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The Effect of Acute Physical Pain on Subsequent Negative Emotional Affect: A Meta-Analysis

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Abstract

Although it is clear that most people attempt to avoid pain and often find it unpleasant in the moment, research suggests that changes in affect following pain are not universally negative. To help advance our understanding of pain-affect relationships, the goal of the current study was to conduct a meta-analysis of studies examining changes in negative affect, as defined by subjective experience and psychophysiology, following the experience of acute laboratory pain. We identified 22 effect sizes from 17 different studies (N = 1717). We tested several different hypotheses based primarily on theories of nonsuicidal self-injury (NSSI), with mixed support. Our main findings were that pain had a small to medium effect in reducing negative affect (d_{av} = −.35, 95% CI [−.58, −.12]), and most robustly regulated negative affect in the context of a negative affect induction (d_{av} = −.37, 95% CI [−.73, −.02]) relative to neutral affect induction (d_{av} = .08, 95% CI [−.09, .26]). Similar reductions were also seen following painful and nonpainful stimulation, calling into question whether pain is necessary or whether any stimulation is sufficient. The results lead to a number of questions to be addressed in future research.

Keywords

Pain; Negative Affect; Nonsuicidal Self-injury; Meta-analysis

As much as people try to avoid the experience of pain, it is inevitable. In some cases, it happens accidentally (e.g., stubbing your toe, burning your tongue on hot coffee) and in other cases, pain is inflicted purposefully (e.g., eating spicy foods, exercising). Although pain is a part of life, persons and contexts can vary in terms of the meanings attributed to pain. On the one hand, pain is unpleasant and associated with negative outcomes (e.g., injury, death). On the other hand, even unpleasant pain can be associated with positive feelings (e.g., pride, group solidarity) or relief from negative affect (Bastian, Jetten, Hornsey, & Leknes, 2014). This meta-analysis was conducted to address the extent to which pain can alter subsequent affect.

Understanding changes in affect following pain can inform theory and research in many areas of psychology. From a neuropsychological perspective, it may help clarify the overlap

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between neural systems that implement pain processing with those that implement emotional processing (Eisenberger, 2011; Ochsner & Gross, 2005; Roth-Deri et al., 2008), which may have further implications for both medical and psychological interventions (e.g., chronic pain patients). From a personality and individual difference perspective, understanding the emotional responses to pain may help explain why some people are more likely engage in behaviors that involve pain or a high likelihood of pain (e.g., sky diving). Similarly, the area of health psychology can benefit from an understanding of why some people engage in healthy exercise, despite the pain, information that can help inform interventions designed to increase exercise. Finally, psychopathology researchers benefit from insights into clinical behaviors that involve seeking out pain. One such behavior of particular interest to this study is nonsuicidal self-injury (NSSI), which is defined as intentional destruction of body tissue in the absence of suicidal intent (Nock, 2009). Despite the absence of suicidality in the definition of NSSI, it is longitudinally related to suicide attempts (Hamza et al., 2012). Interestingly, most theories of NSSI suggest that the behavior is used to reduce the experience of intense, uncontrollable negative emotions (Nock, 2009; Selby & Joiner, 2009). Thus, understanding how pain affects emotions might inform treatments of these behaviors and ultimately prevent loss of life. To help advance our understanding of changes in affect following pain, the goal of the current study was to conduct a meta-analysis of studies examining changes in negative affect, as defined by subjective experience and psychophysiology, following the experience of acute laboratory pain.

Conceptualization of Pain and Affect

A common definition of pain is “an unpleasant sensory and emotional experience associated with actual or potential tissue damage” (Mersky & Bogduk, 1994, p. 210). This definition illustrates that pain involves an objective sensory component (nociception), and an unpleasant subjective appraisal, the latter complicating distinctions between pain and affect. Pain and negative affect can be distinguished in at least two ways. First, there is typically a dissociation between the affective and sensory components of pain, in terms of localization in the brain, with sensory cortex (e.g., S1) being correlated with the sensory component and portions of the anterior cingulate being correlated with pain unpleasantness (Rainville et al., 1997: Rainville, 2002). Second, although there is overlap in the neural networks that implement pain and negative affect (e.g., the cingulate cortex), there are clear dissociations (Vogt, 2005). For instance, the pregenual anterior cingulate cortex is responsive to pain but not negative affect (Vogt, 2005), suggesting that they may be distinct emergent properties.

We focused on acute versus chronic pain in this meta-analysis partly to facilitate distinctions between pain and affect. Although pain has a subjective appraisal component, the experience of acute pain is time-limited, allowing it be differentiated from the affective experience that follows it. Additionally, acute pain induced in the laboratory allows for experimental control to draw causal inferences, and is more similar to clinical phenomena that researchers may want to generalize to. For example, acute pain can simulate the experience of NSSI, as they are both short in duration, and have a more definite endpoint. For the remainder of the paper, we will use the word pain to refer to acute pain.

In a laboratory setting, pain stimuli can be administered in several different ways. Extreme temperatures can be manipulated either by a cold pressor test, which involves immersing a participant's hand in a container of cold water for a certain amount of time, or thermodes attached to the body, which can produce hot or cold temperatures. Electrical shock can be administered through electrodes and involves short pulses of electricity at varying intensities. Another way to administer painful stimuli is through the use of a pressure algomenter. An algometer is used to administer different forces of pressure to a small area of the body, such as the palm of the hand. Because different neuronal pathways implement the experience of different pain modalities (Julius & Basbaum, 2001), it is possible that the relation between pain and emotion may differ depending on modality of pain administration.

Finally, we focus on changes in *negative* affect, an unpleasant state low in valence and high in arousal (Watson, Clark, & Tellegen, 1988; Yik et al., 2011), following pain, and assess different operationalizations of negative affect in this meta-analysis. Negative affect plays an important role in NSSI and suicidal behaviors (Nock, 2009; Joiner, 2005), which are the areas where the implications for this research are perhaps strongest. Our examination of negative affect involved using both experiential and psychophysiological units of analysis, and thus capitalizing on the advantages (and considering the limitations) of each of these two measurement approaches. For example, self-report is subject to individual differences (e.g. gender, culture) in willingness to express emotions, and in awareness of one's valence and arousal (Mauss & Robinson, 2009). Psychophysiological measures are less subjective than self-report, as they are for the most part involuntary. The major limitation of physiological measures is the difficulty in dissociating physiological changes specific to emotional responding versus third variables (e.g., changes in attention; Berntson & Cacioppo, 2000). Thus, we examine effect sizes involving both subjective and physiological assessments of negative affect, as well as the level of coherence across different emotional systems (Mauss et al., 2005; Patrick, Cuthbert, & Lang, 1994), as a function of pain exposure.

Theories of Pain's Effect on Emotion

Although there is an increase in negative affect in anticipation of pain (Grillon et al., 2006; Moberg & Curtin, 2009), here are theoretical reasons why, under some circumstances, pain might lead to a decrease in negative affect. For instance, pain that is intense, novel, or unpredictable can be attention grabbing and distracting (Eccelston & Crombez, 1999). In some situations, this may not leave cognitive resources to process other internal or external stimuli that influence negative affect (e.g., ruminating about poor work performance). Additionally, research shows a high overlap in the neural pathways and neurochemicals that implement emotion regulation and pain regulation (Eisenberger, 2011; Ochsner & Gross, 2005; Roth-Deri et al., 2008); suggesting that reducing the experience of pain may also reduce the experience of affect. Despite these compelling literatures, there has been little quantitative work that can clarify the size of these effects, or under what conditions pain might lead to a decrease in negative affect. Thus, our main research question for the meta-analysis asked whether the direction of change in negative affect (before versus after experiencing pain) would involve increases, decreases or stable levels (Question 1)? We also examined whether this effect differed depending on the modality of affect (i.e., subjective

versus physiological; Question 1a) or of pain (e.g., temperature, pressure; Question 1b), given the various ways these have been operationalized in the literature.

Other research questions were more exploratory, given the smaller literature involved, and focused on the implications of the findings for clinical phenomena. For example, the initial emotional state of the individual will likely affect the direction of change in negative affect following pain, which can be surmised from the literature on NSSI. Specifically, many of the theories on NSSI suggest that people who engage in NSSI experience persistent, intense negative affect and lack effective emotion regulation strategies (Chapman, Gratz, & Brown, 2006; Linehan, 1993; Selby & Joiner, 2009). Therefore, at least for some people, NSSI can serve to temporarily reduce negative affect in the presence of such overwhelming negative emotions. From this perspective, it might be surmised that pain would lead to a reduction in negative affect when negative affect is elevated prior experiencing pain (Questions 2). However, a review of the literature suggests that such reductions in negative affect following pain are observed in experimental studies where they administer a pre-pain negative affect induction as well as in studies without an affect induction. This suggests that pain-related reductions in negative affect may not require higher pre-pain levels of negative affect (e.g., Bresin et al 2010; Bresin & Gordon, 2013a). Given the mixed literature, a meta-analysis can help determine the generalizability of these findings, and the size of effects, as well as inform the literature on NSSI and the empirical grounding of the emotion regulation theory of NSSI.

Another prediction that may be drawn from the NSSI literature is that, for at least some people, the pain must be intense enough for the negative affect reduction following pain (Question 3). For example, Selby and Joiner's (2009) emotional cascade model suggests that the reason people who engage in NSSI turn to such an extreme behavior is that other forms of stimulation are not intense enough to reduce their negative affect. One study found support for this prediction in individuals with a history of NSSI, in that painful stimulation led to a large reduction in negative affect compared to a nonpainful stimulation, although healthy individuals showed similar sized reductions for either stimulation type (Bresin & Gordon, 2013a). Additionally, the level of stimulation may need to be especially intense to produce a reduction of negative affect under initial negative affect induction. Thus, the effect of painful versus nonpainful stimulation may be strongest when pre-pain negative affect is high versus low (Question 3a).

A final question that arises from the NSSI literature is: do individuals who engage in self-injury respond to pain in the laboratory in the same way that individuals who do not engage in self-injury (Question 4)? Studies using both normative and NSSI samples have found a reduction in negative affective responding following pain (e.g., Bresin et al., 2010; Russ et al., 1992). Moreover, studies including both sample types have found similar reductions for individuals with and without a history of NSSI (e.g., Bresin & Gordon, 2013a; Franklin et al., 2010). This may suggest that pain-related negative affective reductions may not be unique to those who engage in NSSI. Instead, it may be a normative response (perhaps due to overlapping pain-affect regulatory systems) that persons with NSSI are more likely to rely on to reduce their chronic experiences of negative affect. Still, the question of whether or not

people who engage in NSSI respond to pain different than those who do not has yet to be addressed quantitatively.

Current Study

Given that an understanding of changes in negative affect following pain may have implications for many areas of psychology, the primary goal of this study was to conduct a meta-analysis to determine the size and direction of the effect of pain on subsequent negative affect (Pre to Post Pain). Based on the review of the literature above, we predicted that this effect would be negative, different from zero, and small to medium in size. A secondary goal of the study was to test a number of predictions based on models of NSSI to better understand under what conditions pain may lead to changes in negative affect. Based on NSSI theories (e.g., Selby & Joiner, 2009), we predicted that the effect would be larger following a negative affect induction and would be larger following painful versus nonpainful stimulation. Finally, we predicted that the individuals with a history of NSSI would have similar reductions in negative affect following pain as those without a NSSI history, based on results of an admittedly limited literature (e.g., Bresin & Gordon, 2013)

Method

Literature Search

We conducted a literature search using PsychINFO in the spring of 2015. The search terms used were combinations of pain-related keywords (i.e., pain, cold pressor, shock, heat, pressure) with emotion/self-injury related keywords (i.e., negative affect, emotion, psychophysiology, self-harm, self-injury). Searches were conducted to see whether these words were anywhere in the text of articles. Initially, we looked at the title and abstract for preliminary inclusion criteria (i.e., measurement of emotion before and after physical pain). This search identified 47 papers. Articles were reviewed more closely at a secondary stage using the following inclusion criteria: participants are administered physical pain (e.g., cold pressor, heat, pressure, shock) and at least one measurement of negative affect occurs at *both pre- and post-pain periods*. We operationalized negative affect in number of ways based on previous literature (Gruber & Keltner, 2007; Mauss & Robinson, 2009). Specifically, we considered self-report, eye blink startle magnitude, heart rate, blood pressure, skin conductance, electrophysiological indices (e.g., late positive potential), and fMRI activity of the amygdala as measures of negative affect. In addition to published research articles, we included unpublished dissertations in an attempt to reduce possible publication bias (cf., Ferguson & Brannick, 2012). Exclusion criteria were: participants with developmental disabilities, non-human subjects, as well as studies that focused on anticipation of pain or solely measured affect during the experience of pain. After the secondary stage, there were 27 studies remaining (22 published, 3 dissertations, and 2 unpublished).

Upon further review, data necessary to conduct the meta-analysis were missing for 15 of the 27 studies. Researchers were contacted via email. After contacting researchers, we received the necessary data for 5 of those studies. For the others, the researchers no longer had access to the data ($k = 3$; all published papers), or we received no response from the researchers (k

= 7; 2 dissertations; 5 published papers). In total, there were 22 effect sizes from 17 different papers (14 published, 1 dissertation, 2 unpublished) used in our analyses.

Study Coding and Data Extraction

We extracted sample size, *M* and *SD* of the affect measure before and after the experience of pain, and the correlation between pre and post affect measures, in order to calculate effect sizes. If these data were not provided in the paper, we recorded *t*-values and degrees of freedom instead to calculate effect sizes. For moderator analyses, we coded type of affect measurement (with self-report consisting of self-reported negative affect, valence, and/or arousal, and psychophysiology consisting of startle magnitude, blood pressure [diastolic and systolic], and heat rate), pain modality (e.g. heat, cold, pressure, or shock), and NSSI history of the sample. To characterize the studies, we also coded year of publication, percent women, and mean age of the sample.

Effect size calculation

Following the recommendations of van Aert, Wicherts, and van Assesn (2016), we used two different methods to calculate the meta-analyzed effect sizes: random effects and *p*-uniform. We used these two different methods to capitalize on the advantages of each, allowing us to draw firmer conclusions than using either method alone. Random effects modeling follows the traditional practice of averaging all effect sizes weighted by the inverse of the variance, taking into account both within-study and between-study sampling error. We choose a random effects model over fixed effects for two reasons. First, the studies included varied on assessment of affect, pain modality, and key sample characteristics, indicating that there was a high likelihood that we would be examining a variety of effects not one homogenous effect. Ignoring this heterogeneity by using a fixed effects model could lead to inaccurate results (Koetse, Florax, & Groot, 2010). Second, in the case of zero heterogeneity of the effect, a random effects model gives the same estimate as a fixed effect model (Borenstein, Hedges, Higgins & Rothstien, 2007). The advantage of a random effects model is that it allows for the modeling of between-study variation in the effect size. Its disadvantage is that it is highly susceptible to publication bias (van Assesn, van Aert, & Wicherts, 2015).

In contrast, *p*-uniform is a new method of calculating meta-analyzed effects that is based on the distributions of *p*-values for significant effects. This method uses the conditional *p*-value distribution to estimate effect sizes and confidence intervals and tests for publication bias. A major advantage of *p*-uniform is that, because it relies only on significant effects, it is very robust to publication bias, at least under certain conditions (van Aert et al., 2015; van Assesn et al., 2015). Specifically, van Assen et al., (2015) found that when heterogeneity of the effects was low, *p*-uniform outperformed other methods of dealing with publication bias (e.g., trim-and-fill); however, *p*-uniform performs less well under conditions of high heterogeneity (I^2 >.9). Thus, using these two methods allowed us simultaneously to address issues related to heterogeneity of the effect (e.g. random effects model) and publication bias (*p*-uniform).

For the random effect models, we used d_{av} as our measure of effect size (Cumming, 2012; Lakens, 2014). With repeated measures designs, there are a number of options for

standardizing the mean difference (pre versus post-pain negative affect). d_{av} standardizes on the average of the two standard deviations (i.e., pre, post). The primary advantage of d_{av}, over other effect sizes for within-subject designs is that d_{av}, is robust to the correlation among repeated measures, unlike d_z, which is overly liberal when correlations are large among repeated measures, and d_{rm}, which can be overly conservative when the pre-post correlation is large (Cumming, 2012; Lakens, 2014). Moreover, correlations between repeated measures are rarely reported in publications.

In order to calculate the variance of d_{av}, we used the formula provided by Dunlap, Cortina, Vaslow, & Burke (1996), which takes into account the correlation between measures. Given that few studies reported the correlation between repeated measures, we calculated variance estimates for three different correlations (r = .3, .5, .8). However, because the differences in the results with the three different variance estimates were small (differences were in the 3rd and 4th decimal place), we reported the results with the estimated correlation as .8, which is most similar to the test-retest reliability of the measures of emotion included in the study (e.g., Lang, Greenwald, Bradley, & Hamm, 1993; Walters et al., 1987; Watson & Clark, 1994).

For *p*-uniform, we recorded the *t*-test value and *n* for each study. These numbers were used to calculate the meta-analyzed effect size and confidence intervals. Because *p*-uniform outputs d_z, which can overestimate effect sizes, we transformed the results into d_{rm} by multiplying d_z the square root of 2*1-*r*, where *r* is the correlation between repeated measures (cf. Lakens, 2014). As above, when correlations were not available, .8 was used. Because d_{av} has no direct relation to the test statistic, we were not able to transform d_z to d_{av}. Therefore, d_{rm} seemed like a better choice than d_z because any bias that would exist would be underestimating, making it less likely to find a significant result.

Effect sizes were calculated such that negative values indicated a decrease in negative affect following pain (i.e., post - pre). When multiple effect sizes existed for one study, we used an average effect size in the analysis. The exception to this was for Question 1, when we compared self-report to psychophysiology, where effects were only pooled within one measurement modality of negative affect for each study.

Data analytic plan

To examine Question 1, we calculated the overall effect size across all 22 effects from the 17 papers. For Question 1a, we conducted a moderator analysis in which we compared the effect size from self-report measures (k = 14) relative to physiological measures (k = 8) of negative affect (3 studies had both). For Question 1b, we compared the effect size for different modalities of pain (i.e., cold pressor, pressure, shock, heat). This was done by examining whether there was a main effect of pain modality and using follow-up tests to compare the effects for each pain modality. For Question 2, we compared the effect size for the effect of pain following a negative affect induction (k = 7) to the effect size following a neutral affect induction (k = 6) in a subset of studies with those conditions (6 having both, and one only a negative affect induction).

To determine whether painful versus nonpainful stimulation is necessary, Question 3 compared the effect of the change in negative affect following painful stimulation obtained from Question 1 ($k = 22$) to that of nonpainful stimulation (e.g., water at room temperature) in a subset of eight studies (from the 22) that included the latter condition. Question 3a compared the effect of painful stimulation following a negative affect induction ($k = 8$) to the effect of nonpainful stimulation following a negative affect induction ($k = 8$). For our final research question (Question 4), we compared the effect of pain on subsequent negative affect for individuals with a history of NSSI ($k = 7$) to those without a history of NSSI ($k = 6$). Seven studies included both groups, and one study only had individuals with a history of NSSI. In all cases we followed a similar strategy for *p*-uniform; however, because *p*-uniform only uses significant effects, the number of studies included differed from the random effects model. Table 1 displays the number of studies for each analysis.

The random effects models analyses were conducted using the metafor package in R (R. Core Team, 2015; Viechtbauer, 2010), whereas *p*-uniform results were obtained using the online calculator (van Alert et al., 2015). Model estimates were used to calculate both a point-estimate for the effect size and 95% confidence interval for the effect. Effect sizes were interpreted with Cohen's (1992) guidelines of $d = .2$, $d = .5$, and $d = .8$, corresponding to small, medium, and large effect sizes, respectively. For all effect sizes, the confidence intervals were used to infer whether the effect was significantly different from zero (i.e., 0 is not in the interval). For questions comparing two effect sizes (Questions 2–5), the intervals were also used to determine whether there was strong evidence for a population difference (i.e., when the two intervals do not overlap), some evidence of the population difference (i.e., when the two intervals overlap by less than ½ the margin of error), or no evidence of a population difference (i.e., substantial overlap in the intervals; Cumming, 2012). Heterogeneity in the effect size across studies was tested with the *Q*-statistic, which tests the null of no heterogeneity of effect sizes across studies. In addition, we report I^2 which is the total heterogeneity divided by the total variability. Higgins and Thompson (2002) recommend interpreting I^2 values of 25%, 50%, and 75% as small, medium, and large amounts of heterogeneity respectively.

Publication bias is a concern in meta-analysis. Unfortunately, most tests for publication bias perform poorly under conditions of high heterogeneity (Francis, 2013; Peters, Sutton, Jones, Abrams, & Rushton, 2007; Van Assen et al 2015). A recent simulation study by McShane, Bockenhold, and Hasen (2016), however, suggested that selection methods, as proposed by Vevea and Woods (2005; also see Vevea & Hedges, 1995), out preformed traditional methods. Selection models model the data (i.e., effect sizes) and a selection method (i.e., how effects are "selected" for publication). This allows for the estimation of effect sizes under different selection situations. We used pre-defined weights that signify moderate (nonsignificant results are less likely to be published) and severe bias (nonsignificant results are almost never published) selection models (see Vevea & Woods, 2000 for weights).

A limitation of this approach is that because predefined weights are used, standard errors and confidence intervals are not estimable. Therefore, these analyses are used as a sensitivity analysis, to see whether there are changes in the estimated effect size under different selection conditions. To interpret the results of these models, the researcher looks for large

changes in the meta-analyzed effect across selection models. Instability, particularly attenuation of the effect, for different selection parameters is a sign of publication bias. We made two comparisons to determine whether a reduction was large. First, we looked at the percentage change for the adjusted effect in relation to the original effect (cf. Vevea & Woods, 2000). This was a subjective test because there are not established guidelines for how much change is important. Second, we compared the adjusted effect sizes to the 95% CI for the unadjusted effect. We fit selection models using the weightfunct in R (Coburn & Vevea, 2016).

In addition to selection models, we used the test for publication bias associated with the *p*-uniform procedure. This tests whether the *p*-uniform estimate of effect size significantly differs from the estimate derived from traditional meta-analysis, and a significant result indicates publication bias. Simulation results show that this method outperforms other methods (e.g., trim-and-fill; van Assen et al., 2015). As with the other methods, there was no evidence of publication bias. Finally, it should be noted that we included several nonsignificant results in each of our tests, which serves as further evidence against publication bias.

Results

Study Characteristics

The average sample size across the 22 studies was 78.04 (SD = 62.83), for a total sample size of 1,699. Overall, many studies failed to report basic demographics. Of those that did, they included slightly more women than men (M = 61.01%, SD = 24.36, k = 13 studies reported gender) and had a mean age of 21.21 (SD = 1.95, k = 10 studies reported age), which is not surprising given that the majority of the studies used undergraduate samples. Pressure was the most common method of inducing pain (k = 7), followed by cold temperatures (e.g., the cold pressor; k = 6). Five studies used electric shock and four used hot temperatures. Fourteen studies included self-report measures, whereas only eight included a psychophysiological measure (k = 6 for startle magnitude, k = 2 for blood pressure, k = 3 for heart rate; 3 studies included more than one physiological measure).

Main Analyses (Question 1)

The results for Question 1 (what is the size and direction of the effect of pain on negative affect?) showed that across the 22 effects included, the random effects model yielded an effect size that was small to medium and the confidence interval did not include 0 (d_{av} = −.35, 95% CI [−.58, −.12]), and the *p*-uniform yielded a similar effect size based on the 8 significant studies (d_{rm} = −.44, 95% CI [−.58, −.34]). These results are consistent with our prediction. There was significant heterogeneity across the studies that was large in size, $Q(21) = 500.88$, $p < .001$, $I^2 = 97.80\%$. Given the significant heterogeneity, we conducted follow-up moderator analyses (Questions 1a and 1b). First, the results showed that the point estimate for self-report (random effects: d_{av} = −.44, k = 12; *p*-uniform: d_{rm} = −.45, k = 7) was larger than that for psychophysiology (random effects: d_{av} = −.21, k = 8; *p*-uniform: d_{rm} = −.28, k = 1); however, the confidence intervals overlapped by more than ½ a margin of error (self-report-random effects: 95% CI [−.73, −.15], self-report-*p*-uniform 95% CI [−.65,

−.35], psychophysiology-random effect: 95% CI [−.59, .17], psychophysiology-*p*-uniform: 95% CI [−.67, .48]), providing little to no evidence for a population difference. This was also reflected in the fact that the negative affect measure used explained 0.14% of the heterogeneity in the effect size. Second, we found that pain modality (e.g., temperature, pressure) was not significantly related to effect size, $Q(3) = 6.04$, $p = .109$, explaining only 0.5% of the heterogeneity among effect sizes, and pressure as the pain modality showed a very small effect that was significantly different from zero. Both of these null results should be interpreted with caution given the possibility of a type-II error. Table 1 displays the results.

Secondary Analyses (Questions 2–4)

For the secondary analyses, we first compared the effect size of change in negative affect following pain in experiments that induced negative affect prior to pain, compared to experiments that induced a neutral mood, in a subset of studies that included one (or more) of these conditions (Question 2). Thus, this tested the effect of mood induction holding the experience of pain constant. Similar to the overall effect, experiencing pain following a negative affect induction had a significant small to medium effect (random effects: $d_{av} = -.37$, 95% CI [−.73, −.02] $k = 7$; *p*-uniform: $d_{rm} = -.61$, 95% CI [−1.25, −.41, $k = 4$]), with significant heterogeneity, $Q(6) = 37.67$, $p < .001$, $I^2 = 94.82\%$. Conversely, the effect of pain following a neutral mood induction was not significantly different from zero ($d_{av} = .08$, 95% CI [−.09, .26], $k = 6$, a *p*-uniform estimator could not be estimated because there were no significant results), but still had significant heterogeneity, $Q(5) = 17.30$, $p < .004$, $I^2 = 79.55\%$. Given that the confidence interval for the effect size for the experience of pain following a negative affect induction and the experience of pain following a neutral affect induction overlap less than ½ of a margin of error for either interval (overlap = .07, average ½ margin of error = .13), there is evidence for a population difference between negative and neutral affect inductions. Thus, the affect regulation effects of pain are more robust when individuals are experiencing higher pre-pain levels of negative affect, as indicated by NSSI theories (e.g., Selby & Joiner, 2009).

For Question 3 (does painful stimulation lead to a larger change than nonpainful stimulation?), we compared the effect from Question 1 to the meta-analyzed effect of change in negative affect responding following nonpainful stimulation, using a subset of studies that had a condition with nonpainful stimulation (e.g., water at room temperature). The effect of nonpainful stimulation was medium to large and significantly different from zero (random effects: $d_{av} = -.59$, 95% CI [−1.05, −.12], k = 8; *p*-uniform, $d_{rm} = -.50$, 95% CI [−.80, −.30], $k = 5$). This effect also had a large amount of heterogeneity, $Q(7) = 158.68$, $p < .001$, $I^2 = 97.43\%$. Of critical importance, the confidence interval had substantial overlap with that from Question 1 (random effects: [−.58, −.12]; *p*-uniform: [−.58, −.34]). Given that the confidence intervals overlap almost completely, there is little evidence for a population difference. Thus, these results suggest that any stimulation (painful or nonpainful) is sufficient for the reduction in negative affect responding.

To test Question 3a (does painful versus nonpainful stimulation lead to a larger effect when pre-pain negative affect is high?), we compared the reduction in negative affect following a

negative affect induction for participants who experienced pain versus those who experienced nonpainful stimulation. The results showed that following both painful (random effects: d_{av} = −.88, 95% CI [−1.60, −.17], k = 6, p-uniform: d_{rm} = −1.00, 95% CI [−1.44, −.59], k = 3) and nonpainful (d_{av} = −.70, 95% CI [−1.29, −.12], k = 6, p-uniform: d_{rm} = −.46, 95% CI [−.80, −.15], k = 5) stimulation, there was a significant and medium to large reduction in negative affect. Moreover, the intervals overlapped substantially, indicating minimal evidence of a population effect. These results are consistent with above, that any stimulation may be sufficient in reducing negative affect even under pre-pain negative affect induction. Another interpretation is that that pain does not have an effect over and above the normative return to baseline following a negative affect induction.

To test our final question (Question 4: is the affective response to pain the same for individuals with and without a history of NSSI?), we calculated the reduction in negative affect for individuals with a history of NSSI and compared it to those without a history of NSSI in a subset of studies that included at least one group of individuals with a history of NSSI. The effect size was medium for individuals with a history of NSSI (d_{av} = −.48, 95% CI [−1.07, .11], k = 7, p-uniform: d_{rm} = −.80, 95% CI [−1.50, −.32], k = 3) and those without a NSSI history (d_{av} = −.45, 95% CI [−1.13, .23], k = 6, p-uniform: d_{rm} = −.72, 95% CI [−1.53, .13], k = 2). Interestingly, although both intervals include zero for the random effects estimation, the p-uniform estimation indicated that the effect is significantly different from zero for people with a history of NSSI and not for people with no history of NSSI. In either case, the confidence intervals overlap substantially, suggesting that the effect of pain is similar for individuals with and without a history of NSSI.

Publication Bias

Overall there was limited evidence of publication bias. For all effects, the estimates for both moderate and severe publication bias still fit within the initial confidence intervals (see Table 2). For moderate bias, the average percentage change from the unadjusted effect was 12.04% (*min* = 0, *max* = 35.41%). For severe bias, the average percentage change was 20.79% (*min* = 2.27, *max* = 83.33%). In most cases, the effect adjusted for publication bias was larger than the unadjusted, which indicates that the data used in our study includes more nonsignificant results than would be expected under moderate and severe publication bias, providing further evidence against publication bias being a concern. The two instances where the effect was reduced were for psychophysiological measures of emotion and for nonpainful stimulation in question 2b; however, these changes were modest. In addition, all p-uniform tests for publication bias were not significant, suggesting no publication bias.

Discussion

The overall goal of this meta-analysis was to determine the direction and size of the effect that acute physical pain has on changes in negative affect. We tested several different hypotheses to examine this, with mixed support. Our main findings were that pain had a small to medium effect in reducing negative affect (Question 1), and most robustly regulated negative affect in the context of a negative affect induction relative to neutral affect induction, with a medium to large effect (Question 2). The fact that similar reductions were

seen following painful and nonpainful stimulation, however, complicates the interpretation of these findings. Beyond these main results, most moderator analyses failed to support differences in effect size as a function of the type of negative affect assessment (self-report versus psychophysiology), pain modality (e.g., heat, shocks), or the intensity of the stimulation (painful versus non-painful) used in studies (Questions 1a, 1b, and 3). Finally, we found that those with and without a history of NSSI both experienced a reduction in negative affect (Question 4).

Intuition would suggest that there would be an increase in negative affect following pain (Grillon et al., 2006; Rainville et al., 2005). Conversely, other research suggests that negative affect would reduce following the experience of pain (Bresin et al., 2010). Our results for Question 1 are clearly consistent with the latter rather than the former, suggesting that the negative affect following the experience of pain can be distinguished from the anticipation of the experience of pain or subjective appraisal of the pain itself (which typically involve increases in negative affect). The caveat is that the statement may be better framed as the following: the experience of varying types of stimulation seems to produce subsequent reductions in negative affect.

Although these results seem counterintuitive, they do fit with research showing that pain can be associated with a variety of positive outcomes. For example, Bastian et al. (2014) reviewed evidence that pain promotes affiliation with others through mechanisms such as increasing relational focus and solidarity and arousing empathy in others. Taken together with the results of this meta-analysis, these findings suggest that more theorizing about the role of pain in affective experiences is necessary in order to explain these findings.

Our additional predictions based on the NSSI literature helped to clarify the general effect. Perhaps the two most important findings were that the effect of pain on negative affect is larger for people who are experiencing higher pre-pain levels of negative affect and that nonpainful stimulation leads to a similarly-sized reduction as painful stimulation. These results have some implications for the emotional cascade theory of borderline personality and self-injury (Selby & Joiner, 2009). The emotional cascade theory predicts that part of the reason that some people use NSSI as a way to regulate emotion is because they experience chronically higher levels of negative affect. The larger effect following negative affect inductions compared to neutral inductions is consistent with this prediction. The emotional cascade theory also posits that people use NSSI, as opposed to other emotion regulation strategies, because they need intense stimulation to regulate their intense emotions. Our meta-analytic findings suggesting similar reductions following painful and nonpainful stimulation are inconsistent with this prediction.

The combination of these two results could be interpreted to mean that the reduction in negative affect following the experience of pain is not a true effect; it is actually an artifact that represents a return to affective baseline over time. This would explain why there is a reduction regardless of painful versus nonpainful stimulation (e.g., the return to baseline would have happened regardless) and why the effect is larger following a negative affect induction (i.e., at higher levels of emotional responding, there is more room to reduce). However, given that no single study manipulated both affect induction (negative versus

neutral) and stimulation type (painful versus nonpainful), it is difficult to draw firm conclusions. In addition to adding these conditions in future research, time between the mood induction and pain could be manipulated to further rule in or out the return to baseline hypothesis.

It is worth noting that the emotional cascade theory was designed to explain NSSI in the context of borderline personality disorder. Therefore, it is possible that in samples with clinical levels of borderline traits there may be a difference between painful and nonpainful stimulation. Along these lines, Bresin and Gordon (2013) found that for people without a history of NSSI, painful and nonpainful stimulation lead to similar reductions in in negative affect; however, for people with a history of NSSI, painful stimulation led to a significantly larger reduction than nonpainful stimulation. This suggests that more studies using specialized samples are needed to further understand the cascade theory and whether or not the reduction in negative affect following pain is a unique effect.

An interesting finding was that those with and without a history of NSSI showed similar changes in negative affect following pain. This may suggest that the reduction in negative affect is a normative response across persons, but that people who engage in NSSI leverage this response in a maladaptive way. If this is the case, future NSSI research should seek to identify factors that lead some people to use pain as a way to regulate their emotions. Theories of NSSI have proposed that individual differences in affective responding such as negative emotionality, emotional instability, and low distress tolerance may play a role (e.g., Chapman et al., 2006). Specifically, it is proposed that people who experience persistent, intense negative emotions that they find intolerable and who have a tendency to engage in impulsive behaviors when upset (i.e., negative urgency) will be more likely to engage in dysregulated behaviors including, but not limited to, NSSI to regulate their emotions (Chapman et al., 2006; Cyders & Smith, 2008; Selby & Joner, 2009). In addition to these emotional factors, it is possible that non-emotional factors, such as higher pain tolerance, may lead people to use pain as opposed to other impulsive behaviors (Nock, 2009). Regardless of the pathways to the behavior, our results suggest that NSSI can lead to temporary reductions in negative affect, which may partially explain why many people continue to engage in the behavior when upset.

Beyond NSSI, general psychology research should seek to understand the mechanisms that may be underlying the reduction in negative affect following the experience of pain. Although our results do not identify any specific mechanisms, we mention some possibilities to be explored by future research. As mentioned in the introduction, it is possible that the reduction in negative affect following the experience of pain may be mediated by the demands that pain (or nonpainful stimulation) put on attention and cognition (e.g., Eccelston & Crombez, 1999; Selby & Joiner, 2009). Additionally, this effect may be partially explained by the fact that similar biological systems regulate pain and affect (Bresin & Gordon, 2013b; DeWall et al., 2010). Taking this idea one step further, the termination of pain can be experienced as rewarding (i.e., relief, negative reinforcement). Supporting this prediction, research has found that brain regions sensitive to reward are activated following the end of painful stimulation (e.g., Leknes et al., 2011). Another, non-mutually exclusive possibility is that in the context of pain, a neutral nonpainful state is seen as pleasant or a

least, less bad than it was. Some research has shown that people's emotional responses to pain are influenced by the context in which the pain is experienced (Leknes et al., 2013). Given that our results do not rule in or out any of these possible mechanisms, future research in this area is needed.

All of our results need to be interpreted while also considering some key limitations. First, although we had predicted substantial heterogeneity of effect size in our studies, we did not anticipate such high heterogeneity. High heterogeneity leads to overestimation of effect size and this is true for both random effects and *p*-uniform (van Assesn et al, 2015). The best way to deal with heterogeneity is to create homogeneous subsets of studies; however, our attempts to do this did not reduce heterogeneity. Currently, there are no methods for correcting the bias introduced by heterogeneity. This does not necessarily mean that the effects reported are zero, but they are likely smaller than reported. For instance, in a simulation study, van Assen et al., (2015) found that when the true effect size was .33, random effects and *p*-uniform estimated the effect at .53. Future research should seek to identify sources of heterogeneity across studies and/or introduce standardized methods across studies in order to create homogeneous sets of studies.

Further, caution is warranted in interpreting our nonsignificant results because of the possibility of type-II errors. More studies are needed to understand the role of different pain modalities and different methods of assessing negative affect to develop clear conclusions, as the number of studies were small in the analyses of many of the secondary questions. Finally, we limited our focus to acute laboratory pain, which is very different from chronic pain and acute pain experienced in the real world. Other lines of research are needed to understand how emotional responding to chronic versus acute pain may differ and whether responses to laboratory pain generalize to real world situations.

In addition to these limitations, there are several strengths worth noting. First, we took numerous steps to reduce the effect of publication bias. We included unpublished studies, and used cutting-edge methods for effect size estimation that are robust to publication bias under some conditions. (van Assen et al., 2015). Another strength is that we were able use multiple operationalizations of negative affect and different modalities of pain, enhancing the likelihood of our finding generalizing to future research. Nonetheless, far from providing definitive results, this meta-analysis opens up more questions than answers. As psychology moves toward open practices (e.g., publishing null results), it is likely that more meta-analyses of this type will need to be published.

Although our results did not cleanly map on to our predictions, they help advance theory and research in a number of domains. Neuropsychologists may use our findings to further elucidate the overlap between neural systems that implement pain and emotion processing and regulation. Personality psychologists may want to identify individual differences that predict greater decreases in negative affective response following pain or nonpainful stimulation (e.g., risk taking). Our results may help support psychoeducational efforts to promote exercise (e.g., even if the anticipation of pain is unpleasant, afterwards there is a reduction of negative affect). Finally, our findings have clear implications for NSSI and other clinical behaviors that involve pain to some extent (e.g., binge eating, purging). This

meta-analysis also leaves an important unanswered question to be addressed by future research: by what mechanism(s) does pain reduce negative affect and why do some people use it to regulate their emotions and not others?

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Table 1

Meta-analyzed Results for Main Research Questions

	Random Effects			*p*-uniform		
	d_{av}	95% CI	*k*	d_{rm}	95% CI	*k*
Question 1						
Overall	−.35	[−.58, −.12]	22	−.44	[−58, −.34]	8
Question 1a						
Self-report	−.44	[−.73, −.15]	12	−.45	[−.65, −.35]	7
Psychophysiology	−.21	[−.59, .17]	8	−.28	[−.68, .48]	1
Question 1b						
Shock	−.30	[−.75, .14]	5	−.54	[−.74, −.33]	1
Heat	−.84	[−1.35, −.33]	4	−.75	[−1.56, −.40]	2
Cold	−.44	[−.86, −.02]	6	−.41	[−.86, −.09]	3
Pressure	−.06	[−.43, .31]	7	−.31	[−.44, .04]	2
Question 2						
Negative	−.37	[−.73, −.02]	8	−.61	[−1.25, −.41]	4
Neutral	.08	[−.09, .26]	7			
Question 3						
Nonpainful	−.59	[−1.05, −.12]	9	−.50	[−.80, −.30]	5
Question 3a						
Painful	−.88	[−1.60, −.17]	7	−1.00	[−1.44, −.56]	3
Nonpainful	−.70	[−1.29, −.12]	7	−.46	[−.80, −.15]	5
Question 4						
NSSI	−.48	[−1.07, .11]	8	−.80	[−1.50, −.32]	3
No NSSI	−.45	[−1.13, .23]	7	−.72	[−1.53, .13]	2

Note. *k* = number of studies

Table 2

Results from Publication Bias Analyses

	Moderate Selection Bias *d* (% difference from unadjusted)	**Severe Selection Bias *d* (% difference from unadjusted)**	***p*-uniform test**
Question 1			
Overall (*k* = 22)	−.38 (8.57)	−.42 (20.00)	L_{pb} = −3.37, *p* = .996
Question 1a			
Self-report (*k* =14)	−.48 (9.09)	−.52 (18.18)	L_{pb} = −3.46, *p* = .999
Psychophysiology (*k*=8)	−.16 (23.08)	−.16 (23.08)	L_{pb} = −.91, *p* = .817
Question 1b			
Shock (*k* =5)	−.31 (3.33)	−.32 (6.66)	L_{pb} = −1.72, *p* = .958
Heat (*k* = 4)	−.89 (5.95)	−.92 (10.71)	L_{pb} = −.08, *p* = .533
Cold (*k* = 6)	−.44 (0.00)	−.45(2.27)	L_{pb} = −1.93, *p* = .973
Pressure (*k* = 7)	−.08 (33.33)	−.11(83.33)	L_{pb} = −1.68, *p* = .953
Question 1c			
Negative (*k* = 8)	−.36 (2.07)	−.39 (5.45)	L_{pb} = −3.12, *p* = .999
Neutral (*k* = 7)	.07 (12.50)	.05 (35.00)	_____
Question 2			
Nonpainful (*k* = 9)	−.61 (3.38)	−.64 (8.47)	L_{pb} = −2.89, *p* = .998
Question 2b			
Painful (*k* = 7)	−.91 (3.40)	−.94 (6.81)	L_{pb} = −2.99, *p* = .998
Nonpainful (*k* = 7)	−.50 (28.57)	−.57 (18.57)	L_{pb} = −.548, *p* = .708
Question 3			
NSSI (*k* = 8)	−.65 (35.41)	−.69 (43.75)	L_{pb} = −2.53, *p* = .994
No NSSI (*k* = 7)	−.45 (00.00)	−.49 (8.88)	L_{pb} = −.01, *p* = .506

Note.