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Keep calm or get excited? Examining the effects of different types of positive affect on responses to acute pain

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ABSTRACT

Researchers typically assume that all forms of positive affect (PA) are equally beneficial for attenuating the physiological stress response. We tested whether this association is more nuanced by examining the role of arousal level of PA on physiological responses to acute pain. Participants (N = 283, 75.6% female, M_{age} = 20.6) were randomized to a low, mid, or high arousal (calm, happy, and excited, respectively) induction condition or to a neutral control and then completed an acute pain-inducing cold pressor task. Sympathetic and parasympathetic responses along with self-reported pain and distress were assessed. Results indicated that the calm condition had a flatter sympathetic reactivity and subsequent recovery compared with the control condition. Additionally, calm and excited were associated with steeper *increases* in parasympathetic reactivity versus controls. These results support past PA stress buffering findings and indicate that not all types of PA are equal when it comes to improving the pain stress response.

Abbreviations: PA: positive affect; PEP: pre-ejection period; RMSSD: root mean square of successive differences

In recent years, there has been an increasing interest in the way in which affect can inhibit or exacerbate responses to pain. In general, positive affect (PA) is thought to inhibit pain. One important yet understudied issue in this literature, however, is the possibility that different types of PA may impact responses to acute pain differentially. This may be especially true when PA types vary in arousal, a factor long thought to alter the pain experience (Haslam, 1967). To address this question, the current study examines how different types of induced and arousal-varied PA influence responses to acute pain.

Broadly, experimentally induced PA has been associated with less acute pain (e.g., Bruehl et al., 1993; Rhudy & Williams, 2005; Weisenberg et al., 1998; Zelman et al., 1991). For example, participants asked to re-create a pleasant memory self-reported less pain from a finger pressure pain task compared with neutral controls (Bruehl et al., 1993). Additionally, the pain literature acknowledges the potential importance of PA arousal on pain (e.g., Rhudy & Meagher, 2001) and tends to show less self-reported pain and higher pain tolerance when relaxation (i.e. similar to a type of low-arousal PA) is induced through decreasing respiration rate, progressive muscle relaxation, guided imagery, or meditation (e.g., Bobey & Davidson, 1970; Bruehl et al., 1993; Mandle et al., 1996; Schaffer & Yucha, 2004;

Wescott & Horan, 1977). However, less is known about the influence of PA arousal on autonomic responses to acute pain. The overlapping neurocircuitry responsible for emotion, pain sensation, and the autonomic nervous system (e.g., Appelhans & Luecken, 2008; Benarroch, 2001) suggests that PA might inhibit both the perception of pain and autonomic responses to pain (e.g., Bruehl et al., 1993; Rhudy & Meagher, 2001).

Further evidence that PA inhibits self-reported and autonomic stress responses to pain is provided by the PA-Stress buffering model which posits that PA reduces both the perceptions of and physiological responses to stress (Pressman & Cohen, 2005). However, in most studies, different PA adjectives (and arousal levels) are averaged together or only a single PA type is contrasted against a neutral control instead of examining different PA types separately (Mandle et al., 1996; Martin, 2001; Matz & Brown, 1998; Pressman & Cross, 2018). This leaves many open and unanswered questions about what types of PA are most helpful in the context of stressful pain stimuli.

At rest, PA arousal is associated with physiological changes in the autonomic nervous system (Thayer, 1967, 1970). For example, feeling excited and enthusiastic (high-arousal PA) is associated with increased heart rate activity, while feeling peaceful and calm (low-arousal PA) has the

opposite effect (Kreibig, 2010; Levenson, 2014; Shiota et al., 2011; Witvliet & Vrana, 1995). These differences in autonomic patterns prior to experiencing acute pain could alter the phasic autonomic stress response to pain due to the law of initial values (Berntson et al., 1994). This law states that the level of physiological response depends on the initial level of physiological activation. As such, to truly understand the impact PA has on the physiological response to acute pain, we must examine the changes that occur during pain *from* the PA induction rather than from baseline.

In the current study, we explore two questions no one has examined before. First, we ask what type of PA (low-arousal calm, mid-arousal happy, or high-arousal excited) is most helpful for altering physiological and self-reported responses to an acute and stressful pain task. Next, we also explore what physiological pathway drives this effect: sympathetic versus parasympathic activity. To do so, we assessed the influence of different types of PA on self-reported and autonomic cardiovascular responses to acute pain by randomly assigning participants to one of three PA conditions that varied by arousal level or a neutral control condition and then had participants engage in a standardized cold pressor task. While no one has manipulated different PA types in a single pain study before, based on past-related relaxation studies (e.g., Bobey & Davidson, 1970; Bruehl et al., 1993; Mandle et al., 1996; Schaffer & Yucha, 2004; Wescott & Horan, 1977) and our own theorizing, we hypothesized that the low-arousal calm condition would best buffer the physiological response (i.e. lower sympathetic activation, lower parasympathetic withdrawal) to acute pain compared with the other conditions. If so, this would demonstrate a physiological mechanism through which relaxation and other low arousal PA inducing interventions are beneficial to acute pain.

Methods

Participants

The sample consisted of 283 undergraduate students at the University of California, Irvine who were either recruited through a) the Department of Psychological Science online research subject pool interface (SONA System) and received class credit for their participation or b) through flyers hanging around campus and received 20 USD for their participation. Participants were excluded from the study if they had ever been diagnosed with or currently had any of the following: a psychological disorder (e.g., depression) for which they were being treated (medication or therapy), cardiovascular disease (e.g., a heart condition), or a connective tissue disease (e.g., Raynaud's disease). Participants were also excluded if they had facial musculature disorders or

were not fluent in English. One participant was excluded from analyses for being an extreme outlier across most cardiovascular outcome variables. Additionally, one participant experienced an adverse event and their data were subsequently removed from all analyses. For analyses, we included only individuals who completed the full 2-minute cold pressor task ($n = 195$).

Procedure

After providing written informed consent, participants completed the screening questionnaire. Eligible participants next completed baseline questionnaires assessing characteristics such as demographics, state PA, and stress. Upon completion of these questionnaires, electrodes were placed on participants to record both electrocardiograph (Lead II configuration) and impedance cardiography via BioLab 3.0.13. Participants completed a five-minute resting baseline period. Participants were randomly assigned to one of three PA-induction writing tasks: low-arousal calm, mid-arousal happy, high-arousal excited, or a neutral writing task. They were instructed to write for five minutes about ' … a time in your life when you were the most (relaxed and/or calm; happy and/or content; excited and/or elated) … '. Participants were instructed to 'Try to relive the precise details as much as possible, re-experiencing the events involved' and encouraged to continue writing and thinking about the event for the entire writing period. Those in the neutral control condition wrote about their typical morning routine. Following the writing task, participants answered a questionnaire regarding their state emotion as a manipulation check.

Next, participants completed the cold pressor task. During this task, participants put their non-dominant hand in a bucket of ice water (between 3.8°C and 4.2°C) and attempted to keep their hand in the water for an uninformed ceiling of two minutes. After the cold pressor, participants sat quietly for a five-minute recovery period. Upon completing additional questionnaires regarding pain experienced during the cold pressor task, participants completed a three-minute refresher mood induction, a second manipulation check and an additional task unrelated to our PA-pain research questions. The participants then completed a final set of questionnaires then were debriefed and dismissed. The University of California, Irvine Office of Research Institutional Review Board approved this human subjects study (HS#2013-9798).

Measures

Demographics
Age, sex, and racial/ethnic background were assessed via self-report. Additionally, height and weight were

measured using a medical-grade scale in order to calculate body mass index.

State affect and stress

State affect was assessed using a modified and shortened version of the Profile of Mood States (POMS) Questionnaire (McNair et al., 1971; Usala & Hertzog, 1989). Participants completed the questionnaire three times (at baseline before the induction, after the induction, after recovery from the passive stressor). Subscale scores were calculated by taking the average of the ratings for each self-reported state emotion of the corresponding subscale. For this study, the vigor subscale was used as a measure of high-arousal excited and comprised the following items: 'lively' and 'enthusiastic'. Across ratings, the vigor subscale demonstrated internal consistency, Cronbach's α =.71-.86. The well-being subscale was used as a measure of mid-arousal happy and included 'cheerful' and 'happy'. These items showed internal consistency across ratings, Cronbach's α = .86-.92. The calm subscale was used as a measure of low-arousal calm and included the items 'calm' and 'relaxed' (across ratings, Cronbach's α = .89-.94). We also included the items 'overwhelmed' and 'stressed' (across ratings, Cronbach's α = .81-.84) as a stress subscale (Miller et al., 2004). Responses ranged from 0 (not at all accurate) to 4 (extremely accurate).

Self-reported response to pain

Participants were asked 'What was the maximum level of pain you experienced?' and 'Please rate the distress you experienced during the cold-water task' after the cold pressor task. Responses ranged from 0 (no pain at all/no distress at all) to 100 (worst possible pain/highest level of distress).

Autonomic response measures

Heart rate was recorded as beat-to-beat intervals using BioLab 3.0.13. All raw electrocardiograph (ECG) data were transferred into Mindware HRV 3.0.22 software and edited when artifacts prevented accurate heart rate calculation. Using the Mindware HRV software, dZ/dt was used to approximate respiration rate for each participant (so that respiration rate could be used as a control variable). R peaks were marked unless artifacts made it impossible to identify them. If this occurred, that part of the segment was removed and research assistants ensured the remaining data in that 60-second segment were at least 30 seconds of continuous data. If 30 seconds of data could not be extracted from a 60-second segment, then it was not included in analyses. Once data were cleaned, heart rate (to be used as a control variable) and the root mean-squared

successive differences (RMSSD) between adjacent, normal R-R intervals were measured in milliseconds and calculated for each 60-second segment of data collected. RMSSD is a valid, time-domain measure of the parasympathetic influence on the heart (Hill et al., 2009; Kleiger et al., 2005). Higher RMSSD indicates greater parasympathetic influence.

All raw ECG and impedance cardiography (ICG) data were transferred into Mindware IMP 3.0.22 software and edited in 2 steps. The first step involved the same process of editing the ECG described above and the second step involved visually inspecting the ensemble averages. The B point was visually inspected and estimated, if needed, using the R-Z interval as described by Lozano et al. (2007). Once data were cleaned, pre-ejection period (PEP) was measured in milliseconds and calculated for each 60-second segment of data collected. PEP is a valid, time-domain measure of cardiac contractility and a marker of the sympathetic influence on the heart (Cacioppo et al., 1994; Sherwood et al., 1990). Lower PEP indicates greater sympathetic influence.

Statistical analysis plan

Analyses were conducted using Stata version 14.2 (Stata Corp.). RMSSD was not normally distributed so was log-transformed; however, while all inferential statistics use this transformation, descriptive data and data provided for RMSSD in tables and figures present raw, untransformed values for ease of comparison across studies. Descriptive statistics were examined among relevant variables across the entire sample and within each condition. Following this, we ran several manipulation checks to show that we indeed induced the correct arousal level of PA during the PA manipulation and that we increased self-reported stress during the cold pressor.

To test the PA manipulation, we ran a series of one-way ANOVAs with PA condition as the independent variable and the scores for the POMS subscales taken immediately following the PA manipulation as the dependent variables. To confirm the cold pressor task induced psychological stress, we conducted paired samples t-tests comparing the means of the self-reported stress subscale immediately before and after the cold pressor.

Following the manipulation checks, we tested for PA condition differences in self-reported pain after the cold pressor. Multilevel piecewise regression analyses were conducted to assess the trajectories of RMSSD and PEP over the course of the cold pressor task. RMSSD and PEP were clustered within person using multilevel modeling in all analyses because within person change in RMSSD

and PEP over time violates the assumption of independence of residuals for an ordinary least square regression analysis and violates the homogeneity of variances assumption for repeated measures analysis of variance. Further, piecewise regression analyses allow for examining nonlinear change in time-series data where a known point of change happens (e.g., the point between stress reactivity and stress recovery; Kim et al., 2000; Rabe-Hesketh & Skrondal, 2012). Given that the end of the stressor and the beginning of the recovery period represented a distinct event, a knot was set between reactivity and recovery using the mkspline command in Stata. Placing the knot here estimates the linear reactivity trajectory and the linear recovery trajectory within the same model (as opposed to computing different regression equations for each line segment). The interaction between dummy-coded condition and time was tested in each model. Both RMSSD (log transformed) and PEP were dependent variables of interest and were modeled separately.

Maximum likelihood tests were conducted to examine whether the following potential covariates improved the model: age, ethnicity, body mass index, and sex. For RMSSD, respiration rate was also considered as potential covariates. To maintain parsimony, covariates that did not improve model fit were not included in the final model. In sum, two separate multilevel piecewise regression models estimated the effect of dummy-coded condition on 1) RMSSD during the cold pressor task and 2) PEP during the cold pressor task. Since our hypotheses focused on low arousal calm condition, we tested whether each condition slope differed from zero and whether the low arousal calm condition slopes differed from the other condition slopes. We also report whether slopes differ from the neutral control condition.

There is little guidance in the literature for calculating statistical power for multilevel piecewise regression models; therefore, we attempted to obtain a sample similar in size to past relevant research (Kraft & Pressman, 2012).

Results

Participant characteristics

Descriptive statistics for participant demographics (sex, age, body mass index) and for RMSSD and PEP during mood induction are presented in Table 1 for each condition.

Manipulation checks

Did the PA manipulation induce the correct type of PA?

There were significant effects of the mood manipulation on self-reported low-arousal PA ($F_{(3, 191)} = 4.60$, $p = .004$, $\eta^2 = .067$), mid-arousal PA ($F_{(3, 191)} = 7.10$, $p < .001$, $\eta^2 = .100$), and high-arousal PA ($F_{(3, 191)} = 8.16$, $p < .001$, $\eta^2 = .114$) following the initial mood induction. Consistent with anticipated manipulation effects, compared to the other groups, the calm condition reported the highest mean levels of low-arousal PA ($M = 2.67$, $SD = 1.12$) and the excited condition reported the highest mean levels of high-arousal PA ($M = 1.96$, $SD = 1.12$); however, the excited condition also reported the highest mean levels of mid-arousal PA ($M = 2.42$, $SD = 1.02$). This point will be discussed in our limitations section.

Did the cold pressor task induce self-reported stress?

Participants self-reported significantly higher stress following the cold pressor task ($M = 0.83$, $SD = 0.90$, $t_{(193)} = -4.75$, $p < .001$, $M_{diff} = -0.19$, 95% CI [−0.27, −0.11], $d = 0.34$) compared to their self-reported stress immediately before the cold pressor ($M = 0.64$, $SD = 0.87$).

Did PA condition influence self-reported responses?

Participants self-reported moderate-to-high levels of distress and pain experienced during the cold pressor task. Participants in the high arousal excited condition

Table 1. Descriptive statistics (n = 195).

Study variables	Neutral control (n = 44)	Calm condition (n = 54)	Happy condition (n = 44)	Excited condition (n = 53)	χ^2 or F	p
Females [a]	30	40	31	37	0.48	0.92
Race/ethnicity [a]					12.87	0.61
White	8	13	9	15		
Latino	10	19	17	17		
East Asian	21	16	15	16		
Other	5	6	3	5		
Age [b]	20.8 (3.2)	20.7 (2.9)	19.6 (1.5)	20.1 (2.3)	2.18	0.09
Body Mass Index [b]	25.5 (5.9)	24.5 (4.8)	26.0 (5.6)	24.2 (7.1)	0.92	0.43
Mood induction PEP [b]	112.5 (6.0)	111.7 (5.7)	112.1 (5.5)	110.7 (8.6)	0.65	0.59
Mood induction RMSSD [b]	44.3 (28.0)	41.5 (23.1)	41.9 (21.8)	34.9 (18.6)	1.74	0.16

[a]Numbers in table are the sample size, χ^2 statistic and p value reported in columns on right [b] Numbers in table are the raw mean (standard deviation), F statistic and p value reported in columns on right.

reported the highest amount of maximum pain experienced ($M = 74.96$, $SD = 18.41$) followed by the mid arousal happy condition ($M = 73.64$, $SD = 22.61$), the low arousal calm condition ($M = 69.60$, $SD = 23.15$), and the neutral control condition ($M = 68.90$, $SD = 24.86$). In terms of self-reported distress experienced during the cold pressor task, the neutral control condition reported the highest level of distress ($M = 58.67$, $SD = 28.06$) followed by the high arousal excited condition ($M = 57.37$, $SD = 24.06$), the mid arousal happy condition ($M = 54.61$, $SD = 26.89$), and the low arousal calm condition ($M = 53.00$, $SD = 26.49$). However, there were no significant differences between conditions in self-reported pain or distress from the cold pressor task (p's > .45).

Did PA condition alter physiological responses?

Sympathetic response

For each experimental condition, minute-by-minute PEP response to the painful, cold pressor task is depicted in Figure 1. Restricted maximum likelihood tests revealed that adding a random intercept (AIC = 9,607.10; BIC = 9,633.54) significantly improved model fit compared with a model without it (AIC = 10,558.89; BIC = 10,580.04; likelihood ratio χ^2 (1) = 953.79, $p < .0001$) and adding a random slope (AIC = 9,584.25; BIC = 9,615.98) significantly improved model fit compared with a model with only a random intercept (likelihood ratio χ^2 (1) = 24.85, $p < .0001$). Maximum likelihood tests revealed that no potential covariates

improved model fit (p's > .05) and thus none were included in final models predicting PEP response to the cold pressor task.

As hypothesized, the low arousal calm condition buffered the sympathetic response to acute pain. Specifically, both the low arousal calm and mid arousal happy condition slopes during reactivity did not significantly differ from zero (calm: $dy/dx = 0.04$, $z = 0.15$ $p = .882$, 95% CI [−0.43, 0.50]; happy: $dy/dx = −0.99$, $z = −1.59$, $p = .112$, 95% CI [−2.22, 0.23]) but the other conditions exhibited negative slopes that significantly differed from zero (control: $dy/dx = −0.73$, $z = −2.95$, $p = .003$, 95% CI [−1.21, −0.24]; excited: $dy/dx = −0.94$, $z = −2.22$, $p = .027$, 95% CI [−1.77, −0.11]). When comparing reactivity slopes to each other, participants in the neutral control condition had a significantly greater decrease (i.e. greater sympathetic activation) compared with participants in the low arousal calm condition, ($b = 0.76$, robust $SE = 0.34$, $z = 2.23$, $p = .026$, 95% CI [0.09, 1.43]). However, the neutral control condition did not significantly differ from the mid arousal happy condition, ($b = −0.27$, robust $SE = 0.67$, $z = −0.40$, $p = .691$, 95% CI [−1.59, 1.05]), or the high arousal excited condition, ($b = −0.21$, robust $SE = 0.49$, $z = −0.44$, $p = .663$, 95% CI [−1.17, 0.75]). The low arousal calm condition did not significantly differ from the mid arousal happy condition ($b = −1.03$, robust $SE = 0.67$, $z = −1.54$, $p = .124$, 95% CI [−2.34, 0.28]) but was significantly different than the high arousal excited condition, $b = −0.97$, robust $SE = 0.49$, $z = −2.01$, $p = .045$, 95% CI [−1.93, −0.02].
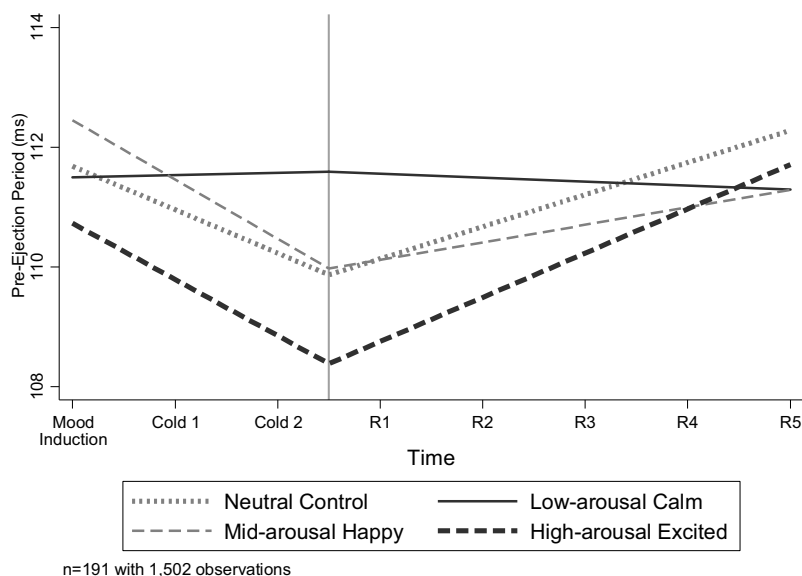

Figure 1. PEP response (ms) to the cold pressor task. Cold = cold pressor task; R = recovery; 1, 2, 3, 4, 5 = minute in respective task. Vertical reference line indicates the completion of the cold pressor task and the beginning of R.

During recovery, participants in both the low arousal calm and mid arousal happy conditions did not experience a significant change in sympathetic activation (calm: $dy/dx = -0.06$, $z = -0.43$, $p = .670$, 95% CI [−0.36, 0.23]; happy: $dy/dx = 0.29$, $z = -1.61$, $p = .106$, 95% CI [−0.06, 0.65]) likely because they had no spike in reactivity to recover from. In contrast, participants in the high arousal excited and neutral control conditions experienced a significant decrease in sympathetic activation likely due to the completion of the cold pressor task (excited: $dy/dx = 0.74$, $z = 3.76$, $p < .001$, 95% CI [0.35, 1.13]; control: $dy/dx = 0.54$, $z = 3.56$, $p < .001$, 95% CI [0.24, 0.83]). When comparing these recovery slopes to one another, the neutral control condition ($b = -0.60$, robust $SE = 0.21$, $z = -2.81$, $p = .005$, 95% CI [−1.02, −0.18]) and the high arousal excited ($b = 0.81$, robust $SE = 0.25$, $z = 3.23$, $p = .001$, 95% CI [0.32, 1.29]) conditions both had greater decreases in sympathetic activation compared with the low arousal calm condition. All other groups did not have significant differences between recovery slopes (happy vs. calm: $b = 0.36$, robust $SE = 0.24$, $z = 1.51$, $p = .131$, 95% CI [−0.11, 0.82]; happy vs. neutral: $b = -0.24$, robust $SE = 0.24$, $z = -1.03$, $p = .302$, 95% CI [−0.71, 0.22]; excited vs. neutral: $b = 0.20$, robust $SE = 0.25$, $z = 0.82$, $p = .412$, 95% CI [−0.28, 0.69]).

Parasympathetic response

For each experimental condition, minute-by-minute RMSSD response to the painful, cold pressor task is depicted in Figure 2. Restricted maximum likelihood tests revealed that adding a random intercept (AIC = 1,070.96; BIC = 1,097.44) significantly improved model fit compared with a model without it (AIC = 2,346.91; BIC = 2,368.10; likelihood ratio χ^2 (1) = 1,277.95, $p < .0001$) and adding a random slope (AIC = 1,029.70; BIC = 1,061.47) significantly improved model fit compared with a model with only a random intercept (likelihood ratio χ^2 (1) = 43.26, $p < .0001$). An unstructured covariance matrix also significantly improved the model fit (AIC = 1,027.81; BIC = 1,064.88; likelihood ratio χ^2 (1) = 3.89, $p = .049$) compared with a model with a random intercept and random slope. Maximum likelihood tests revealed that no potential covariate significantly improved model fit (p's > .05) and thus were not included in the final model.

During reactivity, all conditions had a significant increase in parasympathetic activation in response to the cold pressor task (calm: $dy/dx = 0.15$, $z = 6.72$, $p < .001$, 95% CI [0.10, 0.19]; happy: $dy/dx = 0.09$, $z = 4.22$, $p < .001$, 95% CI [0.05, 0.14]; excited: $dy/dx = 0.14$, $z = 7.18$, $p < .001$, 95% CI [0.10, 0.18; control: $dy/dx = 0.07$, $z = 2.89$, $p = .004$, 95% CI [0.02, 0.12]). However, there were some significant differences in the magnitude of these increases. For example, participants in the low arousal calm ($b = .08$, robust $SE = .03$, $z = 2.32$, $p = .020$, 95% CI [0.01, 0.14]) and high arousal excited conditions ($b = .07$, robust $SE = .03$, $z = 2.31$, $p = .021$, 95% CI [0.01, 0.13]) had a significantly greater parasympathetic activation during reactivity compared with the neutral control condition. All other groups did
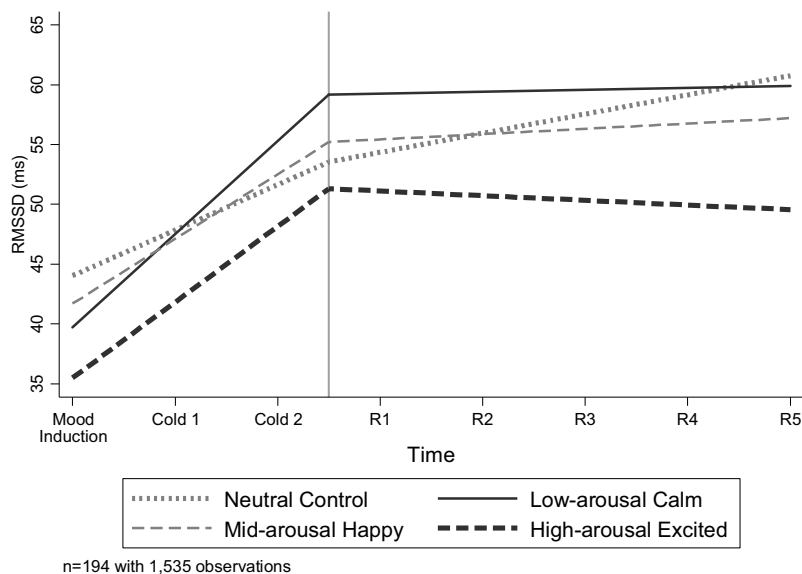

Figure 2. RMSSD response (ms) to the cold pressor task. Cold = cold pressor task; R = recovery; 1, 2, 3, 4, 5 = minute in respective task. Vertical reference line indicates the completion of the cold pressor task and the beginning of R.

not have significant differences between reactivity slopes (calm vs. happy: $b = -.05$, robust $SE = .03$, $z = -1.68$, $p = .092$; calm vs. excited: $b = -.003$, robust $SE = .03$, $z = -0.11$, $p = .916$, 95% CI [−0.06, 0.06]; happy vs. neutral: $b = .02$, robust $SE = .03$, $z = 0.71$, $p = .479$, 95% CI [−0.04, 0.09]).

For parasympathetic recovery, only the neutral control condition was significantly different from zero and was surprisingly positive suggesting a continued increase in parasympathetic activity, $dy/dx = 0.03$, $z = 2.02$, $p = .044$, 95% CI [0.001, 0.067]. None of the other conditions had recovery slopes that were significantly different from zero (calm: $dy/dx = 0.01$, $z = 0.41$, $p = .680$, 95% CI [−0.02, 0.03]; happy: $dy/dx = 0.01$, $z = 0.86$, $p = .390$, 95% CI [−0.02, 0.04]; excited: $dy/dx = 0.004$, $z = 0.29$, $p = .770$, 95% CI [−0.02, 0.03]). Additionally, when comparing the slopes to one another, there were no significant differences between conditions (calm vs. happy: $b = .01$, robust $SE = .02$, $z = 0.31$, $p = .753$, 95% CI [−.03, .05]; calm vs. excited: $b = -0.002$, robust $SE = .02$, $z = -0.09$, $p = .929$, 95% CI [−0.04, 0.04]; calm vs. neutral: $b = -.03$, robust $SE = .02$, $z = -1.28$, $p = .202$, 95% CI [−0.07, 0.02]; happy vs. excited: ?; happy vs. neutral: $b = -0.02$, robust $SE = .02$, $z = -0.99$, $p = .322$, 95% CI [−0.06, 0.02]; excited vs. neutral: $b = -0.03$, robust $SE = .02$, $z = -1.37$, $p = .172$, 95% CI [−0.07, 0.01]).

Discussion

The present study examined how arousal levels of PA impact autonomic responses to acute pain. This study is the first to examine two unanswered research questions about the nuances of the PA-pain interaction. First, it explores the specific arousal types of PA underlying past observed PA-acute pain buffering effects. Second, it examines the precise autonomic mechanisms through which PA impacts cardiovascular responses to acute pain by examining both sympathetic and parasympathetic outcomes.

Consistent with hypotheses, low arousal calm, as compared to the other PA conditions, impacted both branches of the autonomic nervous system by increasing parasympathetic activation during reactivity and attenuating the sympathetic response trajectory. In response to acute pain, generally, participants tend to exhibit parasympathetic withdrawal and sympathetic activation (Koenig et al., 2014). In our study, low arousal calm induction may counteract this natural response to pain at the physiological level. This is especially notable in Figure 1 where the low arousal calm condition has a trajectory that is essentially a horizontal line as compared to the traditional sympathetic reactivity patterns

of the high arousal excited and neutral control conditions. Thus, this study provides potential autonomic mechanisms for past studies showing that a range of relaxation or meditation activities can alter the pain experience (Bobey & Davidson, 1970; Bruehl et al., 1993; Carroll & Seers, 1998; Good, 1996; Mandle et al., 1996; Roykulcharoen & Good, 2004; Schaffer & Yucha, 2004; Seers & Carroll, 1998; Wescott & Horan, 1977). Future studies should examine whether this finding is specific to acute cold pain, or whether it is relevant to other acute pain types (e.g., pressure pain, ischemic pain) more broadly.

As compared to low arousal calm, high arousal excitement produced markedly different stress patterns during recovery to acute pain. Specifically, unlike the low arousal calm condition's flattened sympathetic response, high arousal excitement was associated with increased sympathetic activation followed by a steep return to baseline. This highlights the critical problem of assuming that all arousal levels of PA operate equally in times of stress as well as the more typical averaging approach to the study of PA and health. That said, while high arousal excitement was associated with sympathetic activation during acute pain, it was also associated, like low arousal calm, with significant parasympathetic activation. In other words, the high arousal excitement condition exhibited a coactivation of the parasympathetic and sympathetic nervous systems while the low arousal calm condition exhibited parasympathetic activation only. It is possible then that low arousal calm helped participants reduce their sympathetic reactivity to pain (which did not differ from zero) by increasing parasympathetic activation while high arousal excitement did not activate the parasympathetic system enough to attenuate the sympathetic response. We should note that while this indicates that feeling excited may not be helpful for improving pain, other studies have shown high arousal positive emotions to help with conquering other forms of stress (Brooks, 2014).

One surprising finding was that all conditions, including the neutral control, induced parasympathetic activation *during* reactivity to pain. Typically, researchers observe parasympathetic withdrawal (i.e. deactivation) in response to acute pain (Koenig et al., 2014). However, the increased parasympathetic activity during pain in this study is not unique to this sample. For example, one similar study of healthy undergraduate students assessed parasympathetic activation (measured by respiratory sinus arrhythmia) during a resting baseline, relaxation period, a worrying period, and then a subsequent anticipation stressor where the participants were told they would give a 10 minute impromptu

speech on topics that the experimenter would reveal after the participant sat quietly for three minutes (Hofmann et al., 2005). Participants exhibited a parasympathetic *withdrawal* from baseline to relaxation and a further decline in parasympathetic activation from relaxation to the worry period. However, during the anticipation period, parasympathetic activation began to increase with each minute of anticipation. Thus, our results add to the complex literature on cardiovascular responses to various stressors and indicate that in healthy undergraduate experimental affect and stress research studies there may be some unique patterns (e.g., past study experience, coping strategies, or even the vigor of this particular sample).

As shown in the manipulation checks of the mid arousal happy condition, it is not entirely clear if the psychological and physiological effects of being happy are drastically different from those of excitement. While excitement versus calm was always the more distinct arousal comparison, for completeness of assessing the full range of PA arousal as well as the general public interest in happiness and health, we did include it. Results indicated that the mid arousal happy condition seemed to mirror more the physiological effects of low arousal calm. This demonstrates that sometimes this mid arousal emotion may produce effects similar to low arousal calm while at other times may produce effects similar to high arousal excitement. These types of findings are especially critical to individuals interested in positive psychology interventions as a matter to improve stress. Many interventions focus on increasing this mid arousal range of positivity (e.g., gratitude interventions, focusing on three good things, savoring positive events), which have excellent effects on changing affect (Sheldon & Lyubomirsky, 2007; Sin & Lyubomirsky, 2009). However, based on these findings, approaches that increase low arousal positivity may be most helpful in the context of acute pain.

The difficulty in separating happiness from excitement in our mood manipulation is one limitation of this study. People in the high arousal excited condition also rated their levels of happiness similarly to those in the mid arousal happy condition. This raises the question of whether it is at all possible to feel excited without also feeling happy, and the possibility that some of the observed excitement effects are also due to the associated happiness. The lack of happiness effects in the mid arousal condition, however, makes this unlikely. Another potential limitation is that feelings of calm, happiness, and excitement may vary in valance as well as arousal. Feelings of happiness, for example, may be more positively valanced than being excited or calm (Barrett & Russell, 1998). This study instructed

participants to write about an event where they felt a specific discrete emotion (i.e., calm, happy, or excited). Future iterations of this study could include instructions that exclusively focus on manipulations of arousal level (low arousal, mid arousal, or high arousal positive affect). Additionally, future studies should replicate these findings in both experimental manipulations of affect and by examining the influence of naturally-occurring trait affect on responses to acute pain. Generalizability is limited to samples of healthy college students similar to this study's population. While our sample was racially and ethnically diverse, we were not able to examine racial/ethnic differences due to small samples within racial/ethnic group by condition. Future research should more closely examine whether there are racial/ethnic differences in the impact of PA on responses to acute pain, especially given the known cultural differences in affective ideals and norms (Ruby et al., 2012; Tsai, 2007; Tsai et al., 2006).

This study provides an important next step in unpacking the relationship between PA and acute pain by demonstrating that the benefits of PA on physiological responses depend on PA arousal level. These findings also add to the PA-health literature showing that PA arousal levels matter for outcomes like infectious illness, sleep outcomes, and even longevity (Cohen et al., 2006; Petrie et al., 2018; Pressman & Cohen, 2012; Pressman et al., 2017). Only by understanding these nuances can we finally provide correct and sensitive advice as well as appropriate interventions to help individuals handle acute pain and improve health successfully.

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Data availability statement

The data that support the results of this study are available on The Open Science Framework at https://doi.org/10.17605/OSF.IO/WZQ3E.

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