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Anger Suppression Predicts Pain, Emotional, and Cardiovascular Responses to the Cold Pressor

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Abstract

Background Manipulated anger suppression has been shown to heighten pain and anger responses to pain.

Purpose We examined whether individual differences in self-reported anger suppression predicted pain, anger, and blood pressure responses to acute pain.

Methods Healthy participants ($N=47$) underwent an anger-provoking speech task followed by a cold pressor pain task. Participants reported their degree of suppression of thoughts and feelings related to the speech. Pain intensity ratings were obtained throughout the cold pressor. Self-reported anger, anxiety and positive emotion, as well as ratings of sensory, general distress, and anger-specific elements of pain were obtained following the cold pressor. Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were recorded throughout.

Results Self-reported suppression predicted greater pain intensity ratings, perception of sensory and anger-specific elements of pain, and self-reported anger in response to the

cold pressor. Associations between self-reported suppression and pain intensity and ratings of anger-specific elements of pain were statistically mediated by pain-induced changes in self-reported anger, whereas the effect of suppression on sensory pain ratings was not. Self-reported suppression was also correlated inversely with SBP responses to the cold pressor.

Conclusions Consistent with an ironic process model and prior studies involving experimental manipulation of suppression, self-reported suppression of anger predicted greater pain intensity and perception of the anger-specific element of pain. Findings also suggest that suppression might attenuate homeostatic pressor responses to acute pain.

Keywords Anger · Anger suppression · Blood pressure · Cardiovascular reactivity · Ironic processes · Pain

Introduction

Physical pain is inextricably intertwined with negative emotions [1, 2]. While the role of depression and anxiety has received considerable empirical study, it has in recent years become apparent that the arousal of anger has important implications for the experience of pain. A number of patient groups are characterized by high levels of anger [3–6], and healthy participants exposed to acute noxious stimuli report increases in anger and aggression [7–10]. However, the manner in which anger is regulated appears to predict pain-related outcomes above and beyond the experience of anger alone [for recent reviews, see 11, 12]. Consonant with long-standing clinical anecdote and theoretical conjecture [13–15], trait-like measures of anger suppression—for instance, the “Anger-In” subscale

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of Spielberger's [16] Anger Expression Inventory—have been associated with heightened pain sensitivity in healthy [6, 17, 18] and chronic pain [3, 19, 20] participant samples. As noted in a recent review [11], relations between Anger-In and pain might be accounted for by a host of factors that have little or nothing to do with the actual process of suppression [e.g., negative affectivity 21]. In short, reliance on trait measures of anger suppression such as Anger-In does not afford unequivocal conclusions regarding cause-and-effect associations between an inhibitory process and pain-related outcomes.

Borrowing from the broader literature on thought [22] and emotion [23] suppression, we recently advanced an ironic process model of anger suppression and pain [10]. The central thesis of the model is that efforts to suppress anger ironically amplify cognitive accessibility of anger-related thoughts, feelings, and behavioral inclinations, in turn contaminating [24] the perception of subsequent painful events in an anger-congruent manner [25]. More specifically, Wegner's [22] ironic process theory proposes that efforts to suppress unwanted thoughts and feelings from awareness rouse two interrelated cognitive search processes. The first process is a conscious, resource-dependent "operator" that eliminates unwanted mental content from mind by searching for mental contents that are *consistent* with the desired state of mind (e.g., joy). The second process is an unconscious, automatic, and resource-independent "monitor" that searches for mental contents that are *inconsistent* with the desired state of mind (e.g., anger). Under circumstances in which cognitive resources are readily available, the monitor and operator work together to achieve the desired state of mind by way of the monitor signaling activation of the operator. However, under conditions in which cognitive reserve is taxed (e.g., stressful events), the operator does not have the resources to effectively remove unwanted content from awareness, and so the monitor, now left unchecked, continues to find evidence of thoughts inconsistent with the desired state of mind. Hence, during an anger-provoking stressor, suppression will lead paradoxically to increased accessibility of anger-related thoughts, feelings, and behavioral inclinations. Given the fundamental link between pain and negative emotion [1, 2], and possibly anger in particular [7–9], we argue that highly accessible anger-related data will contaminate or influence perception of the irritating, annoying, and frustrating qualities of the pain experience in particular [for more detailed discussion, see 10].

To test the validity of this model, we advanced a laboratory-based experimental paradigm in which participants are instructed to suppress their emotions or to deal with their emotions however they prefer (i.e., control) either during or following explicit anger-provocation. Subsequently, participants are exposed to a pain-eliciting stimu-

lus. This approach allows for a cause-and-effect analysis. Current findings appear to support an ironic process model. Participants randomized to suppress their emotions during anger-provocation report greater pain and irritation in response to subsequent pain induction than controls [10, 26]. Further, significant variance in self-reported pain severity is accounted for by concurrent changes in anger and irritation, but not anxiety or positive emotion. The effects of suppression appear to extend to clinical pain as well [27]. Among chronic low back pain patients, suppression during an anger-provoking event predicted clinically relevant pain behaviors and self-reported pain severity during a subsequent standardized structured pain behavior task [28]. Again, the effect of suppression on acute clinical pain outcomes was mediated by suppression-induced increases in self-reported anger.

The control condition employed in these investigations has been a "do/think anything" condition in which participants are instructed to regulate their emotions however they prefer or to think about anything that they would like. Hence, it is feasible that participants in the control condition are engaging in suppression under their own volition and to varying degrees. Consistent with this notion, we have observed considerable variability in self-reported efforts to suppress stress-evoked emotional responses within the control condition, suggesting the presence of meaningful individual differences that may be overlooked. It is important to note that trait Anger-In assesses the tendency to respond one way or another to *any* anger-provoking event. In contrast, the variability observed in suppression in our investigations may represent individual differences in suppression that are tightly coupled to a *specific* anger-provoking event, and thus might be associated with pain responses largely independent of third variables [e.g., negative affectivity 21]. Whether variability in state suppression is related to pain and emotional responses consistent with predictions predicated on an ironic process model has not been examined.

In the present study, we conducted additional analyses on extant data (26; Study 2). Participants gave an impromptu speech with explicit anger-provocation, followed by a cold pressor. Based on an ironic process model, we predicted that individual differences in state suppression of thoughts and feelings related to the anger-provoking speech task would predict self-reported pain intensity and greater perception of anger-specific qualities of pain, but not general distress or sensory elements of pain. We also predicted that self-reported suppression would be associated with greater self-reported anger in response to the cold pressor, but not with cold pressor-induced changes in anxiety or positive emotion. Finally, we predicted that increases in pain-induced anger would statistically mediate suppression–pain associations.

We also examined the effect of self-reported “state” suppression on cardiovascular responses to pain. Cardiovascular and pain regulatory systems are mediated by a number of overlapping brain regions [29]. Indeed, there exists in healthy individuals an inverse relationship between blood pressure (BP) and pain sensitivity [for a review, see 30]. Although a number of mechanisms for an inverse BP–pain sensitivity relationship have been proposed, one model suggests that pressor responses to pain activate baroreceptors that, in turn, activate endogenous descending pain inhibitory mechanisms [31, 32; for a discussion of other viable pathways, see 30]. Non-human animal and human studies have indicated that the presence of hypertension, or a family history of hypertension, can eliminate or even reverse this inverse BP–pain relationship [33–40]. Interestingly, empirical data suggest that anger suppression, as well as conceptually related constructs such as defensive and repressive coping, are involved in the pathogenesis of hypertension [41–43]. Hence, we suspected that suppression would predict diminished BP responses to pain in conjunction with exaggerated pain report.

Method

Participants

Forty-seven healthy participants (24 women; 51.1%) from a Midwestern University participated in exchange for partial course credit. Exclusion criteria included (a) cardiovascular disease, (b) any chronic pain condition, (c) Raynaud’s disease, (d) history of bipolar or psychotic disorders, (e) current alcohol and/or substance abuse, or (f) current depressive or anxiety disorder. The sample reported a mean age of 19.18 (SD=1.2); 59.6% were Caucasian, 23.4% Asian; 8.5% Hispanic; 4.3% African American; and 1% Other; 3.1% choose not to indicate an ethnic/racial category.

Experimental Tasks

Anger Induction

Participants gave an impromptu speech during which they argued either for or against a 10% increase in tuition at their educational institution. They were informed that the speech would be 5 min in duration and that it would be evaluated for clarity and quality of content by the experimenter. During the speech, the experimenter sat with a clipboard and appeared to be intently listening to and watching the participant. At 1 min, the experimenter interrupted the participant and asked, “why would you argue that?” At

1 min and 30 s, the experimenter stated, “your speech is making no sense.” At 2 min and 30 s, the experimenter stated, “I still don’t follow what you are saying.” At 3 min, the experimenter stated, “you know what, that’s enough.”

Pain Induction

Pain was induced with a fixed-latency (2 min) cold pressor test on the non-dominant hand. The cold pressor apparatus consisted of a standard 48-quart cooler half-filled with water and half-filled with ice. A metal grate was used to separate the ice and water to prevent direct ice-to-skin contact. The water temperature was maintained between 1°C and 3°C.

Measures

Self-reported Pain Intensity

Participants rated the cold pressor pain intensity on a numeric rating scale ranging from 0 (“no discomfort at all”) to 10 (“extreme discomfort”) at 40-s intervals during the cold pressor.

Pain Adjectives Rating Form

Participants rated each of a series of adjectives that described the cold pressor pain on a scale ranging from 0 (“none”) to 3 (“severe”). These pain descriptors were taken from the full and short-form versions of the McGill Pain Questionnaire [44], a well validated and widely used measure to assess the differential qualities of both acute and chronic pain. We assessed ratings on sensory (throbbing, shooting, pricking, stabbing, sharp, cramping, tingling, hot-burning), general distress (punishing–cruel, frightening, tiring–exhausting, vicious, sickening), and anger-specific (irritating, annoying, irksome, infuriating, frustrating) dimensions of pain. Results of principal components analysis of the descriptors used in this rating form [10] revealed a three-component solution that consisted of sensory, general distress, and anger-specific subscales of pain elements.

Self-reported Suppression

Participants rated the extent to which they tried to suppress their thoughts of, and feelings related to, the speech task on a 0 (“not at all”) to 10 (“very much”) numeric rating scale.

Self-reported Emotion

Participants responded to single items assessing anxiety (i.e., nervous), anger (i.e., mad), and positive emotion (i.e.,

joy) on a 0 (“not at all”) to 10 (“extremely”) numeric rating scale.

Cardiovascular Parameters

Systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) were measured with a Dinamap 1846 SX Monitor (Johnson and Johnson Medical, Inc., NJ, USA). This instrument assesses blood pressure using the oscillometric technique. Readings were taken during baseline at 60, 120, and 180 s and during pain induction at 30, 60, and 90 s.

Procedure

Upon arrival at the laboratory, participants were screened for exclusion criteria and provided written informed consent. They were then seated upright in a comfortable chair and an automated blood pressure cuff was affixed over the brachial artery of the participant’s dominant arm. Participants remained seated quietly for a 5-min period, which served as a baseline, after which they provided emotion ratings based on how they felt “right now.” Participants were then given instructions for a modified dot-probe computer task, completed a nominal amount of practice trials on the task, and subsequently were given instructions for and commenced the impromptu speech task. Following the speech task, participants provided emotion ratings based on how they felt “during the speech.” Participants then underwent the experimental trials of the computer task during which they received their respective think anything/control or suppression instructions. For the purposes of the present study, only participants in the think anything/control condition were included. All participants were instructed to “feel free to think about *anything* that you would like during the computer task.” Following the computer task, participants were provided instructions for the cold pressor task and subsequently immersed their non-dominant hand into the iced water for 2 min, during which they provided ratings of pain intensity at 40-s intervals. Immediately upon termination of the cold pressor, participants provided responses to the Pain Adjectives Rating Form and emotion ratings based on how they felt “when their hand was in the iced water.” All participants were then debriefed. All of the procedures were approved by the appropriate Institutional Review Boards.

Data Reduction and Analytic Strategy

Composite variables representing perceptions of anger-specific, general distress, and sensory components of pain were created by taking the mean of responses on relevant items for each subscale (see above). A mean pain intensity

variable was computed by averaging ratings obtained during the cold pressor. Mean SBP, DBP, and HR variables were computed across measurements obtained during baseline and the cold pressor. Standardized residuals were computed to represent all baseline-to-cold pressor changes in self-reported anger, anxiety and positive emotion, as well as cardiovascular variables by regressing cold pressor values on baseline values. This procedure yields change scores that are uncorrelated with baseline values. Main study hypotheses were examined with zero-order correlations between self-reported suppression, pain intensity ratings, ratings of sensory, general distress and anger-specific elements of pain, and standardized residuals for self-reported emotion (i.e., anger, anxiety, and positive emotion) and cardiovascular parameters (i.e., SBP, DBP, and HR).

To examine whether the suppression–pain relationship was statistically mediated by pain-induced increases in anger, we applied a bootstrapping technique to test simple mediation [45–47]. Specifically, we obtained a 95% bootstrapped confidence interval for indirect effects of suppression on pain. Conceptually, bootstrapping is a nonparametric approach to effect-size estimation and hypothesis testing that does not make assumptions about the form of the distribution of the variables within a given model (i.e., normal versus skewed). Bootstrapping has been recommended as a means of circumventing the power problem introduced by asymmetries and other forms of non-normality in the sampling distribution of $a \times b$ [45]. In the current study, $a \times b$ represents the indirect effect of suppression on pain outcomes and is defined as the product of the suppression-to-anger path (a) and the anger to pain path (b), or $a \times b$.

The bootstrapping approach is completed through taking a large number of samples of size n (where n is the original sample size) from the data, sampling with replacement (and therefore an observation that appears only once in the original data set can appear multiple times in a bootstrapped dataset), and computing the indirect effect, $a \times b$, in each sample. The point estimate of $a \times b$ is the mean $a \times b$ calculated over the bootstrap samples, and the estimated standard error is the standard deviation of the $a \times b$ estimates. For confidence interval estimation, we took 1,000 bootstrap samples. To create the 95% confidence interval, the elements of the vector of 1,000 estimates of $a \times b$ are sorted from low to high. The lower limit of the confidence interval is the 25th score and the upper limit is the 976th score in this distribution.

Bootstrapped confidence intervals were used to test the significance of the indirect effect because recent statistical research has suggested that bootstrapping is more appropriate than a normal-theory test (i.e., Sobel’s test) for studies with smaller sample sizes and non-normally

distributed variables [45–47]. Specifically, a bias-corrected bootstrapped confidence interval was used because type I error rates and statistical power for this method have been shown to be less than other methods, such as the series of regression analyses (i.e., causal steps approach) recommended by Baron and Kenny [48].

Results

Preliminary Analyses

First, it was necessary to establish that the speech task was a valid anger induction paradigm. Given the social-evaluative nature of the task, it was particularly important to establish whether task-induced changes in anger were distinct from changes in anxiety and positive emotion. We conducted repeated measures ANOVAs on baseline and speech task anger, anxiety, and positive emotion ratings (see Table 1 for mean and SD self-reported anger, anxiety, and positive emotion ratings at each experimental epoch). For anger, there was a robust change from baseline, $F(1, 46)=39.56$, $p<0.001$, $\eta^2=0.46$. There was also a statistically significant increase in anxiety from baseline to the speech task, $F(1, 46)=5.82$, $p<0.02$, $\eta^2=0.11$. It is worth noting that the effect size for baseline-to-speech task change in anger is nearly four times more robust than speech task-induced change in anxiety, suggesting that the task primarily (albeit not exclusively) induced anger. There were also significant decreases in positive emotion from baseline during the speech task, $F(1, 46)=17.05$, $p<0.001$, $\eta^2=0.27$.

The sample mean self-reported suppression rating was 3.8 (SD=2.2) with a range of responses ranging from 0 to 10. Examination of frequency distributions indicated that approximately 29.5% of the sample responded 0–2, 53.1% responded 3–5, and 14% responded 6–10. Hence, the majority of participants reported mild-to-moderate levels of suppression, despite no explicit instructions to do so.

Table 1 Mean (SD) self-reported emotion and cardiovascular values at each experimental epoch

	Baseline	Speech task	Cold pressor
Anger	2.00 (1.76)	3.77 (2.61)	3.83 (2.42)
Anxiety	1.45 (2.11)	1.96 (2.00)	1.51 (1.79)
Positive emotion	3.40 (2.44)	2.28 (1.95)	1.87 (1.62)
SBP (mm/hg)	110.71 (12.28)	–	117.62 (15.71)
DBP (mm/hg)	61.81 (6.69)	–	69.65 (10.57)
HR (bpm)	74.22 (10.70)	–	76.88 (13.17)

Next, we were interested in examining whether the variability in self-reported suppression was held in common with variability in speech task-induced changes in anger, anxiety, or positive emotion. That is, we wanted to determine if our index of suppression was tapping an inhibitory process that was not reducible to negative emotional responses to the anger-provoking speech task. To do so, we computed standardized residuals for baseline-to-speech task changes for anger, anxiety, and positive emotion, and subsequently examined zero-order correlations between these residuals and self-reported suppression ratings. Self-reported suppression ratings were not significantly correlated with anger, anxiety, or positive emotion residuals (r values ≤ 0.18 , p values >0.10). These data suggest that the variability observed in self-reported suppression ratings might truly reflect an engagement in a suppressive process and that these ratings were not simply a proxy for negative emotion, as appears to be the case for trait Anger-In scores [11].

Self-reported anger and SBP, DBP, and HR increased significantly from baseline during the cold pressor, $F(1, 46)$ values ≥ 4.25 , p values <0.05 , $\eta^2 \geq 0.08$. Moreover, self-reported positive emotion decreased from baseline during the cold pressor, $F(1, 46)=35.81$, $p<0.001$, $\eta^2=0.44$. Anxiety, however, did not increase significantly from baseline to the cold pressor, $F(1, 46)<1$.

Primary Analyses

First, we examined zero-order correlations between self-reported suppression ratings and self-reported pain intensity and ratings on sensory, general distress, and anger-specific composites (see Table 2). As predicted, self-reported suppression ratings were positively associated with mean self-reported pain intensity ratings obtained during the cold pressor pain task ($r=0.39$, $p<0.01$). Moreover, self-reported suppression was correlated with ratings of sensory ($r=0.37$, $p<0.05$) and anger-specific components of pain ($r=0.31$, $p<0.05$). Self-reported suppression was not associated with ratings for the general distress pain component ($r=0.22$, $p>0.10$).

Zero-order correlations between self-reported suppression ratings and standardized residuals for emotion and cardiovascular variables were then examined (see Table 2). Consistent with our hypothesis, suppression ratings were associated with self-reported baseline-to-cold pressor changes in anger ($r=0.30$, $p<0.05$), but not with changes in self-reported anxiety ($r=0.12$, $p=0.41$) or positive emotion ($r=-0.04$, $p>0.10$). SBP was inversely related to mean pain intensity ratings ($r=-0.28$, $p=0.05$). SBP was not significantly related to anger-specific ($r=-0.04$, $p>0.10$), general distress ($r=-0.22$, $p>0.10$), and sensory ($r=-0.24$, $p>0.10$) pain component ratings. SBP was

Table 2 Zero-order correlations among main study variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Self-reported suppression	–										
2. Mean pain intensity	0.39	–									
3. Sensory pain composite ratings	0.37	0.53	–								
4. General distress pain composite ratings	0.22	0.52	0.64	–							
5. Anger-specific pain composite ratings	0.31	0.62	0.45	0.53	–						
6. SBP Δ	–0.31	–0.28	–0.22	–0.24	–0.04	–					
7. DBP Δ	0.03	0.10	0.06	0.16	0.02	0.40	–				
8. HR Δ	0.00	0.34	0.14	0.22	0.16	–0.16	0.26	–			
9. Anger Δ	0.30	0.65	0.35	0.34	0.68	0.01	0.07	0.16	–		
10. Anxiety Δ	0.12	0.25	0.39	0.21	0.16	0.06	–0.07	–0.01	0.22	–	
11. Positive emotion Δ	–0.04	–0.26	–0.20	–0.10	–0.14	0.07	–0.11	–0.11	–0.28	0.07	–

Change values are standardized residualized change scores for baseline-to-cold pressor task. Correlations coefficients in bold are statistically significant at $p \leq 0.05$

minimally correlated with pain-induced changes in anger, anxiety, and positive emotion (r values ≤ 0.10). Self-reported suppression was *inversely* associated with cold pressor-induced SBP responses ($r = -0.31$, $p < 0.05$). Self-reported suppression was not related to DBP and HR responses to the cold pressor (r values ≤ 0.03).

To summarize, suppression predicted mean pain intensity ratings, ratings on sensory and anger-specific dimensions of pain, and cold pressor-induced increases in anger. Suppression did not predict ratings on the general distress pain component or changes in self-reported anxiety or positive emotion associated with the cold pressor. Lastly, self-reported suppression predicted attenuated SBP responses to the cold pressor, but was not related to cold pressor-induced DBP and HR responses.

Mediation Analyses

In our prior investigations [26, 27], we explored whether differences between suppression and control group on pain ratings were statistically mediated by group differences in pain-evoked anger responses. Here, we attempted to replicate this finding using self-reported suppression ratings as the predictor. Hence, we examined whether cold pressor-induced changes in self-reported anger statistically mediated the observed associations between suppression and mean pain intensity ratings and ratings of sensory and anger-specific components of pain. To test this proposition, we employed simple mediation analyses via bootstrapping as described earlier (see above).

Results of the tests of the simple bootstrapped mediation of state suppression on pain indexes through self-reported anger are provided in Table 3. As can be seen, with mean pain intensity ratings as the DV, the total effect of self-reported suppression on the DV was significant ($p < 0.01$).

Moreover, self-reported suppression was significantly associated with cold pressor-induced changes in anger ($p < 0.05$), which were in turn associated with mean pain intensity ratings ($p < 0.001$). Of note, the direct effect of self-reported suppression on mean pain intensity ratings with cold pressor-induced changes in self-reported anger included in the model was not significant ($p = 0.07$). Of most importance, the 95% bootstrapped confidence interval for the $a \times b$ path did not include zero, thereby providing quantitative evidence for the simple mediation of the suppression–pain relationship through cold pressor-induced changes in self-reported anger. With ratings for the sensory component of pain as the DV, the total effect of self-reported suppression on the DV was significant ($p < 0.01$). Cold pressor-induced changes in anger were not associated with ratings for the sensory pain component ($p = 0.06$). Further, the direct effect of self-reported suppression on the sensory pain component ratings with cold pressor-induced changes in self-reported anger included in the model was significant ($p < 0.05$). The 95% bootstrapped confidence interval for the $a \times b$ path included zero. Taken together, these findings suggest that the association between self-reported suppression and sensory pain component ratings were *not* mediated by cold pressor-induced changes in anger. Finally, with ratings on the anger-specific pain component as the DV, there was evidence for mediation by cold pressor-induced changes in anger. Specifically, the total effect of self-reported suppression on ratings for the anger-specific component of pain, the direct effect of self-reported suppression on cold pressor-induced changes in anger and the direct effect of self-reported cold pressor-induced changes in anger on the ratings for the anger-specific pain component were all significant (p values < 0.05). The direct effect of self-reported suppression on anger-specific pain component

Table 3 Simple bootstrapped ($n=1000$) mediation models of self-reported state suppression on mean pain intensity, sensory pain composite, and anger-specific pain composite ratings through cold pressor-induced changes in self-reported anger

Effect	Estimate	Bootstrap SE	<i>t</i>	95% Bias-corrected CI
DV: mean pain intensity				
Suppression-to-anger	0.13	0.06	2.09*	
Anger-to-DV	0.82	0.16	5.02***	
Suppression-to-DV (total effect)	0.23	0.08	2.81**	
Suppression-to-DV (direct effect)	0.13	0.07	1.85	
Suppression-to-DV (indirect effect)	0.11	0.05	^a	(LL=0.03; UL=0.23)
DV: sensory pain composite				
Suppression-to-anger	0.13	0.06	2.09*	
Anger-to-DV	0.16	0.08	1.90	
Suppression-to-DV (total effect)	0.10	0.04	2.68*	
Suppression-to-DV (direct effect)	0.07	0.04	2.07*	
Suppression-to-DV (indirect effect)	0.02	0.02	^a	(LL=-0.02; UL=0.08)
DV: anger-specific pain composite				
Suppression-to-anger	0.13	0.06	2.09*	
Anger-to-DV	0.47	0.09	5.58***	
Suppression-to-DV (total effect)	0.10	0.05	2.20*	
Suppression-to-DV (direct effect)	0.04	0.04	1.05	
Suppression-to-DV (indirect effect)	0.06	0.03	^a	(LL=0.02; UL=0.14)

Table shows unstandardized coefficients for the indirect effect of self-reported suppression (IV) on pain outcomes (DVs specified within Table) through self-reported cold pressor-induced changes in anger (mediator)

CI confidence interval, LL lower limit, UL upper limit

* $p<0.05$, ** $p<0.01$, *** $p<0.001$

^a A p value for the indirect effect is not provided because this value is depends upon a normal distribution of the indirect effect. Given that $a \times b$ coefficients (i.e., indirect effects of IV on DV) are positively skewed, interpretation of this p value is misleading and should thus not be used as a determinant of statistical mediation

ratings of pain with cold pressor-induced changes in anger included in the model was not significant ($p=0.29$). Most critically, the 95% bootstrapped confidence interval for the $a \times b$ path did not include zero, thereby providing quantitative evidence for the simple mediation of the suppression—anger-specific pain component relationship through cold pressor-induced increases in self-reported anger.

Discussion

The assessment of trait Anger-In is confounded by negative emotion. It is thus difficult to conclude whether inhibitory processes have anything to do with pain-related outcomes associated with trait Anger-In [11]. We [10, 11, 26] recently advanced an ironic process model of anger suppression and pain. According to this model, efforts to suppress anger during or following explicit anger-provocation heighten subsequent pain experience through augmented perception of anger-specific elements of pain and pain-induced anger responses that are attributable to the ironic processes of

suppression [22]. Indeed, in prior studies, participants instructed to suppress their emotions during an initial anger-provoking event report greater pain and anger in response to *subsequent* acute pain induction. We have observed significant variability in self-reported suppression in our “do/think anything” control conditions, suggesting the presence of meaningful individual differences in state anger suppression. In the present study, we examined whether self-reported state suppression predicted pain and emotional responses to acute pain induction in a manner consistent with an ironic process model. Moreover, we examined whether suppression resulted in attenuated homeostatic pressor responses to pain concurrent with greater pain intensity.

Variability in self-reported suppression was correlated with mean pain intensity ratings, as well as ratings on sensory and anger-specific pain components. Self-reported suppression was associated with pain-induced increases in anger but not anxiety, positive emotion, or the perception of general distressing qualities of pain. These results are consistent with an ironic process model and highlight previously overlooked individual differences in state anger inhibitory processes with

implications for acute pain responses. Consistent with results from studies in which suppression was experimentally manipulated [10, 26, 27, 49, 50], pain-induced changes in anger statistically mediated the predictive relationship between suppression and pain intensity and, more interestingly, the anger-specific element of pain but not the sensory element. Hence, individual differences in state anger suppression predicted increases in pain that appear to be attributable to paradoxical increases in anger-related thoughts, feelings, and behavioral inclinations. Consistent with prior studies [e.g., 26], although not directly examined in the current investigation, these findings point to the possibility that exaggerated pain perception associated with suppression might have been driven by contamination [24] of perception of the anger-specific element of pain by highly accessible anger-related thoughts and feelings. That suppression was not associated with pain-induced increases in anxiety or reductions in positive emotion lends some additional reason to suspect ironic processes of suppression as the culprit versus some other mechanism, such as depleted resources for self-regulation [51].

We also found that suppression was associated *inversely* with SBP responses to pain. Given that this pattern of results was found concurrent with a *positive* association between suppression and pain intensity, and a negative association between SBP responses and pain ratings, these findings suggest that state suppression of anger might lead to diminished homeostatic pressor responses to pain, thereby augmenting pain sensitivity. That findings were specific to SBP further supports this argument given the role of the systole versus the diastole in mediating pain modulation [52–54]. Anger suppression, and conceptually related variables such as defensive and repressive coping, is believed to represent a potent psychosocial risk factor for development of syndromes associated with significant alterations in the inverse relationship between blood pressure and pain sensitivity [e.g., hypertension 32]. It is thus plausible that suppression may lead to chronic alterations in homeostatic pressor responses to pain, perhaps in conjunction with underlying pathological changes underlying such syndromes. It is intriguing that pain-induced SBP responses were not associated with emotional responses to pain. It is possible that the emotional costs of suppression are mediated primarily by paradoxical cognitive–emotional processes, whereas the painful costs are driven by cognitive–emotional *in addition to* blood pressure-mediated alterations in endogenous descending pain inhibition. It is crucial to note that the mechanisms that underlie the deleterious effects of anger suppression on pain-related processes and outcomes are likely to be multifaceted, including (but not limited to) neurophysiological, cognitive-perceptual, emotional, and socio-cultural. This is an area that will require further study.

It might be argued that self-reported suppression is, akin to Anger-In, largely a proxy for general negative emotion. This, however, was not the case in the present study. That is, individual differences in suppression were not correlated with speech task-induced changes in anger, anxiety, or positive emotion. Hence, suppression assessed “in the heat of the moment” seems to uniquely tap the process of anger inhibition, and so any effects observed for suppression on pain, emotional, and cardiovascular outcomes in the context of acute pain induction cannot be attributed to negative emotion alone. We believe that this method represents an important advance over the utilization of trait Anger-In measures. Although our measure of state suppression included only a single item, other research has uncovered support for theory-derived hypotheses concerning suppression and outcomes employing single-item assessments of suppression [e.g., 55]. Nonetheless, development and validation of a more comprehensive assessment of state-like inhibitory processes is likely needed to advance our understanding of links between state inhibition and deleterious pain—and more general health-relevant outcomes.

In our prior investigations, we examined the effect of the experimental manipulation of anger suppression on pain-related outcomes. It is possible that some of the variability in outcomes in those investigations was attributable to the added experimental demand associated with asking participants to partake in a difficult, stressful task while also suppressing their emotions. That is, anger, irritation, and annoyance in response to pain might have been attributable not to prior suppression per se, but to added stressfulness associated with suppressing mental contents while emotionally aroused and partaking in a difficult cognitively demanding task. Muddying the interpretative waters further, prior investigations did not include other emotion regulation manipulations (e.g., reappraisal). Evidence that suppression exerts effects on pain above and beyond other manipulated forms of emotion regulation would lend further credence to an ironic process model. The results of the present study, in which participants reported engaging in suppression specifically, strongly hint that the process of inhibition has painful consequences. Future investigations should inquire about other possible forms of emotion regulation that participants engage in to further isolate effects of suppression on pain.

Additional hypotheses should be entertained in future research that can provide further insights into cognitive–emotional mechanisms linking anger inhibition to pain. For instance, a two-stage process of anger inhibition followed by post-event rumination has been proposed [56–58]. Specifically, suppression of anger during anger-provocation might contribute to a subsequent inability to stop thinking about the event and its emotional sequelae, thereby coloring the perception of pain, and its irritating, annoying, and frustrat-

ing qualities in particular. Although ironic and two-stage process models likely are not mutually exclusive [for further discussion, see 59], the latter has not received empirical attention in the context of acute and chronic pain and could provide valuable theoretical insights. Focusing on inhibition and rumination in a two-stage manner might also carry unique implications for pain management, such as teaching patients not only effective means to express their emotions [a counter to suppression; 60–62], but also to learn to distract themselves effectively from unwanted thoughts or engage in goal-directed behavior *irrespective* of their current cognitive and emotional state (a counter to post-event rumination). Further clinical implications can be derived if one considers these findings in the context of acceptance-oriented pain interventions [63, 64]. A core rationale of such interventions is that the inhibition or over-control of negative emotional states and emotion-laden thoughts gives way to insidious physical outcomes. Hence, identification of patients who show a propensity to suppress their negative emotional experiences, and possibly anger in particular, might lead to more effective interventions aimed not at altering suppression *per se*, but an individual's relationship to anger-and pain-provoking events through acceptance and mindfulness. The effect of this change in relationship with a given context would decrease the likelihood of engaging in maladaptive cognitive patterns, such as suppression, thereby curtailing short- and longer term ironic effects on pain-related processes and outcomes.

Some additional limitations deserve mention. First, the outcome variables of the present investigation (less BP and HR) were analyzed in a prior study. Hence, these data need to be replicated in novel samples. Second, the current study was conducted on healthy college students of a relatively restricted age range. This renders relevance of these data to clinical pain somewhat tenuous. However, a number of prior studies have revealed maladaptive effects of trait anger suppression [11, 20, 50, 65] and manipulated (state) anger suppression [27] on clinically relevant parameters in chronic pain patients. It is important to keep in mind that this study was carried out first and foremost to examine whether individual differences in self-reported suppression exerted effects on pain responses consistent with an ironic process model and prior findings concerning the experimental manipulation of suppression. Hence, for the purposes of the aims of the current study, the use of healthy young adults likely offered some advantages. For instance, age-related difference in BP and pain sensitivity, differences in clinical pain severity and pathology, comorbid medical and psychiatric conditions (age- and pain-related), and medication confounds were eluded, thus allowing for a relatively clean test of associations between the constructs of interest. Future studies will need to be conducted in pain patient populations to more fully appreciate the potential

clinical merit of our model and to identify the most appropriate course of treatment.

To conclude, individual differences in state anger suppression yield painful and emotional consequences in a similar fashion as the experimental manipulation of anger suppression. Importantly, the individual differences observed in suppression in the present study appear to tap state inhibitory process, unlike individual differences in Anger-In, which covary with a variety of state and trait indices of negative emotion [11]. Hence, the results observed using the novel assessment methodology of the present investigation seem to support ironic effects associated with anger suppression rather than high levels of negative emotion alone. These results add to the growing literature implicating the suppression of anger as a toxic cognitive and behavioral coping strategy in the context of acute and persistent pain.

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