Is anger management style associated with descending modulation of spinal nociception?

Edward Lannon\textsuperscript{1} | Ellen L. Terry\textsuperscript{2} | Kathryn Thompson\textsuperscript{3} | Jamie L. Rhudy\textsuperscript{1}

\textsuperscript{1}Department of Psychology, The University of Tulsa, Tulsa, OK, USA
\textsuperscript{2}Pain Research & Intervention Center of Excellence, University of Florida, Gainesville, FL, USA
\textsuperscript{3}Department of Psychology, The University of Alabama at Birmingham, Birmingham, AL, USA

Correspondence
Jamie L. Rhudy, Department of Psychology, The University of Tulsa, Tulsa, OK, USA.
Email: jamie-rhudy@utulsa.edu

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Anger management styles (i.e., anger-in and anger-out) characterize a person's typical response to anger. Anger-in, the suppression of anger, and anger-out, the outward expression of anger, have been associated with increased acute and chronic pain. Previous research suggests that anger-in is related to pain because of its shared variance with negative affect; anger-out is believed to be related to pain because of a disruption of endogenous opioid systems. It is currently unknown whether anger management styles promote pain by facilitating central sensitization or spinal nociception. This study assessed the relationship between anger management styles and markers of central sensitization (i.e., temporal summation of pain [TS-pain] and nociception flexion reflex [TS-NFR]), spinal nociception (nociception flexion reflex [NFR] threshold), and measures of pain experience. One hundred nine healthy pain-free individuals completed the study. A bootstrapped mediation analysis was conducted to test whether negative affect mediated relationships with anger-in. Results suggested that anger-in and anger-out were associated with lower NFR thresholds (facilitated spinal nociception), but no other outcome. Negative affect did not mediate either of these relationships. These results suggest that anger management styles may amplify spinal nociceptive processes in healthy humans without altering central sensitization.
INTRODUCTION

Anger management styles (i.e., anger-in and anger-out) are individual differences that characterize one's typical reaction to anger. Individuals with high anger-in suppress verbal and behavioral expressions of anger (e.g., “I keep things in”), while individuals with high anger-out respond to anger with verbal or behavioral expressions (e.g., “I strike out at whatever infuriates me”; Spielberger et al., 1985). Anger management style is known to modulate the perception of pain. Specifically, anger-in and anger-out are associated with increased experimentally induced pain (Bruehl, Chung, Burns, & Diedrich, 2007; Bruehl, al’Absi et al., 2007; Burns, Bruehl, & Caceres, 2004; Burns, Bruehl, & Chont, 2014; Burns, Kubulius, & Bruehl, 2003; Gelpkopf, 1997) and chronic pain (Bruehl, Burns, Chung, Ward, & Johnson, 2002; Bruehl, Chung, & Burns, 2003; Bruehl, Chung, Burns, & Biridepalli, 2003; Bruehl, Chung et al., 2007; Kerns, Rosenberg, & Jacob, 1994; Lombardo, Tan, Jensen, & Anderson, 2005; Materazzo, Cathcart, & Pritchard, 2000; Sayar, Gulec, & Topbas, 2004).

Despite these relationships between anger management style and pain perception, the mechanisms are not completely understood. It is possible that anger management styles activate pain modulatory neural circuits. Indeed, research suggests that there is an overlap between the activation of supraspinal structures associated with pain processing, pain modulation, and anger (Bruehl, Burns, Chung, & Chont, 2009). Specifically, pain has been commonly associated with the activation of the prefrontal cortex, SI, SII, anterior insula, thalamus, and anterior cingulate cortex (Peyron, Laurent, & García-Larrea, 2000), and a meta-analysis found that anger-inducing stimuli are associated with activation in the orbital frontal cortex, the prefrontal cortex, and the anterior cingulate cortex (Murphy, Nimmo-Smith, & Lawrence, 2003). This suggests that pain modulation due to anger may, in part, be related to the activation of these common supraspinal structures.

Importantly though, recent literature suggests that the pain modulating effects of anger-in and anger-out may involve different mechanisms. For example, the relationship between anger-out and increased pain perception may be due to a reduction in the effectiveness of endogenous opioids (i.e., natural painkilling substances; Bruehl et al., 2002; Bruehl, Chung, & Burns, 2006; Bruehl, Chung et al., 2007; Burns et al., 2014). The relationship between anger-in and pain, on the other hand, is thought to be due to the fact that anger-in shares variance with other negative emotions, given that negative emotions enhance pain (for review, see Burns, Quartana, & Bruehl, 2008).

Some pain modulation is mediated by the activation of descending, brain-to-spinal cord circuits that can amplify or dampen incoming pain signals at the spinal level (Ossipov & Porreca, 2005). To study this in intact humans, researchers have assessed a physiological correlate of spinal nociception, the nociceptive flexion reflex (NFR). The NFR is a spinally mediated reflex (e.g., reflexive withdrawal of the foot after stepping on a tack) that occurs following the activation of A-delta nociceptors and is measured from electromyogram (EMG; Sandrini et al., 2005). The NFR is modulated by cognitive-emotional variables thus providing evidence that descending brain-to-spinal cord circuits can influence it (Rhudy, Williams, McCabe, Russell, & Maynard, 2008). To our knowledge, only one study has examined the relationships between anger management style and NFR. Bruehl, al’Absi et al. (2007) assessed the relationship between NFR threshold (the stimulation intensity necessary to elicit the reflex) and anger management styles, but did not find significant relationships. This may be due, in part, to the complexity of the 2-day crossover study design which involved opioid blockade and an attention demanding task.

In addition, no study to date has investigated the relationship between anger management styles and measures of central sensitization. Indeed, it is possible that anger management styles enhance pain by inducing hyperexcitability of central nociceptive neurons (central sensitization). One measure commonly used to study central sensitization is temporal summation of pain (TS-pain), i.e., the increase in pain report that occurs in response to constant-intensity, repetitive, noxious stimuli. TS-pain is believed to represent the psychophysical correlate of wind-up (hyperexcitability of dorsal horn neurons), which has been observed in animal models (Eide, 2000; Li, Simone, & Larson, 1999; Mendell, 1966). However, because TS-pain is dependent on pain report, report bias (or other supraspinal processes) may partially confound this measure. By contrast, temporal summation of the NFR (TS-NFR) is the resulting increase in the
reflex magnitude caused by the repetition of a noxious stimulation of the same stimulus intensity (Arendt-Nielsen, Brennum, Sindrup, & Bak, 1994). Since TS-NFR is a physiological correlate of spinal nociception, this minimizes the potential for report bias and experimental demands.

This study examined the relationships between anger-in, anger-out, NFR threshold, markers of central sensitization (TS-pain, TS-NFR), and subjective ratings of electric stimuli. We hypothesize that anger-in and anger-out will be associated with enhanced pain (higher pain ratings), enhanced TS-pain (greater increase in pain ratings to repetitive stimulation), enhanced TS-NFR (greater increase in the reflex in response to repetitive stimulation), and reduced NFR threshold (less intense stimuli will elicit the reflex). In order to determine whether negative affect explains any observed relationships between anger-in and pain outcomes (suggested by Bruehl et al., 2006), this study used a bootstrapped mediation analyses that used self-reported depressed mood as the mediator.

2 | METHODS

This is a secondary analysis of data from a parent study that examined the influence of catastrophizing on pain outcomes (Terry, Thompson, & Rhudy, 2015). The results presented here are novel and have not been previously published. The experimental protocol was approved by The University of Tulsa Institutional Review Board. Informed consent was obtained from all participants.

2.1 | Participants

Participants were 112 healthy pain-free individuals recruited from the local community or a psychology subject pool. Participants were excluded for the following self-reported conditions: neurological, cardiovascular, or circulatory problems; chronic pain; recent psychological trauma; use of over-the-counter pain medication within 24 hr, and prescription pain medication within 2 weeks of participation; use of antidepressant, anxiolytic, or high blood pressure medications; having a body mass index 35 or above (due to difficulty recording the nociceptiv reflex because of high adiposity); and being under the age of 18. Community participants were compensated $50 for their time, whereas participants recruited from the subject pool received class credit. The sample was composed of 47 (43.1%) women and most participants (77.1%) were White. On average, the participants were 29 years of age ($SD = 11.50$) and had 14.9 ($SD = 2.59$) years of education. Three participants were unable to achieve an NFR before pain tolerance, or the maximum stimulation was reached resulting in a total sample of 109 available for analyses.

2.2 | Apparatus and procedures

Participants were seated comfortably throughout testing procedures in a reclining chair that kept their knee at a constant angle of approximately 160°. The experimenter monitored participant activity from an adjacent room through the use of a video camera. Participants could communicate with the experimenter by speaking in a normal voice which was picked up by the video camera microphone. Participants wore headphones that were used to receive pre-recorded audio instructions as well as to hear the experimenter during testing.

Data collection and stimuli presentation were controlled by a PC with an A/D board (PCI-PCI-6071E; National Instruments, Austin, TX, USA) and LabVIEW (National Instruments). Questionnaires were presented electronically on a 17-inch monitor placed approximately .5 m away from the participant. Participants were able to submit ratings on computer-presented scales with the use of a mouse. Physiological recordings were monitored in real time.

A Digitimer DS7A stimulator (Digitimer Ltd, Hertfordshire, UK) delivered the electrical stimulations through a bipolar stimulating electrode (Nicolet; 30-mm interelectrode distance) placed over the retromalleolar surface of the sural nerve on the left ankle. No stimulations over 50 mA were delivered for participant safety.
Electromyographic (EMG) activity for NFR was recorded with two adjacent Ag-AgCl electrodes placed on the bicep femoris (10 cm superior to the popliteal fossa). An Ag-AgCl sensor was used as a ground and attached to the lateral epicondyle of the left femur. Conductive gel (EC60; Grass Technologies, West Warwick, RI, USA) was applied to all sensors. Prior to any sensor placement, the skin was cleaned using isopropyl alcohol and exfoliated using an exfoliation cream (Nuprep; Weaver and Company, Aurora, CO, USA) in order to reduce skin impedance below 5 kΩ. EMG activity was sampled at 1,000 Hz and was amplified and filtered using Grass Technologies Model 15TL amplifier (AC Module 15A54).

2.3 | Anger management style

The Anger Expression Inventory was used to measure anger management style. The Anger Expression Inventory is a reliable and valid scale that is used to measure individual differences between two behavioral expressions of anger (anger-in and anger-out; Spielberger et al., 1985). This 20-item scale describes anger-in as an internalization of anger and anger-out as an outward behavioral reaction to anger. Four items are not included in the anger-in/out subscales. Each item was scored on a 4-point Likert-type scale that ranged from 1 (almost never) to 4 (almost always). Scores on both subscales range from 8 to 32, with higher scores representing greater propensity for anger-in or anger-out.

2.4 | Center for Epidemiologic Studies–Depression scale (CES-D)

The Center for Epidemiological Studies–Depression (CES-D) is a 20-item reliable and valid self-report measure that assesses symptoms of depressed mood over the past week (Radloff, 1977). Each item was scored on a 4-point Likert-type scale that ranged from 0 (Rarely or none of the time) to 3 (Most or all of the time). Scores on the CES-D range from 0 to 60 with higher scores representing greater depressed mood. The total CES-D score was used to assess negative affect.

2.5 | Pain and nociceptive outcomes

Painful stimulations consisted of 5 square wave pulses of 1-ms duration with 3-ms interpulse interval (250 Hz). These pulse trains are perceived as a single stimulus. For all pain-related outcomes, electric stimulations were delivered to the retromalleolar surface of the sural nerve at the left ankle.

2.5.1 | NFR threshold

Nociception flexion reflex threshold was assessed using a series of three ascending/descending staircases of stimulations (France, Rhudy, & McGlone, 2009; Rhudy & France, 2007). The reflex was said to be present if average biceps femoris EMG in the 90- to 150-ms poststimulation window exceeded the 60-ms prestimulation baseline window by at least 1.4 standard deviations from baseline. For the first ascending/descending staircase, stimulations increased in 2 mA steps until the NFR was detected and then decreased in 1 mA steps until the NFR disappeared. Two more staircases were conducted in the same manner with 1 mA steps. NFR threshold is defined as the average stimulus intensity of the last two peaks and troughs of the ascending/descending staircases.

2.5.2 | Suprathreshold pain ratings

After each stimulation in the NFR threshold task, the participants rated their sensation on a visual analog scale (VAS) for pain intensity, with anchors that ranged from "no pain sensation" to "the most intense pain sensation imaginable." These ratings were converted to scores that ranged from 0 to 100, with higher values indicating greater intensity.
The average pain ratings from the last two peaks and troughs in the NFR threshold tasks (procedure stated above) were used as suprathreshold pain ratings.

2.5.3 Temporal summation of pain and NFR

Prior to delivering temporal summation of pain and NFR stimuli, three-stimulation threshold was assessed because it ensures reliable NFRs during temporal summation testing (Terry et al., 2011). For the three-stimulation threshold task, participants received several series of three electrical stimulations (0.5-s interstimulus interval). The intensity of the first series started at 0 mA and the series increased by 1 mA until the third stimulation evoked a reflex using the same criteria as described in the NFR threshold section. The intensity of the electric stimuli used to test temporal summation of pain and NFR was set at the higher of 120% of NFR threshold or 120% of three-stimulation threshold. The higher stimulation intensity was selected because it had to be set high enough to elicit reliable reflexes throughout testing.

To assess temporal summation of pain, five single electric stimulations were delivered with an interstimulus interval of 8–12 s. After all five stimulations were delivered, the participant rated the average pain intensity and unpleasantness of these single stimuli using VASs. For pain intensity, the anchors were “no pain sensation” to “the most intense pain sensation imaginable,” and for pain unpleasantness, the anchors were “not at all unpleasant” to “the most unpleasant imaginable.” These ratings were converted to scores that ranged from 0 to 100, with higher values indicating greater intensity or unpleasantness. Next, five series of three electric stimulations were delivered with an interstimulus interval of 0.5 s. Each of the five series had an interseries interval of 8–12 s. Following all five series of three stimulations, the participant rated the average pain intensity and unpleasantness of the stimuli using VASs that measured pain intensity and unpleasantness. Temporal summation of pain was defined as the average intensity and unpleasantness rating of the five 3-stimulus series minus the average intensity and unpleasantness rating of the five single stimuli.

Temporal summation of NFR was assessed from the five 3-stimulus series and defined as the average change in NFR magnitude from the first to the third stimulation. To calculate NFRs within each series, the average biceps femoris EMG in the 60 ms prior to the first stimulus in the series was subtracted from each of the three 70- to 150-ms poststimulus intervals (a different interval is used from NFR threshold because the onset latency shortens when a series of stimuli are used; Terry et al., 2011). Each EMG trial (series) was visually inspected for problems and excluded if baseline activity in the 60 ms prior to stimulations #2 or #3 was >10 μV, because this represents muscle activity unrelated to nociceptive processing (i.e., voluntary muscle tension following a previous stimulation). Participants were only included in analysis of TS-NFR if they had ≥2 of 5 valid trials. Twenty-two participants were excluded from TS-NFR analyses: four quit before completing temporal summation testing, while the remainder did not have sufficient data after visual inspection. No differences in anger-in, \( t(107) = 1.85, p > .05 \); anger-out, \( t(107) = 0.706, p > .05 \); NFR threshold, \( t(107) = 0.868, p > .05 \); age \( t(107) = 0.572, p > .05 \); or sex \( \chi^2(1, N = 109) = 1.43, p > .05 \) were noted between participants that were excluded from TS-NFR analysis and those who were not. Similar rates of rejection of electrophysiological data have been seen in other TS-NFR studies (Terry et al., 2011).

3 RESULTS

Means and SDs for anger management style were anger-in (\( M = 15.08, SD = 4.4 \)) and anger-out (\( M = 13.87, SD = 2.97 \)). Means and SDs for pain outcomes and correlations with anger management styles are presented in Table 1. Given that stimulation intensity was individually calibrated for each participant (based on NFR threshold and three-stimulation threshold), stimulus intensity was used as a covariate in semipartial correlations for suprathreshold pain ratings and temporal summation of pain. The only significant correlations were between anger-out (Figure 1) and NFR threshold and anger-in and NFR threshold (Figure 2), indicating anger management styles were associated with enhanced spinal nociception (lower NFR thresholds).
3.1 Mediation analysis

Two Sobel tests with bootstrap estimation (10,000 samples) of coefficients (Preacher & Hayes, 2004) were used to test the hypothesis that negative affect (as measured by the CES-D) mediates the relationship between anger-in and NFR thresholds, but not anger-out and NFR thresholds (Bruehl et al., 2006). If the indirect effect through CES-D

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**TABLE 1** Mean, SDs, and correlations of pain outcomes with anger management styles

<table>
<thead>
<tr>
<th>Pain outcome</th>
<th>Means (SD)</th>
<th>Anger-in</th>
<th>Anger-out</th>
</tr>
</thead>
<tbody>
<tr>
<td>NFR threshold (mA)</td>
<td>20.14 (11.63)</td>
<td>-0.26*</td>
<td>-0.23*</td>
</tr>
<tr>
<td>Suprathreshold pain intensity ratings (0–100)</td>
<td>49.77 (30.48)</td>
<td>0.01</td>
<td>-0.12</td>
</tr>
<tr>
<td>Temporal summation of NFR (ΔμV)</td>
<td>5.00 (5.68)</td>
<td>-0.08</td>
<td>-0.05</td>
</tr>
<tr>
<td>Temporal summation of pain intensity (Δ ratings)</td>
<td>14.88 (13.31)</td>
<td>-0.02</td>
<td>0.18</td>
</tr>
<tr>
<td>Temporal summation of pain unpleasantness (Δ ratings)</td>
<td>18.08 (13.80)</td>
<td>-0.1</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Note. NFR, nociceptive flexion reflex.

*aSemi-partial correlations were used to control for stimulus intensity.

*p < .05

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**FIGURE 1** The relationship between anger-out and nociceptive flexion reflex (NFR) threshold (mA)

**FIGURE 2** The relationship between anger-in and nociceptive flexion reflex (NFR) threshold (mA)
is significant (i.e., the confidence interval does not contain zeros), then this is evidence that negative affect does, at least partially, mediate the relationship with NFR threshold. The indirect effect was not significant for anger-in ($B = -.12, SE = .14, 95\% CI [-.46, .12]$) or anger-out ($B = -.15, SE = .12, 95\% CI [-.53, .01]$), indicating the relationships with NFR threshold are not mediated by negative affect.

4 | DISCUSSION

This study investigated the relationships between anger management styles (anger-in, anger-out) and measures of pain, spinal nociception (NFR threshold), and central sensitization (TS-NFR, TS-Pain). In contrast to the work by Bruehl, al’Absi et al. (2007), our study revealed negative relationships between both measures of anger management style and NFR threshold. These relationships were not mediated by negative affect (as measured by the CES-D). Together, these findings provide preliminary evidence that both suppression and outward expression of anger may engage descending, brain-to-spinal cord circuits to amplify spinal nociception and that this effect is not due to general activation of negative affect.

Interestingly, measures of central sensitization (i.e., TS-pain, TS-NFR) were not associated with anger management styles. This could be due, in part, by different physiological mechanisms that are thought to support NFR threshold versus TS-pain/TS-NFR. TS-pain and TS-NFR are thought to depend on activation of N-Methyl-D-aspartate receptors, while the NFR may be primarily dependent on the activation of α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid/kainite receptors (Arendt-Nielsen et al., 1994; Yoshimura & Nishi, 1992). Thus, anger management styles may activate descending circuits to primarily affect α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid/kainite receptors.

Surprisingly, this study did not find a relationship between anger management style and suprathreshold pain ratings (Bruehl, Chung, & Burns, 2008; Bruehl et al., 2002; Bruehl, Chung et al., 2007; Burns et al., 2014; Gelkopf, 1997; Janssen, Spinhoven, & Brosschot, 2001). One possible explanation is that many studies that found this relationship in healthy participants have used prolonged, tonic painful stimuli (e.g., tolerance measures; Bruehl et al., 2002, 2008; Bruehl, Chung et al., 2007; Burns et al., 2014; Gelkopf, 1997; Janssen et al., 2001) that may produce sensations closer to clinical pain than those produced by punctate electric stimuli. Consistent with this, Bruehl, al’Absi, et al. (2007) did not find a relationship between anger management styles and pain in response to electrocutaneous stimuli. Alternatively, the lack of relationship may be due to the fact that the stimuli used to evoke suprathreshold ratings were individually calibrated to each participant based on their nociceptive reflex threshold.

4.1 | Implications

This study found a negative relationship between NFR threshold and both anger-in and anger-out implying that these anger management styles were associated with increased activation of brain-to-spinal cord circuits that facilitate nociception. These results add to the literature suggesting that these anger management styles have negative implications for pain processing. For example, in a treatment study for individuals with chronic pain conducted by Burns, Johnson, Devine, Mahoney, and Pawl (1998), anger-out was associated with reduced lifting capacity, while anger-in was negatively correlated with improvements in depression and general activities in men. In male veterans, maladaptive anger management styles were positively correlated with pain intensity and low self-efficacy (Lombardo et al., 2005). Furthermore, in a diary study investigating the relationship between anger and pain in women with fibromyalgia, trait anger inhibition was positively correlated with increased pain (Middendorp et al., 2010).

Because anger-in and anger-out are associated with facilitation of nociceptive signaling, this means that other methods of managing anger may be more adaptive for pain patients. Indeed, a number of empirically validated emotion-regulation strategies (e.g., cognitive restructuring and relaxation training) improve anger, but also have
beneficial effects on nociceptive processing (Beck & Fernandez, 1998; Butler, Chapman, Forman, & Beck, 2006). These should be considered when patients are known to have issues with anger and pain.

4.2 Study limitations

This study had a number of strengths including (1) studying the pain modulatory mechanisms of anger management styles; (2) being the first study to investigate the relationships between measures of central sensitization and anger management styles; (3) using physiological markers of nociception as a way to eliminate self-report biases; and (4) using bootstrapped coefficient estimation to test for mediation by negative affect.

However, this study had several limitations. This study was conducted in healthy pain-free individuals. Thus, these results may not generalize to populations with chronic pain. Also, some data were lost due to EMG artifacts in the TS-NFR measures. This could have reduced the statistical power to detect relationships with this outcome. However, the generalizability of these findings do not seem to be impaired as no differences in anger-in, anger-out, NFR threshold, age, or sex were noted between participants that were excluded from TS-NFR analysis and those who were not. Also, this study was correlational. Hence, no causal statement can be made about the relationships between anger management styles and NFR threshold. Likewise, there is no way of knowing if the participants were already engaging in cognitive restructuring or relaxation techniques.

5 SUMMARY

In summary, we found that anger-in and anger-out are associated with enhanced NFR (lower NFR threshold), but not pain ratings nor measures of central sensitization. This effect was not mediated by negative affect. Given this, anger management styles may modulate nociceptive signals at the spinal level before they reach the brain. Hence, treatments for anger should not focus on suppression of anger or outward expression of anger. Although research is needed, it is possible that other anger management strategies (e.g., cognitive restructuring and relaxation training) may be more adaptive.

CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

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