger reduction lags behind that on other emotional problems, results of meta-analyses indicate that CBT-based anger reduction interventions have moderate to strong effects on outcomes ($d = .70$ to 1.0 ; Beck & Fernandez, 1998). Extension of these techniques to treating problematic anger and anger regulation among chronic pain patients may prove profitable. However, only one study to our knowledge has attempted to alter anger-related factors in order to improve patient pain and function (Slavin-Spenny, Lumley, Thakur, Nevedal, & Hijazi, in press). Of note, our results indicate that *both* increases in behavioral anger expression *and* inhibition have negative relationships with patient pain and function. The application and extension of anger management techniques to treating problematic anger and anger regulation for chronic pain patients may need to simultaneously consider control of anger arousal, reducing overt expressive behaviors, and increasing anger awareness to reduce inhibition.

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Table 1

Demographic Characteristics

	Patient	Spouse
Gender (female)	48.6% (n = 51)	51.4% (n = 54)
Age in years (M,SD)	46.30 (12.1)	45.96 (13.2)
Hispanic	4.8% (n = 5)	5.7% (n = 6)
African American	15.2% (n = 16)	18.1% (n = 19)
Caucasian	80.0% (n = 84)	76.2% (n = 80)
Employed	40.0% (n = 42)	63.8% (n = 67)
Disability Insurance	34.3% (n = 36)	13.3% (n = 14)
Length of Marriage (M,SD)	14.30 (14.0)	—
Pain Duration (M,SD)	9.04 years (7.8)	—

Table 2

Means and Standard Deviation of Key Study Variables

Variable	M	SD
State Anger	3.51	3.59
Anger Inhibition	4.90	3.41
Anger Expression	1.24	1.18
Pain Intensity	3.09	1.63
Pain Interference	2.69	1.86
Downtime	2.43	1.48
Spouse-Observed Pain Intensity	2.49	1.49
Spouse-Observed Pain Behavior	2.08	1.44
Spouse-Observed Activity	2.43	1.09

Table 3

Zero-Order Correlations Between Averaged Levels of Anger and Pain Variables

	1	2	3	4	5	6	7	8	9
1. State Anger	1.00	0.63	0.38	0.54	0.49	0.39	0.39	0.38	0.05
2. Anger Expression		1.00	0.13	0.26	0.21	0.08	0.22	0.17	0.03
3. Anger Inhibition			1.00	0.30	0.28	0.08	0.21	0.16	-0.17
4. Pain Intensity				1.00	0.85	0.62	0.68	0.56	-0.06
5. Pain Interference					1.00	0.72	0.71	0.58	-0.07
6. Downtime						1.00	0.60	0.44	-0.11
7. Spouse-observed Pain Intensity							1.00	0.83	-0.05
8. Spouse-observed Pain Behaviors								1.00	-0.06
9. Spouse-observed Activity									1.00

Note. All coefficients greater than .20 are statistically significant at $p < .05$. All coefficients greater than .26 are statistically significant at $p < .01$.