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Associations between daily chronic pain intensity, daily anger expression, and trait anger expressiveness: An ecological momentary assessment study

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Abstract

Links between elevated trait anger expressiveness (anger-out) and greater chronic pain intensity are well documented, but pain-related effects of expressive behaviors actually used to regulate anger when it is experienced have been little explored. This study used ecological momentary assessment methods to explore prospective associations between daily behavioral anger expression and daily chronic pain intensity. Forty-eight chronic low back pain (LBP) patients and 36 healthy controls completed electronic diary ratings of momentary pain and behavioral anger expression in response to random prompts 4 times daily for 7 days. Across groups, greater trait anger-out was associated with greater daily behavioral anger expression ($P < 0.001$). LBP participants showed higher levels of daily anger expression than controls ($P < 0.001$). Generalized estimating equation analyses in the LBP group revealed a lagged main effect of greater behavioral anger expression on increased chronic pain intensity in the subsequent assessment period ($P < 0.05$). Examination of a trait $\times$ situation model for anger-out revealed prospective associations between elevated chronic pain intensity and later increases in behavioral anger expression that were restricted largely to individuals low in trait anger-out ($P < 0.001$). Trait $\times$ situation interactions for trait anger suppression (anger-in) indicated similar influences of pain intensity on subsequent behavioral anger expression occurring among low anger-in persons ($P < 0.001$). Overlap with trait and state negative affect did not account for study findings. This study for the first time documents lagged within-day influences of behavioral anger expression on subsequent chronic pain intensity. Trait anger regulation style may moderate associations between behavioral anger expression and chronic pain intensity.

Keywords

Chronic pain; Anger-out; Anger-in; Anger expression; Pain intensity; Electronic diary; Ecological momentary assessment

Conflict of interest statement

The authors have no conflicts of interest to report.

1. Introduction

The pain-related effects of how anger is regulated have received increasing research attention. The tendency to regulate anger via direct verbal or physical expression, termed trait anger-out, has been the focus of more than 25 cross-sectional studies [4]. Results consistently indicate that greater trait anger-out is associated with elevated acute and chronic pain (CP) responsiveness [6,8,10,11, 14,15,20,27,34,37,43,48]. However, nearly all of these studies have only examined pain-related effects of a self-reported predisposition to express anger rather than testing what is the presumed core of the anger-out construct: the pain-related effects of expressive behaviors actually used to regulate anger when experienced.

The few studies exploring the impact of such behavioral anger expression on pain responses have taken a “trait $\times$ situation” perspective [5,19,57]. For example, Burns et al. [19] found somewhat unexpectedly that behavioral anger expression produced analgesic rather than hyperalgesic effects on experimental acute pain responses in healthy individuals, although this apparent analgesic effect only occurred in high trait anger-out individuals. A more recent study reported a similar effect, although greatly extended these results to links between behavioral anger expression and CP intensity in daily life [57]. In a diary study examining female fibromyalgia patients [57], anger regulation strategies used to manage one significant daily emotional event were retrospectively recorded at the end of each day along with end-of-day pain ratings. Findings indicated that trait anger-out and behavioral anger expression appeared to interact, with the lowest pain intensity among high anger-out individuals who behaviorally expressed anger [57]. Taken together, findings from a handful of studies suggest a trait $\times$ situation model, in which individuals elevated in trait anger-out might reduce acute and chronic pain responsiveness through the behavioral expression of anger.

Although the results of van Middendorp et al. [57], in particular, are intriguing, these issues have never been examined using state-of-the-art ecological momentary assessment (EMA) methodology that incorporates repeated random sampling of both CP intensity and behavioral anger expression over the course of a day. EMA methodology permits examination of concurrent and bidirectional lagged relationships between pain intensity and behavioral anger expression over relatively brief periods within a day, potentially clarifying causal direction of previously observed associations. The present study was designed to provide the first test of prospective associations between daily behavioral anger expression and daily pain intensity in a CP sample using EMA methodology.

The study had 3 primary aims: (1) to determine whether patients with CP differ from healthy controls in their level of daily behavioral anger expression, (2) to determine what effect anger expressive behaviors occurring spontaneously in the course of daily life have on subsequent CP intensity, and (3) to determine whether a trait $\times$ situation model is supported, that is, whether trait anger-out moderates associations between daily behavioral anger expression and daily CP intensity. A secondary aim of the study was to provide information to help characterize the extent to which behavioral anger expression is associated with gender, general negative affect, and trait anger-out.

2. Methods

2.1. Design

This study employed a prospective mixed between/within-subjects design incorporating EMA diary assessment techniques with repeated daily sampling of target variables. The 2 primary within-subject target variables were CP intensity over time and behavioral anger

expression over time. A between-subject trait anger-out variable was also incorporated in the study design to permit evaluation of the trait $\times$ situation model.

2.2. Participants

Participants included 48 individuals with chronic low back pain (LBP) and 36 healthy controls (Healthy). All participants completed this electronic diary study as part of their participation in a laboratory study of endogenous opioid mechanisms contributing to associations between anger and pain [5]. The sample size in the current study was reduced from the original study due to loss of diary data from the first 93 consecutive participants in the laboratory study as the result of a data storage error (master database was accidentally overwritten with a blank database during installation of a minor software revision). Participants with and without diary data did not differ significantly in healthy vs chronic pain status, race, ethnicity, baseline chronic pain intensity, age, depression (Beck Depression Inventory [BDI]), anxiety (State-Trait Anxiety Inventory [STAI]), or anger-out scores. Participants with diary data (ie, those participating later in the laboratory study) were significantly ($P < 0.001$) more likely to be male due to selective oversampling of males later in the primary study to help insure overall gender balance in the final laboratory study sample.

All individuals from the original study with diary data available were included in this study. Inclusion of both Healthy and LBP participants permitted this study to examine possible effects of LBP on daily anger expression. All participants were recruited through on-line advertisements on the Vanderbilt University e-mail recruitment system or advertisements in local print media. General criteria for participation included age 18–55 years; no self-reported history of cardiovascular disease, hypertension, liver or kidney disorders, posttraumatic stress disorder, diabetes, seizure disorder, or opiate dependence; no use of antihypertensive medications; and no daily use of opioid analgesics (confirmed via urine opiate screen before each laboratory study session, ie, at the beginning and end of the diary assessment period). As in our past studies [6,12], additional inclusion criteria for the LBP group were chronic daily low back pain of at least 3 months' duration with an average past month (retrospective) severity of at least 3/10. Potential participants who were pregnant (determined by urine pregnancy screens) were excluded due to the study drug used in the laboratory portion of the study. No participants in either group were taking neuroleptic medications, antidepressants, or as-needed opioid analgesics.

Characteristics of both study subgroups are summarized in Table 1. Although both groups were largely of non-Hispanic Caucasian ethnicity/race, Healthy controls were significantly more often of non-Hispanic ethnicity than were LBP participants. Gender distribution and age across groups were also significantly different, with the LBP participants significantly older than Healthy participants, and more likely to be male. Both groups were similar in terms of trait anger-out. However, LBP participants had significantly higher scores on the STAI [49] and BDI [3]. Both groups were nonetheless in the nondepressed range on the latter measure. The low level of depressive symptoms in the LBP group was due to selection criteria for the primary study, particularly the exclusion of patients taking daily opioid analgesics, which resulted in a sample with less severe pain and less pain-related interference and distress than might be observed in a typical tertiary care pain clinic setting.

2.3. Psychometric measures

To assess trait anger-out, participants completed the Spielberger Anger Expression Inventory (SAEI) [50]. The SAEI includes a subscale assessing anger-out (managing anger through direct verbal or physical expression), as well as a subscale assessing anger-in (managing anger via suppression). This anger-out scale has demonstrated previous

associations with measures of acute and chronic pain (see ref. [4] for a review). Participants were also asked to complete the trait form of the STAI [49] and the BDI [3] for use in analyses as controls for potential confounding effects of trait negative affect, as in our past work [6,8,14,17,18].

2.4. Diary assessment of pain and behavioral anger expression

Electronic pain diaries were used that employed signal contingent sampling, in which participants were randomly signaled during their normal daily activities to provide momentary ratings of pain and behavioral anger expression. The EMA diary methods used in this study were intended to minimize retrospective bias [54], reduce reactivity effects to monitoring [24,32], and permit the accurate capture of intra-day variability [45,51]. All participants completed the electronic pain diary for the 7 days between the 2 laboratory sessions in the primary study.

Diary data were obtained using HP IPAQ rx3715 handheld computers (Hewlett Packard, Palo Alto, CA, USA) running custom software (Pain Electronic Calendar, New England Research Institute, Watertown, MA, USA) [31,39-41]. Similar to Peters et al. [45], participants were prompted 4 times per day by an audible alert that occurred randomly within 4 predetermined time frames between 8:00 AM and 10:00 PM (corresponding to morning, mid-day, late afternoon, and evening). At each prompting, participants were asked to use a stylus to electronically record ratings of their current pain on a visual analog scale (VAS) anchored with 0 = “No Pain” and 10 = “Worst Possible Pain”. At each prompting, participants were also asked to rate using a VAS the extent to which they were experiencing 4 emotions (Anger, Fear, Anxiety, and Happiness). Each emotion was tapped by 3 items consisting of an emotional word stem (eg, “Angry”, “Mad”, and “Annoyed” for Anger) rated using a VAS scale anchored with 0 = “None” and 10 = “The Most Possible” [23]. Given the focus of the study on daily behavioral anger expression, a branching algorithm was incorporated in the diary software. When any single anger subscale item was rated as non-zero (operationalized as 5% or more beyond the left anchor), the diary branched to 9 anger expression items asking: “When recently feeling angry, mad, or annoyed, to what extent did you do the following?” Items on this behavioral anger expression subscale paralleled (with minor wording modifications as necessary) the anger-out items on the SAEI. Each was rated on a VAS scale identical to that used for rating emotional state. These 9 items were summed as in the anger-out scale of the SAEI, and were used as a summary index of momentary behavioral anger expression. Pain and behavioral anger expression diary variables reported below were rescaled to range from 0-100.

Because of the exclusive focus of this study on pain-related effects of how anger is regulated (ie, behavioral anger expression and trait anger regulation style) rather than effects of emotional state in general, VAS data for daily emotional state as they relate to daily pain are not detailed here in the interests of space. Within the chronic pain group who were the focus of the longitudinal diary analyses, 2 subjects failed to respond to one data prompt each, and one subject failed to respond to 2 assessment prompts, for an overall response rate of >99% (4 out of 1165 total assessment points).

2.5. Procedure

All procedures were approved by the university Institutional Review Board. After providing written informed consent, participants provided demographic information and completed the psychometric screening packet, including the SAEI, STAI, and BDI. Participants then engaged in a laboratory pain assessment protocol detailed elsewhere [5]. At the conclusion of the first laboratory session, participants were provided with the electronic diary and instructions were provided, including completion of a sample diary entry (omitted from

analyses). Participants were asked to carry the diary with them throughout the day for the 7 days between the 2 laboratory sessions, and respond to the diary items as soon as possible when prompted.

2.6. Statistical analysis

Basic demographic characteristics and anger expressive behaviors were compared between control and LBP groups using χ^2 tests or t -tests, as appropriate. Primary analyses of longitudinal associations between measures of momentary behavioral anger expression and momentary pain ratings (in the LBP group only) were conducted using Generalized Estimating Equation (GEE) methods, which take into account the correlated repeated measurements within each participant. Autoregressive AR (1) correlation structure was specified to allow more precise estimates, although GEE is fairly robust against a wrong choice of correlation structure [36]. We applied a generalized log link function associated with GEE to assess the effects of concurrent, prior, and subsequent anger expression as potential predictors of current pain ratings. The latter can be interpreted as reflecting the influence of current pain on subsequent behavioral anger expression. The main analytic model can be expressed by the following:

$$PainRating_{it} = \alpha + \beta_1 AngerExp_{it} + \beta_2 AngerExp_{i(t-1)} + \beta_3 AngerExp_{i(t+1)}$$

Where $PainRating_{it}$ represents the reported pain score for participant i on time t , $AngerExp_{it}$ indicates the anger reported on time t , which is the current level of behavioral anger expression, $AngerExp_{i(t-1)}$ indicates the behavioral anger expression reported at time $t-1$ (prior anger expression), $AngerExp_{i(t+1)}$ indicates the behavioral anger expression reported on time $t+1$ (subsequent anger expression), and β_1 , β_2 , and β_3 are regression coefficients defining the effects of behavioral anger expression at different times.

In a subsequent analysis, the hypothesized trait $\times$ situation model was tested using a GEE model incorporating main and interactive effects of both daily behavioral anger expression and trait anger-out on daily chronic pain intensity. Given possible trait $\times$ situation mismatch effects for trait anger suppression (anger-in) and behavioral anger expression, a comparable GEE analysis was also conducted to examine interactions between these 2 variables.

Due to known conceptual and statistical overlap between general negative affect and anger regulation style [6,17,18,42], significant models in analyses above were re-run including BDI and STAI values as covariates to evaluate the possibility that results for anger regulation variables could be accounted for by trait negative affect confounds. Similarly, significant models were also re-run using the appropriate state anger and state negative affect (mean of the Anxiety and Fear subscales) variables from the diary data to control for potential confounding effects of these 2 state variables on associations between anger regulation and pain.

All time effects in GEE analyses were found to be nonsignificant and did not alter interpretation of primary results; therefore, they are not detailed. Throughout, a 2-tailed probability value of $P < 0.05$ was used as the criterion for statistical significance. All statistical analyses were carried out using SAS version 9.3 (SAS Institute Inc, Cary, NC, USA).

3. Results

3.1. Characteristics and correlates of daily behavioral anger expression

In the combined samples, mean intensity of behavioral anger expression during episodes of anger arousal was relatively low (Mean 15.6, SD 13.8; Min 0.5, Max 92.2). When averaged over all data collection periods (ie, anger expression coded as 0 for periods with no anger arousal), mean daily behavioral anger expression levels, not surprisingly, were lower (Mean 7.0, SD 11.6). Pearson correlations revealed significant positive associations between mean daily behavioral anger expression and trait anger-out ($r = 0.41$, $P < 0.001$), BDI scores ($r = 0.30$, $P = 0.005$), and STAI scores ($r = 0.39$, $P < 0.001$), but no significant association with trait anger-in ($r = 0.17$, $P = 0.12$). Nonparametric tests indicated that mean daily behavioral anger expression did not differ significantly as a function of gender in the combined samples (Wilcoxon 2-sample test: $z = -1.75$, $P = 0.08$.) or within the LBP subsample alone (Wilcoxon 2-sample test: $z = -0.68$, $P = 0.5$).

Effects of trait anger-out on daily behavioral anger expression were also subjected to analysis within a GEE model. Results indicated that trait anger-out was a significant predictor of daily behavioral anger expression for the entire sample ($\beta = 0.212$, $SE = 0.021$, $P < 0.001$). Given the significant correlations above, this GEE analysis was then re-run with BDI and STAI scores included as covariates to determine whether observed links between trait anger-out and behavioral anger expression might be accounted for by common influences of general negative affect. Trait anger-out remained a significant predictor even after statistically controlling for trait negative affect ($\beta = 0.154$, $SE = 0.019$, $P < 0.001$). A significant independent effect of elevated trait anxiety (STAI) as a predictor of greater daily behavioral anger expression was noted in this model ($\beta = 0.047$, $SE = 0.020$, $P = 0.02$), although effects for the BDI were nonsignificant ($P = 0.45$).

3.2. Does daily anger expression differ by chronic pain status?

Average levels of daily anger expression across all assessment periods were low in both study subgroups in absolute terms, but were significantly higher in participants with LBP than in healthy participants (LBP: 8.8 ± 14.98 ; Healthy: 2.0 ± 3.04 ; $t = 6.36$, $P < 0.001$). This effect remained significant even after statistically adjusting for the influence of gender differences (Estimated Marginal Means: LBP = 9.72, Healthy = 3.55; $F = 5.55$, $P < 0.05$). Similar group differences as a function of chronic pain status were also noted in terms of mean intensity of anger expression reported during actual anger episodes (LBP: 17.9 ± 14.98 ; Healthy: 12.9 ± 11.95 ; $t = 10.41$, $P < 0.001$). The maximum observed anger expression value was notably higher in the LBP group (92.2) than in the Healthy group (57.8).

3.3. Main effects of daily behavioral anger expression on daily chronic pain

Mean daily back pain intensity in the LBP group was relatively mild, but variable (23.0 ± 28.23 ; Max = 88). A simple GEE model examining main effects of behavioral anger expression on daily CP intensity revealed a significant lagged effect for current level of behavioral anger expression on subsequent CP intensity ($\beta = 0.003$, $SE = 0.002$, $P < 0.05$; partial $r = 0.12$). Participants engaging in higher levels of anger expressive behavior were found to report greater subsequent back pain intensity in the hours immediately following that anger expression. Concurrent associations between anger expression and daily pain ($P = 0.84$) and lagged effects of pain on subsequent anger expression ($P = 0.11$) were nonsignificant. This GEE analysis was re-run with BDI and STAI scores included as covariates to determine whether lagged effects of behavioral anger expression on subsequent pain intensity might be accounted for by common influences of general trait negative affect. Behavioral anger expression remained a significant predictor of subsequent chronic pain

intensity even after statistical control for trait negative affect ($\beta = 0.004$, $SE = 0.002$, $P < 0.05$). Similar re-analyses to examine potentially confounding influences of momentary emotional state indicated that statistical control of lagged state anger levels actually increased the magnitude of the lagged daily anger expression effect ($\beta = 0.005$, $SE = 0.002$, $P < 0.001$). Finally, simultaneous control for trait negative affect (BDI, STAI), lagged state negative affect, and lagged state anger also did not alter the significant findings, indicating that elevated daily anger expression predicted greater subsequent chronic pain intensity ($\beta = 0.006$, $SE = 0.002$, $P < 0.001$).

For comparison with results for behavioral anger expression above, effects of trait anger expressiveness (anger-out) were also examined in a simple main effects GEE model. Results indicated no significant main effect of trait anger-out on daily pain intensity alone ($P = 0.86$) or with influences of trait negative affect statistically controlled ($P = 0.97$).

3.4. Trait $\times$ situation models

Table 2 summarizes the results for the GEE analysis examining a trait $\times$ situation model for trait anger-out. Results revealed a significant main effect for elevated chronic pain intensity on subsequently increased behavioral anger expression, although this main effect was superseded by a significant trait anger-out $\times$ subsequent anger expression interaction. This interaction indicated that trait anger-out moderated the influence of pain intensity on subsequent behavioral anger expression. Fig. 1 displays the source of this interaction, revealing that the greatest behavioral anger expression was observed when low anger-out individuals experienced the most intense back pain. This significant trait $\times$ situation interaction for trait anger-out remained essentially unchanged after statistically controlling for influence of general trait negative affect. Control for concurrent and lagged state anger levels also did not alter the pattern of significant trait anger-out $\times$ daily anger expression findings above ($\beta = 0.0007$, $SE = 0.0001$, $P < 0.0001$). There was no evidence for a trait $\times$ situation model involving influences in the opposite direction (effects of behavioral anger expression on subsequent pain intensity moderated by trait anger-out).

A comparable trait $\times$ situation model for trait anger-in was also evaluated, given the possibility of trait $\times$ situation mismatch effects in which the highest pain might occur in those predisposed to suppress anger but forced by circumstances to express it [16,22]. These results are also summarized in Table 2. Within this model, 2 key effects emerged. Greater behavioral anger expression predicted significantly increased pain in the following assessment period, as in the simple main effects GEE model described previously. In addition, trait anger-in was found to moderate an influence of greater pain intensity on subsequent behavioral anger expression. The negative beta weight for this latter interaction indicates that the link between elevated pain and greater subsequent behavioral anger expression was strongest in those with the lowest predisposition to suppress anger (low trait anger-in). All significant effects in the trait $\times$ situation model for trait anger-in remained unchanged after statistically controlling for effects of general trait negative affect. Control for concurrent and lagged state anger levels also did not alter the pattern of significant trait anger-in $\times$ momentary anger expression findings ($\beta = 0.0007$, $SE = 0.0001$, $P < 0.0001$).

4. Discussion

Pain-related effects of the tendency to regulate anger via direct verbal/physical expression (trait anger-out) have been widely explored, indicating that elevated trait anger-out is associated with greater pain responsiveness [4]. However, pain-related effects of the presumed core of the anger-out construct, expressive anger regulatory behaviors used in anger-provoking situations, have received little direct investigation. Only one previous diary study has examined the effects of behavioral anger expression on CP intensity in daily life,

assessing pain and anger expression once daily [57]. Building on this work, the current study used electronic EMA diary methodology [52,55,56] to randomly sample pain intensity and behavioral anger expression multiple times daily, thereby permitting for the first time exploration of prospective associations between behavioral anger expression and chronic back pain intensity over relatively short within-day periods.

The first key finding was that in a main effects model, greater behavioral anger expression predicted subsequent increases in CP intensity during the hours immediately following that expression. Moreover, these effects were due to anger expression itself rather than momentary levels of anger or other negative affective states being experienced at the time. Findings of lagged associations between behavioral anger expression and increased subsequent pain intensity are consistent with the large cross-sectional literature reporting positive associations between trait anger-out and pain responsiveness [6,8,10,11,14,15,20,27,34,37,43,48]. Past work examining associations between pain intensity and behavioral anger expression assessed once per day via paper diary [57] did not permit determination of whether anger expression causally influenced pain within days, given that once-daily ratings effectively averaged over all periods within-day. The current findings therefore represent an important advance in understanding the nature of behavioral anger expression-pain links.

The second key finding relates to the trait $\times$ situation model for trait anger-out proposed to explain results of 2 previous studies. It was suggested that for people high in trait anger-out, behavioral anger expression might produce analgesia rather than hyperalgesia in the context of daily chronic pain [57] and laboratory acute pain [19]. The current study did support a trait $\times$ situation model, but not the one suggested by this prior work. When interactions between trait anger-out and behavioral anger expression as they related to daily pain intensity were examined, results suggested that elevated pain had little impact on behavioral anger expression levels among high anger-out individuals already prone to behaviorally express anger, but that elevated pain made low trait anger-out individuals act like high anger-out individuals by increasing their degree of anger expression. In effect, elevated CP intensity may change a person who normally expresses little anger into someone who might be described as “having a bad temper,” a change potentially associated with negative effects in the family and workplace. This is consistent with clinical findings that CP patients reported greater irritability when they experience prolonged pain flareups, even though they were by nature even-tempered and rarely “lost their temper” prior to the onset of their pain [30,58].

This interpretation is supported by another finding of this study. CP itself appeared to be associated with greater levels of daily behavioral anger expression compared to healthy controls. We considered that significant differences in gender composition between groups might have confounded these comparisons, particularly given suggestions that social constraints on anger expression may be greater in women [46]. However, analyses indicated that gender did not exert a significant effect on daily behavioral anger expression, nor did statistical control of gender alter results demonstrating significantly greater behavioral anger expression in the group with CP. These observations of links between CP and greater direct anger expression are consistent with suggestions that anger in CP patients is common and often directed at external targets [44].

Although not a primary study focus, we also considered the possibility of trait $\times$ situation effects involving trait anger suppression (anger-in). Based on trait $\times$ situation mismatch models [25], we hypothesized that individuals who tend to suppress anger (high trait anger-in), when forced by circumstances to express their anger (high behavioral anger expression), might in such situations experience the most intense pain. The direction of effects did not

support this trait $\times$ situation mismatch hypothesis. Rather, an association between elevated pain and greater subsequent behavioral anger expression was strongest in those least inclined to suppress anger (low trait anger-in). This finding is somewhat surprising given the trait $\times$ situation pattern observed for trait anger-out. Taken together, results seem to suggest that individuals who tend not to use high levels of suppression or direct expression to regulate anger show the largest increases in anger-expressive behaviors when experiencing increased pain. Trait anger-out and anger-in are proposed to be orthogonal dimensions [50], and both have shown positive associations with CP intensity in past work [4,21]. Finding of a similar pattern of trait $\times$ situation influences related to daily pain intensity for both dimensions might therefore not be entirely unexpected, although underlying reasons remain to be explained.

Several previous laboratory studies support an absolute opioid dysfunction model to explain positive correlations between trait anger-out and pain, with underlying opioid system dysfunction hypothesized to contribute simultaneously to both elevated pain intensity and impaired ability to modulate anger expression [9]. Present findings are somewhat consistent with this model. Greater opioid dysfunction in CP patients [1,7,13,28,35,38] might produce impaired ability to modulate anger and therefore greater overt behavioral anger expression, in line with the greater anger expression noted in CP than Healthy participants in the current study. Moreover, impaired opioid modulatory function, particularly if exhibiting within-day variability, might also serve as a common substrate for main effect associations between momentary behavioral anger expression and subsequent back pain intensity. However, EMA studies using long-acting opioid antagonists would be necessary to directly test this hypothesis.

Results of this study provide additional information to help characterize the little-explored construct of behavioral anger expression. It is related positively to general negative affect, but as in several previous studies, its pain-related effects are not accounted for by overlap with negative affect [6,8,14,17,18,42,57]. Somewhat surprisingly, only 2 prior studies have reported on associations in daily life between trait anger-out and behavioral anger expression. However, outside the pain context [46], a moderately large association was reported between anger-out and daily behavioral anger expression ($r = 0.48$) similar to the current study ($r = 0.41$). Overall, available diary data, combined with laboratory findings that greater trait anger-out predicted greater verbally expressed anger during provocation [26], do support the construct validity of the trait anger-out scale.

Nonetheless, absence of main effect associations between trait anger-out and daily pain intensity in the current study was somewhat surprising, given relatively consistent findings of such associations in cross-sectional clinical studies [4]. It is unknown whether this might be the result of the cross-sectional literature over-estimating the frequency of these associations due to “file drawer effects” (nonpublication of negative findings), or rather, whether some unique characteristic of the current sample might have accounted for these inconsistencies. The bimodal distribution for anger-out/pain associations suggested by Fig. 1 (z axis) supports the latter. The present study clearly indicates that trait anger-out and behavioral anger expression are not identical constructs, as low anger-out persons sometimes engaged in high levels of behavioral anger expression when pain levels were higher. The latter fits with retrospective findings that direct verbal aggression is common, occurring in 49% of anger episodes [2].

Several potential study limitations must be acknowledged. The sample size was not large, and therefore, inadequate power might have influenced null results in some tests, although power issues would not have affected interpretation of the key positive findings reported. The reason for this somewhat limited sample size is another potential concern. It resulted

from accidental overwriting of the master diary database with blank data during a minor software upgrade. This loss of more than 90 cases unfortunately also produced the gender imbalances across groups discussed previously that potentially confounded some study findings. Despite the obvious limitation with electronic diaries that data can be lost, electronic EMA diary methods also have numerous advantages over other diary methods by reducing the likelihood of data fabrication. EMA data are less influenced by numerous potential sources of bias in recalled memory of pain, including recency effects, peak experiences, biasing effects of mood, and the impact of current pain intensity on recalled pain [29,33,47,53]. Obvious advantages support future use of signal-contingent sampling to explore behavioral anger expression and its associations with pain.

In summary, this EMA diary study examined within-day associations between behavioral anger expression and momentary chronic pain intensity. Findings indicated significant lagged effects, with elevated behavioral anger expression linked to greater subsequent pain intensity. A trait $\times$ situation model indicated that increased pain intensity was followed by later elevations in anger expression, but only among individuals low in trait anger-out.

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Fig. 1. Associations between momentary chronic pain intensity and subsequent behavioral anger expression as a function of trait anger-out levels. The greatest level of behavioral anger expression is observed among low trait anger-out individuals experiencing high pain intensity.

Table 1

Participant characteristics by participant type.

Measure	LBP (n = 48)	Healthy controls (n = 36)
Gender (% male) **	68.8	36.1
Race:		
Caucasian	66.7	77.8
African-American	18.8	16.7
Ethnicity *:		
Non-Hispanic	85.4	100.0
Age (years) *	34.9 ± 9.05	30.0 ± 9.13
VAS chronic pain intensity (0–100)	49.4 ± 17.43	–
Pain duration (median, in months)	51.4	–
Anger-Out	20.6 ± 4.47	19.9 ± 3.42
Anger-In	21.0 ± 4.89	20.8 ± 4.13
BDI **	7.2 ± 6.27	3.2 ± 3.47
STAI *	37.0 ± 9.71	33.2 ± 6.53

Note: Summary statistics are presented as percentages or means (±SD).

LBP, low back pain; BDI, Beck Depression Inventory; STAI, State-Trait Anxiety Inventory.

* $P < 0.05$.

** $P < 0.01$.

Table 2

Results for Generalized Estimating Equation (GEE) models testing interactions between daily behavioral anger expression and measures of trait anger regulation style as predictors of diary ratings of chronic low back pain intensity.

	β	SE	P-value
Model: Behavioral anger expression $\times$ trait anger-out			
Concurrent anger expression	0.0021	0.0090	0.8129
Prior anger expression	0.0079	0.0068	0.2445
Subsequent anger expression	0.0222	0.0031	<0.0001
Anger-out	-0.0162	0.0273	0.5519
Anger-out $\times$ concurrent express	0.0000	0.0003	0.9287
Anger-out $\times$ prior express	-0.0001	0.0003	0.6505
Anger-out $\times$ subsequent express	-0.0006	0.0001	<0.0001
Model: Behavioral anger expression $\times$ trait anger-in			
Concurrent anger expression	0.0064	0.0096	0.5070
Prior anger expression	0.0161	0.0067	0.0158
Subsequent anger expression	0.0234	0.0024	<0.0001
Anger-in	0.0168	0.0224	0.4547
Anger-in $\times$ concurrent express	-0.0002	0.0004	0.6603
Anger-in $\times$ prior express	-0.0005	0.0003	0.0958
Anger-in $\times$ subsequent express	-0.0008	0.0001	<0.0001

Note: "Prior Anger Expression" effect indicates that current chronic pain intensity was predicted by the level of anger expression in the prior assessment period. "Subsequent Anger Expression" effect indicates that current chronic pain intensity predicted the level of anger expression in the subsequent assessment period.