HHS Public Access

Author manuscript

Pain. Author manuscript; available in PMC 2021 June 01.

Published in final edited form as:

Pain. 2020 June ; 161(6): 1140–1149. doi:10.1097/j.pain.0000000000001828.

Positive affect and chronic pain: a preregistered systematic review and meta-analysis

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Abstract

Chronic non-cancer pain (CNCP) is a significant health burden among adults. Standard behavioral therapies typically focus on targeting negative affect (NA) and yield only modest treatment effects. The aims of this study were to systematically review and investigate the association between positive affect (PA) and pain severity among adults with CNCP. Databases search included MEDLINE (PubMed), PsycINFO, CINAHL, ProQuest Dissertations and Theses, OLASTER, Open Grey, and PsyArXiv (inception to July 23, 2019). We analyzed studies that: (1) employed observational, experimental, or intervention study designs; (2) enrolled individuals with CNCP (pain ≥ 12 weeks); and (3) reported full quantitative results on outcomes. Two researchers independently screened articles, extracted data, and assessed the risk of bias. The main meta-analysis was followed by subgroup analyses. All analyses were performed using random-effects models. Formal tests for heterogeneity (Q-statistic; I^2) and publication bias (p-curve and p-uniform*) were performed. We meta-analyzed 29 studies with 3521 participants. Results demonstrated that PA inversely impacts pain severity in people with CNCP ($r = -0.23$). Subgroup analyses showed a significant effect for gender and marginally significant effects for age in studies that adjusted for NA. On average, effect sizes for observational studies were larger in studies with a higher proportion of female respondents and in studies that did not adjust for NA. Finally, larger effect sizes were found in intervention studies with older compared with younger samples.

Keywords

Positive affect; chronic pain; pain severity; systematic review; meta-analysis

1. Introduction

It is estimated that 70 million Americans—more than the number affected by diabetes, heart disease, and cancer combined—suffer from chronic non-cancer pain (hereafter referred to as

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Conflicts of Interest

The authors have nothing disclose.

CNCP) each year [11]. With an increased prevalence among persons ages 65 and over [84], chronic pain is a significant health burden—not just in terms of pain-related health care expenditures and disability, but also in terms of the inestimable costs to families and individual daily living and quality of life. Pain severity, a core clinical measure of chronic musculoskeletal pain [50], is associated with greater disability, sleep impairment, psychosocial difficulties [35; 83; 94] and increased prevalence of mental health disorders including depression, anxiety, and substance abuse among patients with CNCP [2; 74; 83]. Although there is increasing interest in the use of evidence-based non-pharmacological approaches to managing chronic pain severity [12; 62], standard behavioral therapies, such as cognitive-behavioral therapy (CBT) and mindfulness-based stress reduction (MBSR), typically focus on targeting negative affective states (e.g., anxiety and depression) [48; 49] and yield only modest treatment effects [25; 31]. Efforts are therefore needed to develop more effective psychological treatments for chronic pain by identifying new targets for intervention.

A growing body of theory and research suggest that positive affective states (e.g., gratitude and happiness) play a uniquely important role in promoting psychological adjustment in the face of chronic pain [40; 60; 64; 65]. Specifically, positive affect (PA) has been theorized to facilitate adaptive coping in the context of chronic pain by countering the negative effects of fear on attention [90]; buffering negative pain-related cognitions (i.e., rumination, helplessness, magnification) [63]; reducing inflammation [82]; promoting neutral reappraisal processes related to pain [46]; and enhancing engagement in valued activities in the face of pain [91].

Although narrative reviews have been conducted [28; 38; 57], to date, there has not been a comprehensive quantitative review relating PA to chronic pain severity. Howell et al. [45] meta-analyzed experimental studies and found that induced PA was associated with higher pain tolerance. More recently, Kushlev et al. [53] examined data from nearly 2.5 million U.S. respondents and found an inverse relationship between PA and previous day physical pain. Notably, both studies focused on acute pain responses. Thus, there is a need to establish whether these findings generalize to chronic pain. The primary aim of this systematic review and meta-analysis was to comprehensively review the literature examining the association of PA and pain severity in people with CNCP. We use systematic methods and standardized procedures [59; 72] for locating and evaluating the relevance and quality of observational, experimental, and intervention studies. Observational studies consisted of both ambulatory and longitudinal studies. Ambulatory studies used experience sampling methodology across several days or weeks to examine how changes in PA relate to pain. Longitudinal studies explored whether levels of PA predict future levels of pain across more extended periods. Experimental studies determined the effects of induced or manipulated PA on concurrent pain. Intervention studies examined the efficacy of PA-enhancing treatments on pain severity prospectively over time. The study's secondary aims were to investigate moderators of the relation between PA and pain severity in people with CNCP, determine the quality of the studies, and examine potential publication bias.

2. Methods

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement [59]. All methods and planned analyses were preregistered on the Open Science Framework (OSF).

2.1 Data sources and searches

MEDLINE (PubMed), PsycINFO, and CINAHL databases were searched electronically from inception to March 21, 2017. An updated search was performed (from April 7, 2017 through September 24, 2018) to include research from gray literature sources (ProQuest Dissertations and Theses, OLASTER, Open Grey, and PsyArXiv) and to identify new publications. All databases and gray literature sources were searched again on July 23, 2019 to capture additional relevant literature published between 2017 and 2019. Search terms for PA used keywords drawn from prior reviews of health outcomes associated with PA [9; 15; 70], and included variations of *happy, cheerful, joy, vigor, excited, elated, enthusiastic, energetic interest, content, amused, humor, calm, relaxed, grateful, satisfied, positive affect, positive emotions, and positive mood*. Search terms for chronic pain included variations of *widespread pain, recurrent pain, persistent pain, and long-term pain*. The details of the full search strategy are presented in eAppendix 1.

2.2 Eligibility criteria and study selection

Studies were eligible for inclusion if they met the following criteria: (1) the study design was observational, experimental, or intervention (e.g., randomized controlled trial); (2) participants were adults (18 years or older) with CNCP (12 weeks or more in duration); and (3) results were reported in sufficient quantitative detail to discern a directional effect of PA on pain severity. For all intervention studies, data from the baseline to first follow-up were included, with the baseline vs. first follow-up contrast serving as the primary outcome. In the case of two or more groups receiving different PA interventions within one study, all were independently included.

Articles were excluded if they: (1) were not an empirical study; (2) did not involve human subjects; (3) did not include a measure of PA or a positive mood manipulation (e.g., humorous films, pleasant images); (4) used a reversed indicator of negative affect (NA) as a measure of PA (e.g., hopelessness vs. hopefulness; pessimism vs. optimism; fatigue vs. vigor/vitality); (5) did not include a subjective or objective measure of pain severity; (6) assessed affect only in regard to a specific life experience (e.g., “How happy are you about being pregnant?”); (7) examined only the directional effect of pain on PA or mean differences in PA between pain impaired and non-impaired samples; (8) did not enroll individuals identified as suffering from chronic pain; (9) assessed acute or experimentally induced pain; (10) did not employ an eligible study design; (11) did not examine or report a directional effect of PA on pain; (12) exclusively used a sample of patients with a primary cancer diagnosis; (13) were published in languages other than English; (14) did not include adults (18 years and above); (15) exclusively used a sample of patients presenting with a primary psychological disorder; (16) did not directly target PA; (17) did not include sufficient quantitative information on study outcomes; (18) full text of article could not be

located; or (19) were a duplicate identified during full-text screening. When possible, we also contacted authors for further information.

After duplicates were removed, titles and abstracts were screened by two independent reviewers (K.R. and K.G.) to determine whether the citation met eligibility criteria. Subsequently, two independent authors (A.D.O. and F.T.) assessed the full text of potentially eligible studies for inclusion. Conflicts were resolved by consensus. Figure 1 presents the study selection process and indicates the number of articles excluded at each phase of screening.

2.3 Data extraction and quality assessment

Two reviewers (A.D.O. and F.T.) independently extracted study characteristics and outcome data from published articles. Risk of bias was independently assessed by two reviewers (A.O. and F.T.) using the Effective Public Health Practice Project (EPHPP) tool [72]. Specifically, the included studies were assessed for (1) selection bias, (2) study design, (3) confounders, (4) blinding, (5) data collection, and (6) withdrawals/dropouts. Each domain was rated as strong, moderate, or weak, and domain scores were averaged to provide a global rating for each study (inter-rater reliability 88%, Cohen's kappa .79). Discrepancies were resolved by consensus.

2.4 Data synthesis and analysis

Meta-analyses were performed using the *metafor* and *meta* packages [76; 88] in R, version 3.4.3 (R Project for Statistical Computing). For each study, individual effect sizes were calculated within each independent sample. For observational studies (ambulatory and longitudinal), standardized regression coefficients were extracted and used as an effect size index [67]. For experimental and intervention studies, effects sizes (Hedges' g) were extracted from descriptive statistics. Similar to Cohen's d, Hedges' g effect sizes of 0.00 to 0.32 can be considered as small, effect sizes of 0.33 to 0.55 as moderate, and effect sizes of 0.56 to 1.20 as large [54]. Although effect sizes were computed separately for each of the three study designs (observational, experimental, and intervention), we also computed an overall effect size ($r_{\text{equivalent}}$) [75] from exact t-values reported across studies. Specifically, we report $r_{\text{equivalent}}$ effect sizes on the Fisher Z-transformed metric, and used a standard error, as suggested by Rosenthal and Rubin [75], defined as the square root of $N-3$. Meta-analyses yielded a point estimate, confidence interval, and p-value, along with statistics for heterogeneity (assessed using the Cochran Q_E-statistic and the Higgins-Thompson I^2 values) [22]. Publication bias was evaluated using the Egger test (with $p < .10$ indicating asymmetry [26]), and visual inspection of funnel plots. For completeness, we conducted p-curves [79] (the distribution of statistically significant p values for a set of findings, with right-skewed p-curves suggesting findings that contain evidentiary value) and p-uniform* test (a publication bias test based on the effect size in a set of studies) [87].

2.5 Subgroup analyses

A priori subgroup analyses were performed to explore moderators of the PA-pain severity relation, including (1) risk of bias quality rating: weak, moderate, and strong; (2) demographics: percentage female, percentage racial minority, and mean age; (3) chronic

pain status: fibromyalgia, rheumatoid arthritis, osteoarthritis, back pain, and multiple; (4) PA measurement: state and trait; and (5) covariate adjustment: unadjusted negative affect (NA) and adjusted NA. For categorical moderators that explained significant variance in the effect sizes (i.e., $p < 0.05$ for Q_M), *post hoc* contrasts were performed to determine which groups were statistically different. For continuous moderators, meta-regression analyses were used to determine whether variation in the effect sizes was explained by the moderator. A false discovery rate (FDR) Type I error control was used for all comparisons to correct for multiple testing [8].

3. Results

3.1 Study characteristics

From a total of 3,063 retrieved articles, 151 were identified based on title and abstract screening for full-text review. Of these, 38 studies fulfilled eligibility criteria and were included in the systematic review. Descriptive details of the studies are presented in Table 1. The included studies were published between 1981 [58] and 2018 [39]; came from ten countries, with 25 studies from the United States; had sample sizes ranging from 8 [58] to 360 [39], and included a total of 4,229 participants (mean [SD] age was 54 [9.57] years and 76% were women). It should be noted that not all studies reported the exact age or number of non-White participants.

Among the 38 included studies, 11 were observational (eight ambulatory [17; 27; 29; 36; 52; 55; 61; 95], three were longitudinal studies [69; 77; 81]); 9 were experimental [1; 19; 44; 47; 58; 68; 73; 92; 93]; and 18 were interventions [3; 5–7; 10; 20; 23; 30; 32; 34; 37; 39; 41; 60; 66; 78; 85; 96]). Among observational studies, the majority used self-report adjective ratings of positive valence (e.g., *active, energetic, happy, cheerful, joyful*) to assess level of PA. State levels (momentary, daily) of PA were typically assessed in ambulatory studies, whereas trait levels (global ratings) of PA were typically measured in longitudinal studies. Among experimental studies, examples of PA-based inductions included viewing emotionally evocative images, humorous film clips, and guided imagery. Finally, in the intervention research reported here, a variety of methods were used to increase PA in people with CNCP, including expressing thanks, practicing acts of kindness, and savoring positive moments, among others [39].

3.2 Risk of bias

The assessment of the quality of the study methodology for the five domains (selection bias, study design, confounders, blinding, and data collection) is reported in eFigure 1 in the Supplement. Risk-of-bias assessments for individual studies included in the qualitative review are summarized in Table 1. Following the EPHPP tool, eight studies [3; 5; 34; 39; 41; 47; 66; 96] (21.05%) were classified as “strong” or having low risk of bias; 13 studies [6; 7; 10; 19; 20; 30; 37; 58; 60; 73; 78; 85; 93] (34.21%) were categorized as having “moderate” risk; and 17 studies [1; 17; 23; 27; 29; 32; 36; 44; 52; 55; 61; 68; 69; 77; 81; 92; 95] (44.74%) were categorized as “weak” or having high risk of bias. Weakness ratings derived from the inadequate control of confounders and insufficient information regarding study design, as well as lack of blinding.

3.3 Meta-analyses

A total of 29 studies (N = 3,521) fulfilled the eligibility criteria and were included in the final meta-analysis [5; 7; 10; 19; 20; 23; 29; 30; 32; 34; 36; 37; 39; 41; 44; 52; 58; 60; 61; 66; 69; 73; 77; 78; 81; 85; 92; 95; 96]. Pooling the results of the 29 studies, an average $r_{equivalent}$ effect size of −0.23 (95% CI −0.36 to −0.13; P < .0001) was observed between PA and chronic pain severity.

3.3.1 Observational studies—Eight observational studies [29; 36; 52; 61; 69; 77; 81; 95] were included in the primary analysis, totaling 1,482 participants. Figure 2 displays the forest plots for the meta-analyses of the association between PA and pain severity in this group of studies. PA was associated with decreased chronic pain severity (β = −0.13, z = −4.60, P < 0.001), but heterogeneity across studies was substantial (I^2 = 89.25%; 95% CI 73.6 to 96.3; Q_E (13) = 84.69, P < 0.001), indicating significant variation in the effect sizes. Egger's test for funnel plot asymmetry yielded a result significant at the 0.10 level (z = −1.78, P = 0.08). The funnel plot however (eFigure 2 in the Supplement) suggested no readily detectable presence of bias, as effect sizes tended to cluster somewhat evenly within the funnel. We also conducted p-uniform* as an additional measure of publication bias. This test did not yield a significant result (L_{pb} = 0.83, P = 0.66), failing to indicate the presence of publication bias. Observed p-curve for observational studies are reported in eFigure 3 in the Supplement. The shape of the p-curve was signficantly right-skewed (z = −9.23 P < 0.001), indicating the set of studies contains evidentiary value.

3.3.2 Experimental studies—Five experimental studies [19; 44; 58; 73; 92] provided data on pain severity for 282 participants. Contrary to expectations, PA was not associated with pain severity in experimental studies (Hedges' g = −1.02; Z = −1.55; P = 0.12). Heterogeneity was significant and high (I^2 96.1%; 95% CI 87.3 to 99.6; Q_E (4) = 33.87, P < .001), and funnel plots (eFigure 4 in the Supplement) and the Egger's test (z = −5.18, p < 0.0001) suggested asymmetry. Likewise, the p-uniform* test yielded a significant result (L_{pb} = 5.83, P = 0.05), indicating the likely presence of publication bias. As shown in the forest plot in Figure 3, there were two studies with small sample sizes but relatively large effect sizes [58; 73], whereas the larger studies all had effect sizes that were much closer to zero. eFigure5 in Supplement reports the observed p-curve for expertimental studies, which was signficantly right-skewed (z = −10.1, P < 0.001), suggesting the set of significant findings contains evidentiary value.

3.3.3 Intervention studies—Sixteen intervention studies [5; 7; 10; 20; 23; 30; 32; 34; 37; 39; 41; 60; 66; 78; 85; 96] provided data on pain severity for 1,757 participants. As shown in Figure 4, PA was inversely associated with chronic pain severity (Hedges' g = −0.36; Z = −3.54; P < 0.001). Heterogeneity was high (I^2 = 73.8%; 95% CI 52.6 to 90.1; Q_E (17) = 59.53, P < 0.001), and funnel plots (eFigure 6 in the Supplement) and the Egger's test (z = −2.34, P = 0.02) suggested potential asymmetry. However, the p-uniform* test yielded a non-significant result (L_{pb} = 0.31, P = 0.86), indicating lack of evidence for publication bias. eFigure 7 in Supplement reports the observed p-curve for expertimental studies, which was signficantly right-skewed (z = −9.69, P < 0.001), suggesting the set of significant findings contains evidentiary value.

3.4 Subgroup and exploratory analyses

A priori subgroup analyses within observational and intervention studies are shown in eTables 1–2 for categorical moderators and eTables 3–4 for continuous moderators (see Supplement). Subgroup analyses were not conducted on experimental studies due to the small number of studies in this cluster. With respect to the categorical moderators, lower effect sizes were found for observational studies that adjusted for NA (β = −0.09; Z = −2.91; P = 0.003) compared to those that did not (β = −0.18; Z = −4.39; P = < 0.0001); however, the difference between effect sizes did not reach conventional levels of statistical significance, Q_M = 3.52, p = 0.06. For continuous moderators, the gender composition of the sample moderated the relation between PA and chronic pain severity, such that observational study samples with a higher proportion of female participants reported larger effect sizes on average, Q_M= 22.68, p < 0.0001. Finally, larger effect sizes were found in intervention studies with older compared with younger samples, Q_M = 5.82, p = 0.015. This moderating effect, however, became non-significant following FDR correction, p = 0.125.

Post hoc exploratory analyses were conducted to examine intervention studies that reported data on the magnitude of change in PA (n = 6) and average rates of depression in the sample (n = 5). Among the included studies, effect sizes did not differ significantly as a function of reported PA change, Q_M= 0.05, p = 0.823. Studies reporting higher rates of depression had effect sizes that were not significantly different compared to those reporting lower rates of depression, Q_M= 0.17, p = 0.680.

4. Discussion

4.1 Main findings

This systematic review and meta-analysis provides quantitative evidence that PA is associated with reduced pain severity among adults with CNCP. Previous narrative reviews [28; 38; 57] have reported links between PA and pain. In the present review, we undertook a meta-analysis of observational, experimental, and intervention studies, enabling the quantification of these links and the exploration of key sources of heterogeneity across studies. Pooling the results of 29 observational, experimental, and interventions studies, we found an average r-effect size of −0.23 between PA and pain severity among people with CNCP. This effect size is similar to the effect size between PA and acute physical pain (r = −.18) found by Kushlev et al. [53], and smaller than the effect size reported by Howell et al. [45] in their meta-analyses examining the effects of laboratory-induced PA on pain tolerance (r = .32).

Effect sizes were relatively small in intervention studies (g = −.36). This finding is in line with two previous meta-analyses of RCT's on the effects of PA-based interventions on other pain-relevant outcomes, including depression and anxiety. Hendriks et al. [43] reported relatively small effects on these outcomes (effect sizes ranged from −.35 to −.39), and a recent met-analysis by Chakssi et al. [13] conducted among clinical samples with psychiatric or somatic disorders also reported small effects for depression (g = −.23) and anxiety (g = −.36), respectively. Within observational studies, a small significant effect size of −.13 was found between PA and chronic pain severity. The magnitude of this effect is similar to that

found in a prior meta-analysis examining the association between NA-based predictors (e.g., fear avoidance) and pain intensity [51].

In addition, we examined the impact of categorical (risk of bias, chronic pain status, state vs. trait PA measurement, NA adjustment) and continuous moderators (age, gender, race). Subgroup analyses revealed mainly non-significant associations between PA and chronic pain severity. These analyses showed only three significant moderating characteristics of the sample: gender and age. Notably, PA was associated with lower chronic pain severity in observational studies that adjusted for NA and had a higher percentage of female vs. male participants. Additionally, within intervention studies, age moderated the link between PA and pain severity, with larger effect sizes evident in studies with older compared with younger samples. Finally, in exploratory analyses, neither reported change in PA nor depression composition was found to significantly moderate the relation between PA and chronic pain severity.

4.2 Strengths and limitations

There are several strengths to this review, including its pre-registered design, comprehensive search strategy, systematic study inclusion, thorough assessment of study quality, use of a priori subgroup analyses, and formal tests for heterogeneity (Q-statistic; I^2) and publication bias (p-curve and p-uniform*). There are also limitations to our review. First, although inspection of the funnel plot and Egger test did not identify strong evidence of publication bias in any of our analyses, we found high heterogeneity in terms of study population. Second, although moderating analyses revealed mainly non-significant associations, the small number of studies within each cluster prevented us from performing high-powered subgroup analyses [97]. Third, study quality did not prove to be a significant moderator of the PA-pain effect sizes. However, it is possible that our risk assessment instrument did not adequately capture the range of biases inherent in different types of study designs (e.g., observational, experimental, and intervention studies). That is, we assigned quality ratings based on an overall assessment of risk [72]; however, an alternative approach would be to use design-specific criteria to assess common sources of bias specific to certain types of study designs [89]. Fourth, only a small number of experimental (n = 2) and intervention studies (n = 6) reported data on the magnitude of change in PA, thus leaving unanswered the question of whether PA is the active psychological component in the causal chain. Thus, it is critical that researchers report data on the magnitude of change in the primary outcome (i.e., PA) as a means of assessing the efficacy of experimental and intervention procedures. Such data can then be used as sample-specific moderators in subsequent meta-analyses of PA-pain effect sizes. Likewise, few observational studies (n = 2 ambulatory; n = 3 longitudinal) examined relationships between PA and chronic pain severity while controlling for measures of NA. An important methodological issue in studies of PA and health is whether relationships are independent of negative affective states [71; 80]. In subgroup analyses, we found marginally lower effect sizes in observational studies that adjusted for NA compared to those that did not. However, with such a small number of studies, definitive conclusions cannot be drawn. Similarly, few of the included intervention studies (n = 5) assessed rates of depression. It is known that depression is highly comorbid with the occurrence of chronic pain [4; 18], and there is some evidence that depression may be an important moderator of

interventions targeting PA regulation in chronic pain patients [20; 96]. Given the limited number of studies reporting on depression, firm conclusions on the effects of PA-enhancing interventions on chronic pain severity among clinical populations cannot yet be made. Fifth, the current meta-analysis focused on pain severity as the primary outcome, but the effects of PA on other salient outcomes (e.g., pain interference, pain catastrophizing) in people with CNCP need to be established in future research. Finally, despite the inclusion of gray literature, we used English search terms, which may have prevented us from identifying relevant studies published in other languages.

4.3 Implications for research and practice

Our findings support guideline recommendations [16; 24] that encourage clinicians to consider psychological treatments in the care of patients with CNCP, particularly interventions that have a PA component. Nevertheless, it is unclear whether existing nonpharmacological treatments for CNCP that incorporate elements of PA enhancement (e.g., MBSR [33]; acceptance and commitment therapy [ACT][42]; and emotional awareness and expression therapy [EAET][56]) are sufficient in reducing pain severity or whether the efficacy of these treatments to boost PA can be further strengthened [28]. An additional critical question is whether psychological treatments for CNCP that promote PA have greater benefits than those that are aimed at reducing NA. There is evidence that treatment modalities that incorporate mindfulness-based strategies (e.g., relaxation, present-focused awareness) may be an effective treatment alternative to standard CBT [14; 21; 86]. However, as Finan et al. [28] have noted, treatment approaches for CNCP like CBT, ACT, and MBSR typically emphasize minimizing negative thoughts and emotions associated with pain. As a consequence, it is currently unclear which therapeutic mechanisms (PA-enhancing or NA-reducing strategies) should be optimized in existing psychosocial treatments for CNCP. Finally, it also may be possible that interventions that integrate both PA-based and NA-based strategies could augment the therapeutic impact of current empirically-supported treatments for CNCP [39].

5. Conclusion

To our knowledge, this is the first pre-registered systematic review and meta-analysis to examine the association between PA and pain severity in adults with CNCP. The results indicated that PA is associated with a modest decrease in pain severity across observational, experimental, and intervention studies. The findings suggest that among adults with chronic non-cancer pain, PA may be a factor that promotes resilience in the face of chronic pain.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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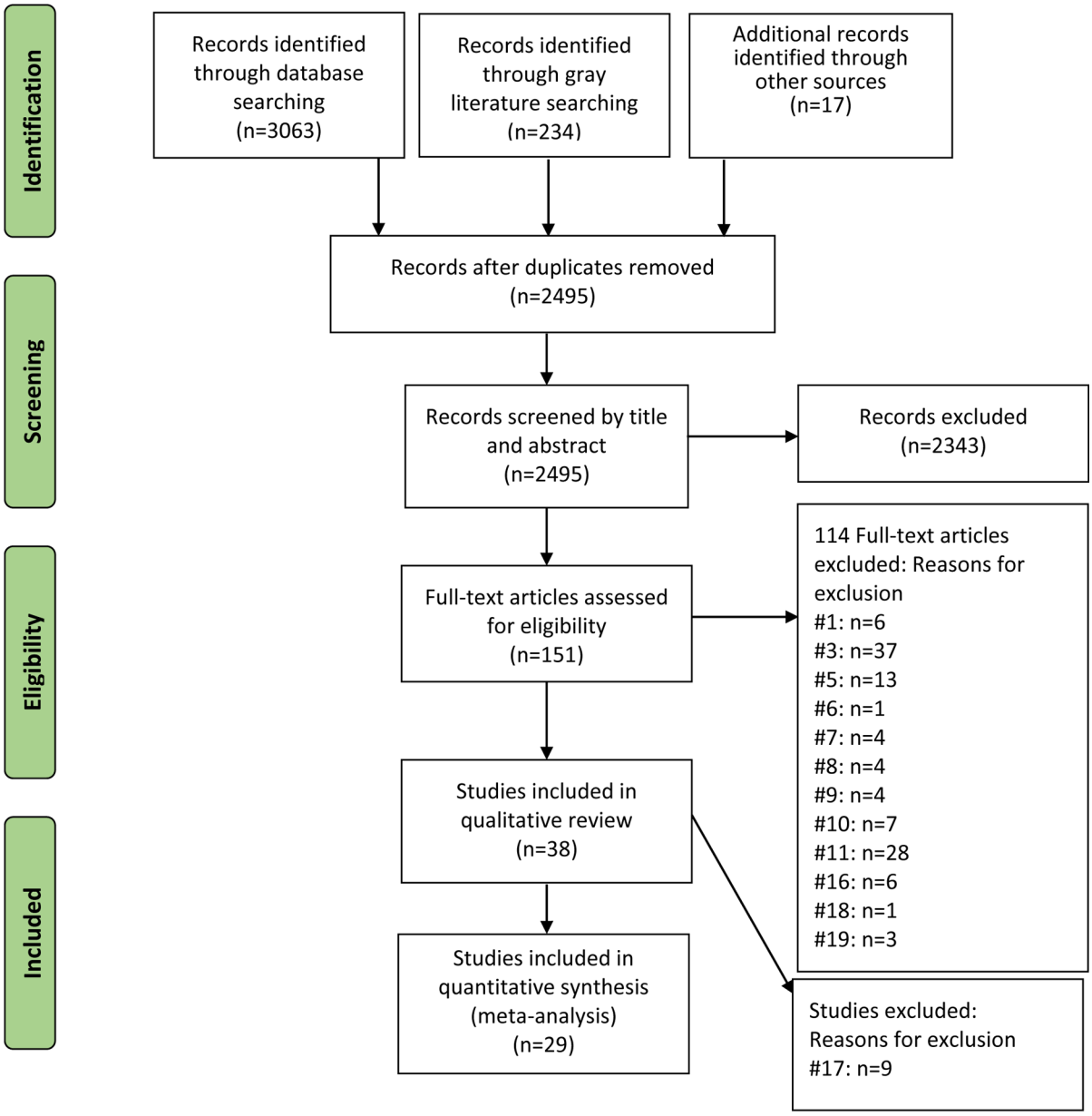

Figure 1.

Study flow diagram. Articles were excluded if they met any one of the following criteria: 1) were not an empirical study; (2) did not involve human subjects; (3) did not include a measure of PA or a positive mood manipulation (e.g., humorous films, pleasant images); (4) used a reversed indicator of negative affect (NA) as a measure of PA (e.g., hopelessness versus hopefulness; pessimism versus optimism; fatigue versus vigor/vitality); (5) did not include a subjective or objective measure of pain severity; (6) assessed affect only in regard to a specific life experience (e.g., "How happy are you about being pregnant?"); (7) examined only the directional effect of pain on PA or mean differences in PA between pain impaired and non-impaired samples; (8) did not enroll individuals identified as suffering from chronic pain; (9) assessed acute or induced pain; (10) did not employ an eligible study

design; (11) did not examine or report a directional effect of PA on pain; (12) exclusively used a sample of patients with a primary cancer diagnosis; (13) were published in languages other than English; (14) did not include adults (18 years and above); (15) exclusively used a sample of patients presenting with a primary psychological disorder; (16) did not directly target PA; (17) did not include full quantitative results on outcomes; (18) full text of article could not be located; or (19) were a duplicate identified during full-text screening.

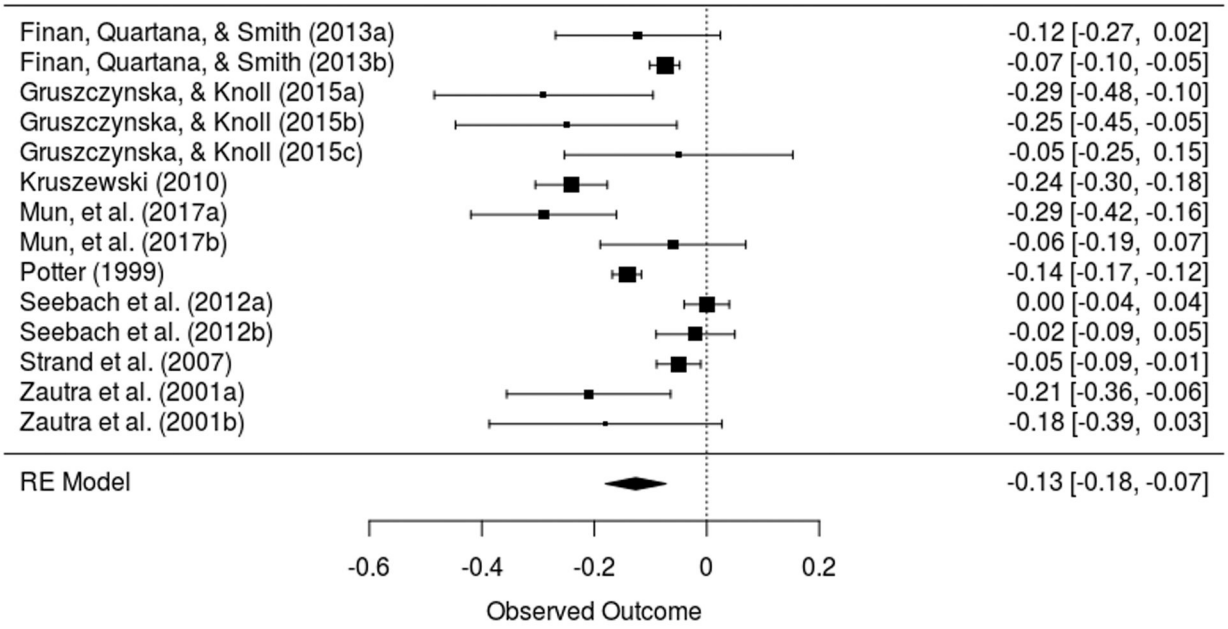

Figure 2.
Forest plot for positive affect and pain in observational studies.

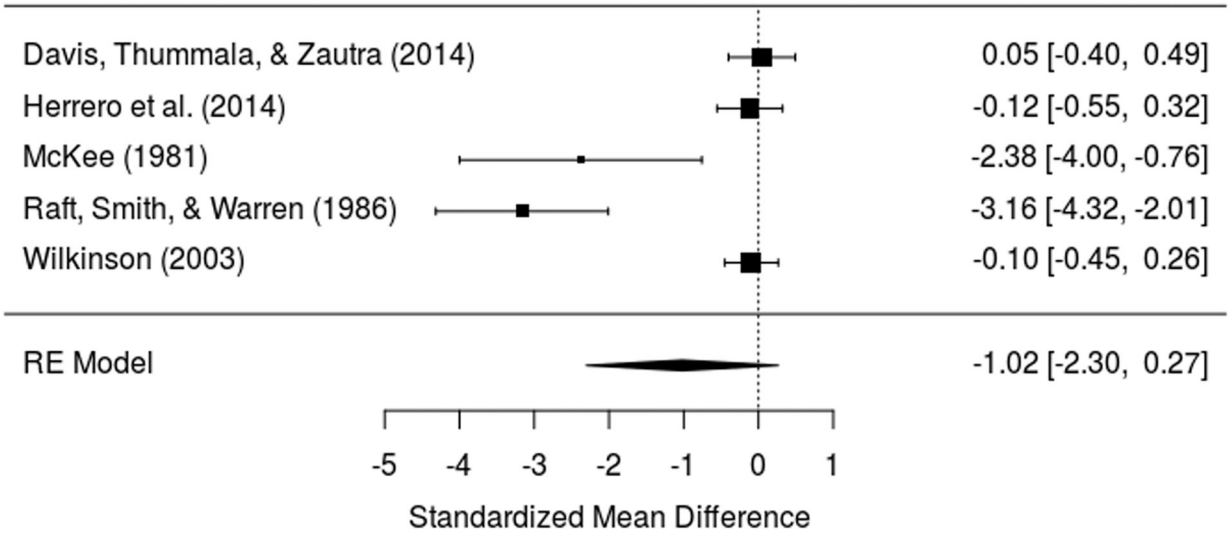

Figure 3.
Forest plot for positive affect and pain in experimental studies.

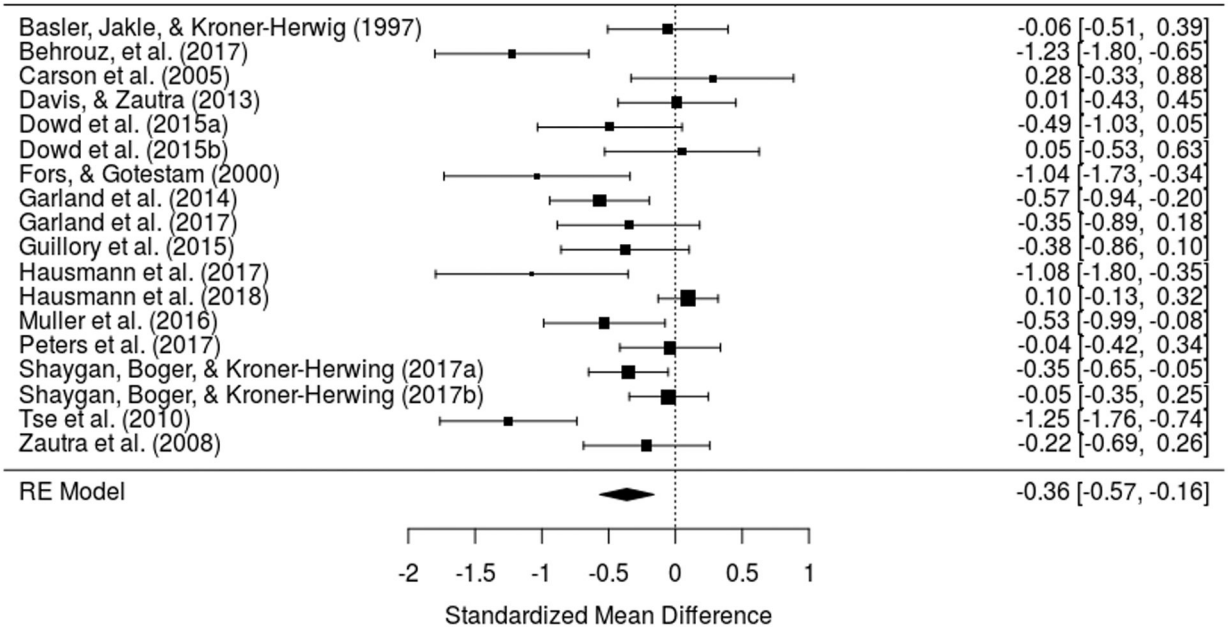

Figure 4.
Forest plot for positive affect and pain in intervention studies.

Table 1:

Descriptive Characteristics of Included Studies

Source [Country]	Year	Study type	Risk of bias	Sample size	Mean age	% white	% female	Pain condition	PA Manipulation/Measure	Pain Outcome
Arnold et al [1] [Germany]	2008	E	W	120	49	---	83	M	Mood induction procedure using the IAPS; PANAS	Pain intensity via NRS
Baird et al [3][US]	2006	I	S	28	73	---	100	O	GIR; mood measured as a subscale of HRQOL	Pain frequency and intensity via NRS
Basler et al [5] [Germany]	1997	I	S	76	49	---	76	B	CBT	Pain intensity and pain control via NRS
Baxter et al [6][New Zealand]	2012	I	M	8	55	---	50	B	Character strengths & gratitude intervention; DESP; OHQSF	Pain intensity via VAS
Behrouz et al [7][Iran]	2017	I	M	55	74	---	71	M	Humor therapy	Pain intensity via BPI (Modified German Version)
Carson et al[10] [US]	2005	I	M	43	51	63	61	B	Loving-kindness meditation	Pain intensity and pain rating index via MPQ; Usual and worst pain via BPI
Connelly et al [17][US]	2007	A	W	94	56	91	72	R	PANAS	Pain intensity via VAS
Davis & Zautra[20] [US]	2013	I	M	79	46	83	98	F	MB intervention targeting socioemotional regulation; PANAS	Daily pain intensity and coping via NRS
Davis et al [19][US]	2014	E	M	110	57	91	100	M	Positive and neutral mood induction; PANAS-X	Clinical pain via NRS
Dowd et al[23] [Ireland]	2015	I	W	124	44	---	90	M	MBCT with emphasis on emotional regulation; SLS; MAAS	Pain intensity and interference via BPI
Finan et al [27][US]	2009	A	W	260	57	88–93	100	M	PANAS	Daily average pain via NRS
Finan et al[29] [US]	2013	A	W	151	61	62	68	O	PANAS-X; POMS - Bipolar	Pain severity via WOMAC
Fors, & Götestam[30] [Norway]	2000	I	M	58	46	---	100	F	GI	Pain intensity via VAS
Garland et al[34] [US]	2014	I	S	115	48	65	68	M	MB, CBT	Pain intensity and interference via BPI
Garland et al [32][US]	2017	I	W	55	49	75	62	M	MB, CBT	Pain intensity via NRS
Gruszczynska et al[36] [Poland]	2015	A	W	95	51	---	100	R	Folkman & Lazarus PA Scale	Daily pain via VAS

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Source [Country]	Year	Study type	Risk of bias	Sample size	Mean age	% white	% female	Pain condition	PA Manipulation/Measure	Pain Outcome
Guillory et al [37][US]	2015	I	M	68	49	63	75	M	Social support text messaging intervention; PAM	Pain intensity and interference via NRS
Hausmann et al [41] [US]	2017	I	S	42	68	57	17	O	PPI; PANAS; SLS	Pain severity and functional difficulty via WOMAC
Hausmann et al [39] [US]	2018	I	S	360	64	50	24	O	PPI; International PANAS-SF; SLS	Pain severity and functional difficulty via WOMAC
Herrero et al [44] [Spain]	2014	E	S	40	49	---	100	F	Virtual reality; Mood state via VAS; Mood intensity via NRS	Pain intensity via NRS
Kamping et al [47] [Germany]	2013	E	S	32	52	---	100	F	Mood induction procedure using the IAPS	Pain intensity via VAS
Kruszewski [52][US]	2010	A	W	143	58	89	100	M	PANAS	Average daily pain via NRS
Litt et al [55][US]	2004	A	W	30	36	80	87	TMD	PANAS; Circumplex Model of Mood	Pain intensity via NRS
McKee [58] [US]	1981	E	M	20	36	90	50	M	GIR	Pain intensity via NRS
Mun et al [61][US]	2017	A	W	220	51	78	87	R	Shortened PANAS	Overall daily pain intensity via NRS
Müller et al[60] [US]	2016	I	M	96	59	96	70	O	Tailored PPI; PANAS; PWI-A	Pain severity via NRS
Peters et al[66] [the Netherlands]	2017	I	S	284	49	---	85	F	Internet-based PPI; Present happiness via NRS; SCS-SF; BMIS	Pain intensity via NRS
Pintard [68][US]	1986	E	W	60	52	77	65	M	Humor induction; HA; POMS; CHS	Present pain intensity and pain rating index MPQ
Potter[69] [US]	1999	L	W	285	63	---	100	M	PANAS	Aggregate of average level of pain over last week,
Raft et al[73] [US]	1986	E	M	52	---	85	60	M	Pleasant imagery task	Pain intensity via VAS
Seebach et al[77] [US]	2012	L	W	141	59	81	58	B	PANAS	Pain intensity via BPI
Shaygan et al[78] [Germany]	2017	I	M	88	53	---	67	M	Visual stimuli (e.g., pictures of loved ones, landscapes); Valence and arousal via SAM	Average pain intensity via NRS
Strand et al[81] [Norwary]	2007	L	W	163	50	---	79	M	PANAS	Past week's average pain via NRS
Tse et al[85] [Hong Kong]	2010	I	M	70	79	---	54	M	Humor therapy; SHS; Revised LSI-A	Cantonese VRS

Source [Country]	Year	Study type	Risk of bias	Sample size	Mean age	% white	% female	Pain condition	PA Manipulation/Measure	Pain Outcome
Wilkinson[92] [US]	2003	E	S	60	---	---	75	M	SHRQ	Current pain rating and location via NRS
Willmarth[93][US]	1998	E	M	96	45	---	41	M	Hypnotically-induced positive mood	Sensory, affective and overall global pain ratings via VAS
Zautra et al[95] [US]	2001	A	W	175	64	95	100	M	PANAS	Average past week pain via NRS
			W	89	44	---	100	F	MAC	Current pain via NRS, averaged across body parts
Zautra et al[96] [US]	2008	I	S	144	52	89	68	R	CBT; MB intervention targeting emotion regulation and adaptation; PANAS	Average daily pain via NRS

Notes. PA = Positive Affect; US = United States. Study type: A = ambulatory, L = longitudinal; E = experiment, I = intervention. Risk of bias: W = weak; M = moderate; S = strong. Pain conditions: F = fibromyalgia, O = osteoarthritis, R = rheumatoid arthritis, B = back pain, TMD = temporomandibular dysfunction, M = multiple. Measure and intervention abbreviations: **BMIS** = Brief Mood Introspection Scale; **BPI** = Brief Pain Inventory; **Cantonese VRS** = Cantonese Verbal Rating Scale; **CBT** = Cognitive Behavioral Therapy; **CHS** = Coping Humor Scale; **DESP** = Differential Emotions Scale Probe; **GI** or **GIR** = Guided Imagery or Guided Imagery Relaxation; **HA** = Humor Appreciation; **HRQOL** = Health-related Quality of Life; **IAPS** = International Affective Picture System; **International PANAS-SF** = International Positive and Negative Affect Schedule Short Form; **MAAS** = Mindful Attention Awareness Scale; **MAC** = Mood Adjective Checklist; **MB** = Mindfulness-based; **MBCT** = Mindfulness-based Cognitive Therapy; **MPQ** = McGill Pain Questionnaire; **NRS** = Numeric Rating Scale; **OHQSF** = Oxford Happiness Questionnaire Short Form; **PAM** = Photographic Affect Measure; **PANAS** = Positive and Negative Affect Schedule; **PANAS-X** = Positive and Negative Affect Schedule, Expanded Form; **POMS** = Profile of Mood States; **POMS-Bipolar** = Profile of Mood States – Bipolar; PPI = Positive Psychology Intervention; **PWI-A** = Personal Wellbeing Index (Adult version); Revised **LSI-A** = Revised Life Satisfaction Index – Form A; **SAM** = Self-assessment Manikin; **SCS-SF** = Self-compassion Scale Short Form; **SF-12** = Short-Form Health Survey; **SF-MPQ** = Short-form McGill Pain Questionnaire; **SF-MPQ-2** = Short-form McGill Pain Questionnaire 2; **SHRQ** = Situational Humor Response Questionnaire; **SHS** = Subjective Happiness Scale; **SLS** = Satisfaction with Life Scale; **VAS** = Visual Analog Scale; **WOMAC** = Western Ontario MacMaster Universities (Osteo-)Arthritis Index.