

Efficacy of Biofeedback in Chronic back Pain: a Meta-Analysis

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Abstract

Purpose The aims of the present analysis were to investigate the short- and long-term efficacy and treatment moderators of biofeedback as a psychological treatment option for chronic back pain.

Method A literature search using PubMed, PsycINFO, and the Cochrane Library identified 21 eligible studies including 23 treatment conditions and 1062 patients.

Results Meta-analytic integration resulted in a significant small-to-medium effect size for pain intensity reduction (Hedges' $g = 0.60$; 95 % confidence interval (CI) 0.44, 0.76) that proved to be stable with a significant small-to-large effect size (Hedges' $g = 0.62$; 95 % CI 0.40, 0.84) over an average follow-up phase of 8 months. Biofeedback also proved to be effective in reducing depression (Hedges' $g = 0.40$; 95 % CI 0.27, 0.52), disability (Hedges' $g = 0.49$; 95 % CI 0.34, 0.74), reduction of muscle tension (EMG; Hedges' $g = 0.44$; 95 % CI 0.22, 0.65), and improving cognitive coping (Hedges' $g = 0.41$; 95 % CI 0.26, 0.57). These effects remained comparatively stable at follow-up and for controlled studies only. Moderator analyses revealed longer biofeedback treatments to be more effective for reducing disability and a greater proportion of biofeedback in the treatment to be more effective for reducing depression. Publication bias analyses demonstrated the consistency of these effects.

Conclusion It is concluded that biofeedback treatment can lead to improvements on various pain-related outcomes in

the short and long terms, both as a standalone and as an adjunctive intervention.

Keywords Chronic back pain · Biofeedback · Psychological treatment · Meta-analysis

Introduction

Chronic pain is one of the major challenging health problems in Western societies. The (lower) back is the most common site for chronic pain [1–3], with a lifetime prevalence of 30 to 40 %. First episodes of low back pain have been reported for children and adolescents. The early onset of back pain has been found to be a significant predictor for chronic back pain in the adulthood [4, 5]. Individuals suffering from chronic back pain report substantial impairments in their daily activities, child care, social functioning, and work functioning, as well as lower overall quality of life [1, 6]. In addition to pain and disability, high muscle tension, low self-efficacy, and depression are common side effects of chronic back pain. Furthermore, chronic back pain is associated with high medical expenses, interferences with employment like work absenteeism, and disability, and therefore results in high socioeconomic costs [7–9]. Hence, it appears essential to identify effective and economical treatments for chronic back pain and associated impairments.

Psychological interventions have been shown to be effective in the treatment of chronic pain by reducing “pain, disability, psychological distress, and catastrophic ways of thinking” ([10], p.15). Henschke and colleagues [11] demonstrated in their systematic review that psychological treatments with cognitive behavioral elements are more effective than physical therapy, medication treatments, or back school in the short term, but there were no significant differences among specific psychological treatments (operant, cognitive,

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or respondent). Biofeedback, as a psychological treatment, is a very popular intervention among therapists and patients due to its combination of physiological and psychological methods. It is performed both as a standalone approach and as an additional element within cognitive behavioral therapy (CBT) or physical therapy. During biofeedback sessions, patients receive auditory, visual, or tactile feedback about physiological processes from their autonomous or central nervous system such as muscle tension, heart rate, or skin conductance. Biofeedback can be described as “operant conditioning of physiological activity” ([12], p. 35), by which “the patient learns to self-regulate his or her physiological processes with the help of feedback information” ([12], p. 36), and can comprise different sites, modalities, and procedures. There are various objectives biofeedback can target, e.g., developing more awareness or control for physiological processes and thus, consciously reducing muscle tension or influencing muscle imbalances. This is especially interesting in light of findings of higher baseline muscle activation and abnormal EMG responding to stress in chronic back pain patients [13–15]. But also, increases in self-efficacy and coping strategies can be aims of biofeedback treatment. Although, electromyographic (EMG) biofeedback is the most common used modality in the treatment of chronic pain, heart rate variability (HRV) or respiratory biofeedback, e.g., to support relaxation training and posture biofeedback, are also common. To date, there is no clear evidence of what is the primary mechanism of action for biofeedback in the treatment of chronic back pain. The beneficial effects of biofeedback have been demonstrated in pain conditions including chronic headache, temporomandibular disorders, and fibromyalgia [16–19].

Studies of biofeedback treatment in chronic back pain patients have shown inconsistent results. In a meta-analysis on psychological interventions for chronic low back pain (CLBP), Hoffman and colleagues [20] found that biofeedback was more effective than cognitive behavioral approaches in reducing depressive symptoms in CLBP patients. In addition, Flor and Birbaumer [21] found biofeedback treatment, relative to CBT and a waitlist control group, to be more effective at reducing pain severity and producing changes on affective, cognitive, and behavioral variables over the long term. Magnusson and colleagues [22] examined the effectiveness of postural biofeedback added to a conventional physiotherapy treatment for CLBP; results indicated an advantage of the enhanced treatment condition at 6-month follow-up, but these results should be viewed with caution due to the small sample size ($n = 10$). However, in a randomized controlled trial with a highly disabled sample, Glombiewski and colleagues [14] compared the effectiveness of CBT, CBT enhanced with biofeedback, and a waitlist control condition, and observed comparable improvements on pain-related outcomes in the two treatment groups over both the short and long terms, while the waitlist control group did not significantly improve. Two other studies demonstrated little to no improvement in chronic back pain after biofeedback [23, 24].

It is difficult to draw firm conclusions based on extant studies due to variability in sample size and characteristics, biofeedback modality (EMG, postural, or respiratory), treatment conditions, and control groups. Some studies examined biofeedback as a standalone intervention, while others examined biofeedback as an additional feature in conventional treatments. Control groups have included waitlist control groups, CBT, and physiotherapy, while other studies have not included control groups.

Thus, the effectiveness of biofeedback in reducing the symptomatology of back pain patients remains unclear. We therefore conducted a meta-analysis of controlled and uncontrolled chronic back pain treatment studies that included biofeedback, to examine short-term and long-term effects of biofeedback on pain-related outcomes. This meta-analysis focuses on studies which report biofeedback treatments as a standalone intervention as well as part of any treatment with at least 25 % biofeedback intervention of the total treatment time. Secondly, given the methodological variability in existing studies, another aim of the present meta-analysis was to determine the specific efficacy of biofeedback compared to various different control groups. In addition, moderator analyses were conducted to identify potential moderators of treatment effects.

Methods

Search Procedure

The meta-analysis was conducted in accordance with the QUORUM guidelines, taking into account the recent updates to these guidelines (“PRISMA guidelines” [25]). Studies were identified by searching PubMed, PsycINFO, and the Cochrane Library using the search term *biofeedback* combined with the term *back pain*. Studies published between the first available year and April 14, 2014, were included in the meta-analysis. In addition, reference lists from relevant studies and review papers identified in the database searches were manually reviewed. It was determined a priori that only published studies would be included. These search procedures identified 412 unique articles, which were then further examined by two independent reviewers (RS and JAG) for potential inclusion in the meta-analysis.

Determination of Outcome Variables

The Initiative on Methods, Measurement and Pain Assessment in Clinical Trials (IMMPACT) [26, 27] recommends the inclusion of a set of core outcome domains (e.g., pain, physical functioning, emotional functioning, symptoms,

and adverse events) and supplemental outcome domains (e.g., coping, interpersonal functioning) in clinical trials of pain treatments. Following these recommendations, we included average pain intensity as a primary outcome [28] and as a measure of the core outcome domain “pain.” Other outcome measures included the following: disability, as a measure of the suggested core outcome domain “physical functioning”; depression or another affective state (if depression was not assessed), as a measure of the core outcome domain “emotional functioning”; self-efficacy or coping (subsequently referred to as cognitive coping), as a measure of the supplemental outcome domain “coping”; and reduction of muscle tension (EMG), as an additional outcome, since pain is often associated with muscle tenseness.

We also examined biofeedback treatment dose, proportion of treatment time spent on biofeedback, sample size, and methodological quality of the studies as potential moderators.

Study Selection

Inclusion criteria for studies were as follows:

1. Study included patients with chronic back pain (in any region of the back)
2. Study included an adult sample (age 18 or older)
3. Study employed a biofeedback intervention of any kind for at least 25 % of the total treatment time
4. Study reported measures of at least one of the main outcome variables (see above) at both pre- and post-intervention or pre-intervention and follow-up
5. Study provided sufficient data to perform effect size analyses

If available, follow-up data (from the longest available follow-up) and data for control groups were included. Publications in English and German were considered.

Studies meeting the following criteria were excluded:

1. The study was a case study.

Validity Assessment

No additional methodological criteria were applied, and the meta-analysis included randomized controlled trials (RCTs) as well as uncontrolled or nonrandomized studies. However, to allow for comparison of effect sizes for RCTs and less methodologically sound studies and to control for confounding effects of study quality on effect size [30], we rated the quality of each study on a validity scale and analyzed this quality score as a moderator of the study findings. The validity scale was developed in a previous study by one of the authors (JAG,

[14])¹ by adapting Jadad criteria for pharmacological trials [31] and following PRISMA recommendations [25]. The validity scale includes aspects of internal, external, and construct validity and includes 20 dichotomous items, with a maximum score of 20. For each study, validity was assessed independently by two reviewers (JAG and RS) and inter-rater reliability was calculated. Disagreements were resolved through discussion.

Data Extraction

For each study, two of the authors (JAG and RS) independently selected psychometrically validated measures of pain intensity, depression, disability, cognitive coping, and reduction of muscle tension (EMG); these authors also extracted numerical data for analysis of changes from pre- to post-treatment and from pre-treatment to follow-up. Numerical data from 50 % of the studies was double checked by two of the authors (JAG and RS) to ensure reliability. Cohen’s kappa was calculated for categorical items, and intraclass correlations (ICCs) were used for items measured on an interval scale. Cohen’s kappa was 0.96 (95 % CI 0.95–0.97). All variables had significant ICCs of 0.97 or higher, with the exception of dropout rates. Differences were discussed and clarified.

Quantitative Data Synthesis

All analyses were completed manually or using the software program Comprehensive Meta-Analysis, version 2 [32]. Intention-to-treat (ITT) data were analyzed when available; when ITT data were not available, data from study completers were included. We calculated separate effect sizes for continuous measures of pain intensity, depression, disability, cognitive coping, and reduction of muscle tension (EMG) using within-group pre-post treatment differences for all studies and also for the group of controlled studies. Effect sizes were calculated using Hedges’ *g*, a variation of Cohen’s *d* that corrects for biases due to small sample sizes [33], and its 95 % confidence interval. Within-group effect sizes were calculated using the following formula:

$$d = \left(\frac{Y_1 - Y_2}{S_{\text{Difference}}} \right) \sqrt{2(1-r)},$$

where Y_1 is the pre-treatment sample mean, Y_2 is the post-treatment sample mean, $S_{\text{Difference}}$ is the standard deviation of the difference, and r is the correlation between pre-treatment

¹ The full version of the validity scale is available upon request from one of the authors (JAG).

and post-treatment scores. Hedges' g is computed by multiplying d by the correction factor

$$J(df) = 1 - \frac{3}{4df-1},$$

where df is the degrees of freedom to estimate the within-group standard deviation.

The effect sizes for controlled studies were computed using the following formula:

$$g = \frac{\Delta_{BFB} - \Delta_{CONT}}{\sqrt{\frac{(n_{BFB}-1)SD_{CONT}^2 + (n_{CONT}-1)SD_{BFB}^2}{(n_{total}-2)}}} \times \left(1 - \frac{3}{4(n_{BFB} + n_{CONT}) - 9}\right),$$

where Δ is the mean pre- to post-treatment change, SD is the standard deviation of post-treatment scores, n is the sample size, BFB refers to the treatment condition, and $CONT$ refers to the control condition.

The magnitude of Hedges' g can be interpreted using Cohen's [34] recommendations of small (0.2), medium (0.5), and large (0.8).

Although the correlation between pre-and post-treatment measures is needed in order to calculate the pre-post effect sizes, insufficient information on this correlation was included in the studies. We used a conservative estimate of $r = 0.7$, as recommended by Rosenthal [35].

Effect sizes for average pain intensity, depression, disability, cognitive coping, and reduction of muscle tension (EMG) were pooled across studies to obtain a summary statistic. It was decided a priori (based on previous pain research results) that effect sizes for individual studies greater than Hedges' $g = 3.0$ would be considered outliers and would be excluded from the analyses. No studies were determined to be outliers using this criterion. Effect size estimates were calculated using a random-effects model rather than a fixed-effects model because the studies were not functionally identical [36, 37]. Effect size estimates for follow-up data were also calculated in the manner described above.

Sensitivity Analysis

Publication bias may impact the results of a meta-analysis, as studies with nonsignificant results are less likely to be published than studies with significant results.

To address this potential for publication bias, we computed the *fail-safe* N [35], which indicates the number of studies that would be required to reduce the

overall effect size to a nonsignificant level. The fail-safe N was calculated using the following formula:

$$N = \frac{K \left(KZ^2 - 2.706 \right)}{2.706},$$

where K is the number of studies in the meta-analysis and Z is the mean obtained from the K studies. The effect size can be considered robust if the number of studies (X) required to reduce the overall effect size to a nonsignificant level exceeds $5K + 10$ [35]. In addition, we constructed a funnel plot with the pre-post effect sizes for all outcomes and used the Trim and Fill method to examine the symmetry of the plot, which allowed us to determine whether negative or positive trials were over- or under-represented, accounting for the sample size. This information can then be used to re-calculate the effect size estimate.

Moderator Analysis

Four potential moderator variables were tested based on previous research. Quality of studies (assessed with a validity score), proportion of treatment time spent on biofeedback (relative to total treatment time), biofeedback treatment dose (total number of hours spent in biofeedback interventions), and sample size were chosen as potential moderators.

Moderating effects were examined using meta-regression analyses.

Results

Study Selection

The study selection process is shown in Fig. 1. Of the 412 potentially relevant articles identified in initial searches, 21 studies met all selection criteria. As noted above, no studies were excluded due to unusually high effect sizes ($g > 3.0$). These 21 studies included 23 treatment conditions and 1062 patients with back pain (see Tables 1 and 3). As required by the inclusion criteria, all 21 studies provided data for continuous measures of at least one relevant outcome variable at pre- and post-treatment. Eleven of the studies provided data at follow-up.

Characteristics of the Study Sample

Studies and Patient Characteristics

Table 1 provides information about the studies and treatment conditions included in the meta-analysis. Of the 21 studies included in our analysis, 18 used an EMG-based biofeedback

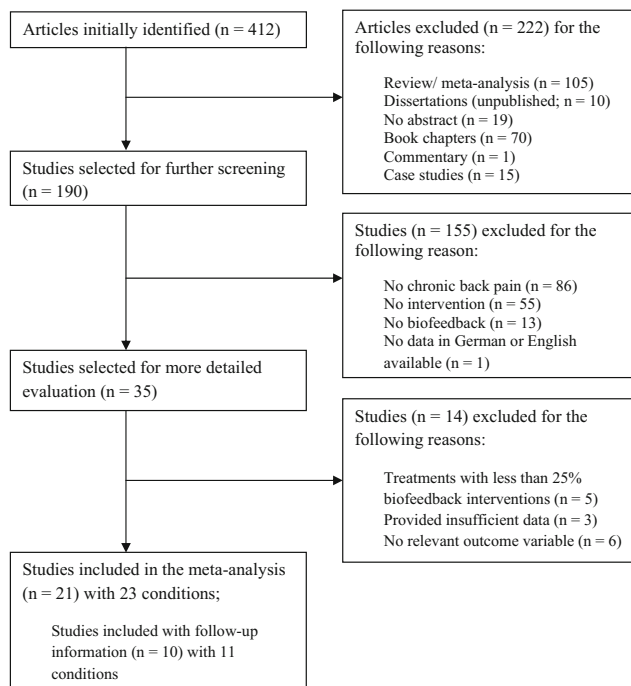


Fig. 1 Study selection process

training ($n = 921$), two ($n = 71$) used respiratory biofeedback, and one ($n = 70$) used postural biofeedback. Fourteen studies placed electrodes on participants' back muscles ($n = 776$), two studies placed electrodes on the front of the torso ($n = 54$), one study used a combination of back and front muscles ($n = 52$), and four studies did not report the placement of electrodes or did not use EMG-based biofeedback ($n = 180$). In eleven studies, the treatment consisted purely of biofeedback with no other intervention. The other studies combined biofeedback with another treatment, such as CBT, relaxation training, physical therapy, psychoeducation, or a combination of the above. These other interventions accounted for 33–75 % of the time study participants spent undergoing an intervention. The total number of minutes of biofeedback intervention ranged from 40 to 2856 ($M = 603$, $SD = 634$).

Five treatment conditions were uncontrolled or did not specify their control group ($k = 1$). Four control conditions consisted of CBT or operant-cognitive treatment, three consisted of physical therapy or waitlist control plus physical therapy ($k = 1$), four consisted of waitlist control, one consisted of relaxation training, two consisted of psychoeducation with or without placebo ($k = 1$), one consisted of a combination of education, physical therapy, relaxation, and psychological interventions, and one consisted of a placebo (noncontingent biofeedback). Because patients in waitlist control conditions (WLC) typically received treatment-as-usual (TAU), we merged studies employing a WLC condition with those employing a TAU control condition for the purpose of moderator analyses.

For 11 of the treatment conditions, follow-up data were reported, with follow-up periods ranging from 3 to 24 months ($M = 8.18$, $SD = 6.5$). The total number of patients across all studies was 1062, with 722 patients enrolled in treatment conditions and 340 patients in control conditions. The samples were predominantly female (65 % of patients). Twenty treatment conditions ($n = 716$ patients), and 16 control conditions ($n = 163$ patients) included sufficient data to compute dropout rates from pre- to post-treatment. A total of 121 patients (16.76 %) and 59 patients (17.35 %) dropped out of the treatment and control conditions, respectively, indicating comparable dropout rates for the treatment and control conditions.

Quality of Included Studies

The quality scores for each study are shown in Table 1. Scores ranged from 2 to 17 points (out of 20; $M = 10.48$, $SD = 4.35$). Two independent ratings of quality criteria were conducted; interrater reliability was $r = 0.98$. All 21 studies described their interventions sufficiently and defined adequate outcome measures. Eleven studies described dropout rates for each group. One study did not adequately describe inclusion and exclusion criteria. Twelve of the 21 studies implemented a manualized or otherwise standardized intervention.

Pre-Post Effect Sizes and Publication Bias

The pre-post effect sizes (Hedges' g) for pain intensity reduction (based on 21 studies with 23 conditions), depression (based on 11 studies), cognitive coping (based on 9 studies), disability (based on 14 studies), and reduction of muscle tension (EMG; based on 10 studies) are displayed in Table 2. All pre-post effect sizes were significant. According to Cohen's interpretation recommendations [34], the effect sizes for pain intensity reduction and disability were medium with confidence intervals suggesting small to medium effects. The effect sizes for depression, cognitive coping, and reduction of muscle tension (EMG) were small with confidence intervals suggesting small to medium effects. For the effect sizes for disability, depression, and cognitive coping, the Trim and Fill method indicated that the number of missing studies that would be needed to make the plot symmetrical was $n = 0$ studies, so all values remained unchanged. For the effect size for pain intensity, the Trim and Fill method indicated that $n = 4$ studies to the right of the mean would be needed to make the plot symmetrical. The adjusted value was Hedges' $g = 0.700$ (95 % CI 0.536–0.864). For the effect size for reduction of muscle tension (EMG), the Trim and Fill method indicated that $n = 1$ study to the right of the mean would be needed to make the plot symmetrical. The adjusted value was Hedges' $g = 0.479$ (95 % CI 0.268–0.689). The effect sizes for all outcomes for single studies are shown in Table 3.

Table 1 Characteristics of included studies

Authors	Year	Type of treatment	Description of intervention (N/N)	Placement of EMG electrode	Total number of minutes of biofeedback intervention	Type of cointervention (% time spent in cointervention)	Control conditions (N/N)	Longest follow-up in months	Measures	Quality score (x/20) ^a
Adams et al. [45]	1982	BFB	EMG (30/30)	Front	40–680	None	None	None	Pain (Analog Scale db) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension	2
Asfour et al. [46]	1990	BFB	EMG and CPRC (15/15)	Back	176	CPRC (75 % est.)	CPRC (15/15)	None	Pain (Level) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	9
Donaldson et al. [38]	1994	BFB	SMUBT/EMG (12/12)	Back	350	None	Relaxation (12/12) Education (12/12)	3	Pain (MPQ) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension	16
Flor and Birbaumer [21]	1993	BFB	EMG (26/23)	Back	480	None	CBT (26/22)	24	Pain (MPQ) Disability (MPI) Emot. Funct. (MPI) Cognitive (Selfefficacy) Muscle tension	16
Glombiewski et al. [14]	2010	BFB	EMG and CBT (62/52)	Back	540	CBT (60 %)	CBT (54/43)	6	Muscle tension Pain (Diary) Disability (PDI) Emot. Funct. (BDI) Cognitive (Coping) Muscle tension n.m.	16
Hallman et al. [47]	2011	BFB	EMG (12/12)	Front	160	Paced breathing	WLC (12/11)	None	Pain (VAS) Disability (NDI) Emot. Funct. (HADS-D) Cognitive (SF-36 GH) Muscle tension n.m.	13
Huis in 't Veld et al. [48]	2010	BFB	EMG (82/52)	Back	2856 (est.)	None	None	None	Pain (VAS) Disability (PDI) Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	8
Kapitzka et al. [42]	2010	BFB	Respiratory BFB (21/21)	Front	450	None	Noncontingent feedback (21/21)	3	Pain (VAS) Disability (PDI) Emot. Funct. (SCL-GSI) Cognitive n.m. Muscle tension n.m.	17
Keefe et al. [49]	1981	BFB	EMG and Phys. (111/111)	Back	Not reported	Physical therapy, relaxation, education (67 % est.)	None	None	Pain (Pain Intensity) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension	3

Table 1 (continued)

Authors	Year	Type of treatment	Description of intervention (N/N)	Placement of EMG electrode	Total number of minutes of biofeed-back intervention	Type of cointervention (% time spent in cointervention)	Control conditions (N/N)	Longest follow-up in months	Measures	Quality score (x/20) ^a
Kröner-Herwig and Beck [50]	2000	BFB	EMG (13/10)	Back	720	None	WCL (13/10)	None	Pain (Diary) Disability (NRS) Emot. Funct. (NRS 0-10) Cognitive (Self-efficacy) Muscle tension	11
Kröner-Herwig and Beck (b) [50]	2000	BFB	EMG (13/10)	Back	720	None	WCL (13/10)	None	Pain (Diary) Disability (NRS) Emot. Funct. (NRS 0-10) Cognitive (Self-efficacy) Muscle tension	11
Magnusson et al. [22]	2008	BFB	Postural BFB and Phys. (47/24 est.)	Back	150	Phys. (33 % est.)	Phys. (23/12 est.)	6	Pain (VAS) Disability (SF-36) Emot. Funct. n.m. Cognitive n.m. Muscle tension	10
McLaughlin et al. [51]	2011	BFB	Respiratory (29/29)	Not reported	Not reported	Awareness training. Manual therapy (67 % est.)	None	None	Muscle tension n.m. Pain (NPRS) Disability (PSFS) Emot. Funct. n.m. Cognitive n.m.	3
Neblett et al. [52]	2010	BFB	SEMGAS BFB (104/71)	Back	Not reported	Phys. (50 %)	Phys. (36/23)	None	Muscle tension n.m. Pain n.m. Disability n.m. Emot. Funct. n.m. Cognitive n.m.	4
Newton-John et al. [53]	1995	BFB	EMG (group of 4) (16/16)	Back	480	None	CBT (16/16)	6	Muscle tension Pain (Diary) Disability (PDI) Emot. Funct. (BDI) Cognitive (Self-efficacy) Muscle tension n.m.	14
Nouwen and Solinger [54]	1979	BFB	EMG (19/19)	Back	900	None	CG without specif. (7/7)	3	Pain (Report) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension	13
Nouwen [23]	1983	BFB	EMG (10/10)	Back	450	None	WCL (10/10)	None	Pain (Report) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension	10
De Sousa et al. [55]	2009	BFB	EMG and Phys. and Cog. (27/26)	Back and front	Not reported	Cognitive restriction techniques. Physical therapy (67 % est.)	WCL and physical treatment (25/18)	None	Muscle tension Pain (VAS) Disability (Roland-Morris DQ) Emot. Funct. (BDI) Cognitive n.m. Muscle tension n.m.	11

Table 1 (continued)

Authors	Year	Type of treatment	Description of intervention (N/N)	Placement of EMG electrode	Total number of minutes of biofeedback intervention	Type of cointervention (% time spent in cointervention)	Control conditions (N/N)	Longest follow-up in months	Measures	Quality score (x/20) ^a
Spence et al. (a) [56]	1995	BFB	EMG (12/11)	Back	Not reported	None	WCL (12/11)	6	Pain (Index) Disability (WHYMPI Activity level) Emot. Funct. (BDI) Cognitive (Coping) Muscle tension n.m.	13
Spence et al. [56]	1995	BFB	EMG and relaxation (12/9)	Back	Not reported	Relaxation (50 % est.)	WCL (12/11)	6	Pain (Index) Disability (WHYMPI Activity level) Emot. Funct. (BDI) Cognitive (Coping) Muscle tension n.m.	13
Strong et al. [57]	1989	BFB	EMG and relaxation (20/19)	Back	105	Relaxation (50 % est.)	Relaxation (20/18)	3–15	Pain (MPQ) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	8
Stuckey et al. [24]	1986	BFB	EMG (8/6)	Back	360	None	Placebo with education (8/6)	None	Pain (VAS) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	9
Vlaeyen et al. [58]	1995	BFB	EMG and relax. (group) (21/13)	Not reported	990	Applied relaxation (50 % est.)	Operant-cognitive treatment (18/14)	12	Muscle tension Pain (VAS) Disability (Pain Impact) Emot. Funct. (Depression) Cognitive (Self-efficacy) Muscle tension n.m.	11

N/N number of subjects, who began and completed the treatment (began/completed), db decibel, *Muscle tension* reduction of muscle tension (EMG), *CPRC* education, physical therapy, relaxation, psychological intervention, *SMUBT* single motor unit biofeedback training, *MPQ* McGill Pain Questionnaire, *CBT* cognitive behavioral therapy, *MPI* Multidimensional Pain Inventory, *PD/Pain* Disability Index, *BDI* Beck Depression Inventory, *WLC* waitlist control, *VAS* visual analog scale, *NDI* Neck Disability Index, *HADS-D* Hospital Anxiety and Depression—Depression, *SF-36* Short Form 36 Item Health Survey, *SCL-GSI* Symptom Check List—Global Severity Index, *Phys* physical therapy, *NPRS* Numeric Pain Rating Scale, *PSFS* Patient-Specific Functional Scale, *SEMGAS BFB* Surface EMG-assisted stretching biofeedback, *CG* control group, *Cog.* cognitive therapy, *Roland-Morris DQ* Roland-Morris Disability Questionnaire, *WHYMPI* West Haven-Yale Multidimensional Pain Inventory, *Est.* estimated values, *n.m.* not mentioned

^a Range [0–20] with a lower value indicating poorer quality of study

Table 2 Effect sizes for all outcome measures pre-post and pre-follow-up

Outcome	Type of effect	<i>k</i>	Hedges' <i>g</i>	95 % CI	<i>z</i>	<i>p</i> value	<i>I</i> ²	Fail-safe <i>N</i> (1/2tailed)
Pain intensity	Pre-post	22	0.601	0.439–0.763	7.29	<0.0001	77	1785/1251
Pain intensity	Pre-follow-up	11	0.623	0.404–0.841	5.59	<0.0001	67	360/251
Disability	Pre-post	14	0.542	0.339–0.744	5.25	<0.0001	79	608/424
Disability	Pre-follow-up	7	0.437	0.220–0.654	3.95	<0.0001	54	84/57
Emot. Funct.	Pre-post	11	0.398	0.272–0.524	6.20	<0.0001	27	205/141
Emot. Funct.	Pre-follow-up	6	0.486	0.145–0.826	2.80	0.005	78	69/47
Cognitive	Pre-post	9	0.414	0.261–0.567	5.32	<0.0001	36	142/97
Cognitive	Pre-follow-up	6	0.493	0.242–0.743	3.86	<0.0001	60	83/57
Muscle tension	Pre-post	10	0.438	0.221–0.654	3.97	<0.0001	75	239/165
Muscle tension	Pre-follow-up	3	0.309	0.032–0.585	2.19	0.029	44	7/4

Emot. Funct. = emotional functioning; Cognitive = cognitive Coping; Muscle tension = reduction of muscle tension (EMG); *I*² = heterogeneity statistics, values are percentages

The funnel plot for pain intensity is depicted in Fig. 2. The fail-safe *N*s are displayed in Table 2. These analyses suggest that the effect size estimates for all outcome variables were unbiased.

Effects at Follow-up

An effect size analysis from pre-intervention to the last available follow-up time point was conducted to examine the stability of biofeedback intervention effects (see Table 2). All follow-up effect sizes were small-to-medium and significant. For each effect size, except depression, the Trim and Fill method indicated that the number of missing studies that would be needed to make the plot symmetrical was *n* = 0 studies, so the Hedges' *g* values remained unchanged. For the effect size for depression, the Trim and Fill method indicated that *n* = 2 studies to the right of the mean would be needed to make the plot symmetrical. The adjusted value was Hedges' *g* = 0.618 (95 % CI 0.301–0.934). The fail-safe *N*s are displayed in Table 2. These analyses suggest that the effect size estimates for all outcome variables were unbiased.

Controlled Effect Sizes

For studies including control groups, we computed controlled effect sizes that compared the effectiveness of the intervention condition against the control condition. For reduction of muscle tension (EMG), the random-effects analysis of the controlled studies employing any control group comparison condition yielded a significant, medium mean effect size. For pain intensity, depression, and cognitive coping, the random-effects analysis of the

controlled studies yielded small but significant mean effect sizes (Hedges' *g*; see Table 4). The mean effect size for disability was not significant. Publication bias analyses suggested that the reported results are robust. These results should be considered preliminary given the small number of control conditions included in the analysis (range from *k* = 17 for the outcome of pain intensity to *k* = 6 for the outcome of reduction of muscle tension, EMG).

For studies including active control groups, the controlled effect sizes for biofeedback were similar in magnitude to those mentioned above. However, analyses of publication bias indicated that these effects were only robust for reduction of muscle tension (EMG) and pain intensity.

For studies including a wait list control group (range from *k* = 6 studies for pain intensity to *k* = 1 study for reduction of muscle tension, EMG), the controlled effect sizes for biofeedback were medium for depression and cognitive coping. The mean effect size for pain intensity was small, with confidence intervals suggesting small-to-big effect sizes. The mean effect sizes for disability and reduction of muscle tension (EMG) were not significant.

Moderator Analyses

To explore possible moderators of biofeedback treatment outcome, we examined study quality, proportion of treatment time spent on biofeedback, dose of treatment, and sample size in moderator analyses using only within-participants data from the treatment conditions. Results for each outcome measure are reported below.

Table 3 Effect sizes for all outcome measures for single studies

Author, publication year	Targeted symptom	Pre-post			Pre-follow-up		
		Hedges' <i>g</i>	95 % CI	<i>p</i>	Hedges' <i>g</i>	95 % CI	<i>p</i>
Adams et al., 1982	Pain	1.138	0.686–1.590	<0.0001	–	–	–
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	1.140	0.687–1.592	<0.0001	–	–	–
Asfour et al., 1990	Pain	0.454	0.062–0.845	0.023	–	–	–
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
Donaldson et al., 1994	Pain	0.582	0.136–1.027	0.011	0.943	0.441–1.444	<0.0001
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	0.319	–0.101–0.738	0.136	0.496	0.061–0.932	0.026
Flor and Birbaumer, 1993	Pain	1.440	1.022–1.859	<0.0001	1.365	0.933–1.797	<0.0001
	Disability	0.872	0.530–1.214	<0.0001	0.735	0.388–1.082	<0.0001
	Emot. Funct.	0.741	0.413–1.069	<0.0001	1.409	0.970–1.848	<0.0001
	Cognitive	0.589	0.275–0.903	<0.0001	0.975	0.600–1.351	<0.0001
	Muscle tension	0.187	–0.109–0.484	0.216	0.427	0.106–0.747	0.009
Glombiewski et al., 2010	Pain	0.329	0.134–0.525	0.001	0.308	0.080–0.536	0.008
	Disability	0.413	0.214–0.611	0.001	0.359	0.130–0.589	0.002
	Emot. Funct.	0.336	0.140–0.532	<0.0001	0.240	0.015–0.466	0.037
	Cognitive	0.588	0.382–0.795	<0.0001	0.625	0.381–0.868	<0.0001
	Muscle tension	–	–	–	–	–	–
Hallman et al., 2011	Pain	0.618	0.167–1.068	0.007	–	–	–
	Disability	0.735	0.268–1.202	0.002	–	–	–
	Emot. Funct.	0.320	–0.100–0.740	0.135	–	–	–
	Cognitive	0.105	–0.304–0.514	0.616	–	–	–
	Muscle tension	–	–	–	–	–	–
Huis in 't Veld et al., 2010	Pain	0.473	0.254–0.693	<0.0001	–	–	–
	Disability	0.353	0.139–0.566	0.001	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
Kapitza et al., 2010	Pain	0.469	0.132–0.806	0.006	0.674	0.319–1.030	<0.0001
	Disability	0.102	–0.218–0.421	0.533	–	–	–
	Emot. Funct.	0.352	0.023–0.681	0.036	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
Keefe et al., 1981	Pain	0.799	0.587–1.012	<0.0001	–	–	–
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	0.337	0.147–0.527	<0.0001	–	–	–
Kröner-Herwig and Beck, 2000	Pain	0.293	–0.157–0.743	0.202	–	–	–
	Disability	0.693	0.195–1.191	0.006	–	–	–
	Emot. Funct.	0.456	–0.010–0.921	0.055	–	–	–
	Cognitive	0.295	–0.155–0.745	0.199	–	–	–
	Muscle tension	0.028	–0.411–0.467	0.901	–	–	–
Kröner-Herwig and Beck, 2000b	Pain	0.718	0.216–1.220	0.005	–	–	–
	Disability	0.661	0.168–1.154	0.009	–	–	–
	Emot. Funct.	0.194	–0.250–0.638	0.392	–	–	–
	Cognitive	0.803	0.286–1.320	0.002	–	–	–
	Muscle tension	0.058	–0.382–0.497	0.797	–	–	–
Magnusson et al., 2008	Pain	1.092	0.701–1.484	<0.0001	0.917	0.420–1.414	<0.0001
	Disability	0.000	–0.306–0.306	1.000	0.577	0.132–1.022	0.011
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
McLaughlin et al., 2011	Pain	1.831	1.375–2.288	<0.0001	–	–	–
	Disability	1.410	1.017–1.802	<0.0001	–	–	–
	Emot. Funct.	–	–	–	–	–	–

Table 3 (continued)

Author, publication year	Targeted symptom	Pre-post			Pre-follow-up		
		Hedges' <i>g</i>	95 % CI	<i>p</i>	Hedges' <i>g</i>	95 % CI	<i>p</i>
Neblett et al., 2010	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
	Pain	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive Disability	–	–	–	–	–	–
Newton-John et al., 1995	Muscle tension	0.835	0.628–1.043	<0.0001	–	–	–
	Pain	0.718	0.309–1.127	0.001	0.712	0.211–1.213	0.005
	Disability	0.756	0.343–1.170	<0.0001	0.020	–0.419–0.459	0.929
	Emot. Funct.	0.677	0.274–1.081	0.001	0.451	–0.014–0.916	0.057
	Cognitive	0.356	–0.016–0.729	0.061	0.255	–0.192–0.703	0.263
Nouwen and Solinger, 1979	Muscle tension	–	–	–	–	–	–
	Pain	0.613	0.246–0.979	0.001	0.382	0.035–0.728	0.031
	Emot. Funct.	–	–	–	–	–	–
	Cognitive Disability	–	–	–	–	–	–
	Muscle tension	0.578	0.215–0.940	0.002	0.049	–0.285–0.382	0.776
Nouwen, 1983	Pain	0.150	–0.292–0.592	0.506	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive Disability	–	–	–	–	–	–
	Muscle tension	0.678	0.182–1.173	0.007	–	–	–
	Pain	0.530	0.202–0.858	0.002	–	–	–
de Sousa et al., 2009	Disability	0.806	0.451–1.161	<0.0001	–	–	–
	Emot. Funct.	0.206	–0.104–0.515	0.192	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
	Pain	0.277	–0.155–0.709	0.209	0.272	–0.195–0.739	0.254
Spence et al., 1995a	Disability	0.010	–0.413–0.432	0.964	0.143	–0.317–0.603	0.543
	Emot. Funct.	0.174	–0.252–0.600	0.424	0.255	–0.211–0.721	0.283
	Cognitive	0.329	–0.107–0.765	0.139	0.393	–0.085–0.871	0.107
	Muscle tension	–	–	–	–	–	–
	Pain	0.366	–0.057–0.789	0.090	0.589	0.125–1.052	0.013
Spence et al., 1995	Disability	0.098	–0.311–0.507	0.638	0.307	–0.127–0.741	0.166
	Emot. Funct.	0.207	–0.206–0.620	0.326	0.278	–0.154–0.710	0.207
	Cognitive	0.512	0.074–0.949	0.022	0.517	0.063–0.972	0.026
	Muscle tension	–	–	–	–	–	–
	Pain	0.000	–0.421–0.421	1.000	0.840	0.206–1.475	0.009
Strong et al., 1989	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
	Pain	0.322	–0.170–0.815	0.200	–	–	–
Stuckey et al., 1986	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	0.169	–0.312–0.650	0.491	–	–	–
	Pain	0.159	–0.194–0.512	0.377	0.132	–0.264–0.528	0.514
Vlaeyen et al., 1995	Disability	0.862	0.445–1.278	<0.0001	0.931	0.449–1.413	<0.0001
	Emot. Funct.	0.743	0.343–1.144	<0.0001	0.338	–0.068–0.745	0.103
	Cognitive	0.075	–0.277–0.426	0.677	0.086	–0.309–0.481	0.669
	Muscle tension	–	–	–	–	–	–

Pain Intensity

Hedges' *g* for pain intensity reduction was moderated by the quality of studies ($B = -0.028$, $SE = 0.008$, $p = 0.001$), with studies employing less rigorous methodology (i.e., lower validity scores) reporting greater effect sizes.

Depression

Hedges' *g* for depression was moderated by the proportion of biofeedback in the intervention ($B = 0.004$, $SE = 0.002$, $p = 0.05$), with studies employing higher proportions of biofeedback reporting greater effect sizes.

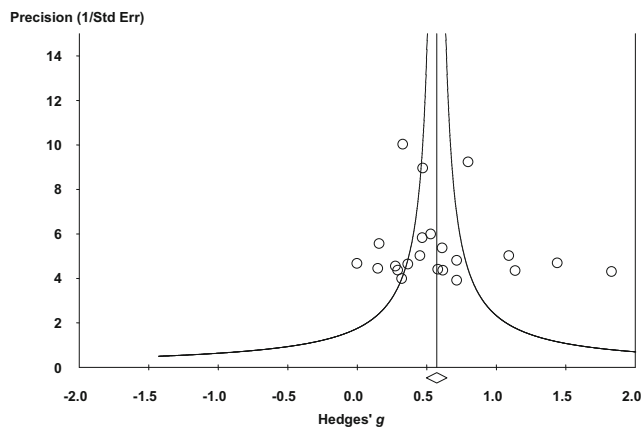


Fig. 2 Funnel plot of precision by Hedges' g for pre-post pain intensity measures

Cognitive Coping

Hedges' g for cognitive coping was moderated by the quality of studies ($B = -0.06$, $SE = 0.029$, $p = 0.031$), with studies employing less rigorous methodology reporting greater effect sizes.

Disability

Hedges' g for disability was moderated by the quality of studies ($B = -0.029$, $SE = 0.012$, $p = 0.016$), with studies with lower validity scores reporting greater effect sizes. The effect size for disability was moderated by dose of treatment ($B = 0.001$, $SE = 0.001$, $p = 0.004$), with studies employing larger dosages (more minutes of biofeedback training) reporting greater effect sizes.

Reduction of Muscle Tension (EMG)

Hedges' g for reduction of muscle tension (EMG) was moderated by the quality of studies ($B = -0.029$, $SE = 0.010$, $p = 0.003$), with studies with lower validity scores reporting greater effect sizes, and sample size ($B = 0.002$, $SE = 0.001$, $p < 0.05$), with studies with bigger sample sizes reporting greater effect sizes.

Sub-Analyses

Further analyses revealed that a pain intensity reduction of at least 30 % was reached in nine out of 22 studies (40.9 %) for

Table 4 Controlled effect sizes for all outcome measures

	k	Hedges' g	95 % CI	z	p	I^2	Fail-safe N (1/2tailed)
All controlled studies ^a							
Pain	17	0.380	0.219–0.541	4.630	<0.0001	0	132/88
Disability	12	0.171	-0.006–0.349	1.892	0.059	0	9/3
Emot. Funct.	11	0.390	0.180–0.600	3.636	<0.0001	14	66/44
Cognitive	9	0.380	0.169–0.591	-3.527	<0.0001	0	41/26
Muscle tension	6	0.707	0.442–0.972	5.232	<0.0001	0	52/35
Active control groups ^b							
Pain	11	0.362	0.171–0.552	3.720	<0.0001	6	56/36
Disability	7	0.133	-0.070–0.336	1.283	0.200	0	0/0
Emot. Funct.	6	0.260	0.042–0.478	2.337	0.019	0	9/5
Cognitive	4	0.269	0.014–0.525	-2.066	0.039	0	3/1
Muscle tension	5	0.694	0.416–0.971	4.896	<0.0001	0	37/25
CBT and education ^c							
Pain	7	0.439	0.182–0.708	3.198	0.001	23	29/18
Disability	4	0.202	-0.096–0.500	1.327	0.184	20	0/0
Emot. Funct.	4	0.220	-0.036–0.475	1.685	0.092	0	2/0
Cognitive	4	0.269	0.014–0.525	-2.066	0.039	0	3/1
Muscle tension	3	0.495	0.080–0.909	2.341	0.019	0	5/2
Waitlist control ^d							
Pain	6	0.465	0.127–0.804	2.692	0.007	0	11/6
Disability	5	0.297	-0.070–0.663	1.588	0.112	0	0/0
Emot. Funct.	5	0.690	0.242–1.138	3.017	0.003	29	20/13
Cognitive	5	0.619	0.244–0.994	-3.233	0.001	0	16/10
Muscle tension	1	0.839	-0.039–1.718	1.872	0.061	0	–

^a Emot. Funct. = emotional functioning; Cognitive = cognitive coping; Muscle tension = reduction of muscle tension (EMG); treatments that included biofeedback were tested against any control condition

^b Treatments that included biofeedback were tested only against active control groups

^c Treatments that included biofeedback were tested against CBT or interventions that used psychoeducation

^d Treatments that included biofeedback were tested against waitlist control conditions

treatments including at least 25 % biofeedback compared to two out of 18 studies (11.1 %) for control groups for pre-post data. At follow-up, the criterion of 30 % plus reduction was reached in seven out of 11 studies (63.3 %) for treatments with biofeedback and three out of eight studies (37.5 %) for control groups.

Correlational analyses were conducted to examine associations between effect sizes for the various outcome measures. For example, we examined the correlation between pain intensity reduction and reduction of muscle tension (EMG), as we anticipated a positive correlation, with lower EMG values associated with less pain. However, no significant correlations were found.

We also attempted to examine whether type of biofeedback treatment (EMG back vs. EMG front; EMG vs. respiration) was related to effect size. We hypothesized that a more specific biofeedback treatment (EMG back) would lead to more specific improvements in pain because it focuses on the region of the pain, while a different type of biofeedback (EMG front, respiration) would be expected to produce a more generalized relaxation. Unfortunately, no meaningful results can be reported because only a few studies used methods other than EMG-based biofeedback for the back, making the comparisons unbalanced.

Discussion

Summary of the Results

In this paper, we presented the results of a meta-analysis on the efficacy of biofeedback treatment for chronic back pain. The studies we analyzed included biofeedback training as at least 25 % of the intervention time and collected data on at least one of the following outcomes: pain intensity, disability, depression, cognitive coping, and reduction of muscle tension (EMG). First, within-participants analyses revealed significant, small-to-medium effect sizes for all reported outcomes at post-treatment. These results suggest that including biofeedback as one component of psychological or physiotherapeutic treatment seems to be helpful for chronic back pain patients on the outcomes highlighted by IMMPACT. Of note, analyses of publication bias suggest that these results can be considered robust. Secondly, findings were comparable when the subset of controlled studies was examined, with the exception of the effect size for pain reduction, which decreased from a medium effect size to a small effect size when including only controlled studies. Surprisingly, the effect sizes for all other outcomes remained relatively stable. Thirdly, the present results suggest that the effects of biofeedback treatment remained stable over long-term follow-up. The results suggest that cognitive behavioral therapy or physical therapy enhanced with biofeedback may lead to greater improvement in well-being compared to standard programs. Similarly, Hoffman and colleagues [20] demonstrated in their meta-analysis of psychological interventions for CLBP that psychological treatments

were “superior to wait-list control conditions in reducing pain ($p < 0.01$, $d = 0.48$)”, but different to the presented results, they could not show differences between CBT and self-regulatory treatments like biofeedback training for any outcome. The results of the current meta-analysis are in line with those of previous reviews (e.g., [11, 20]). Furthermore, Donaldson et al. [38] compared the effects of biofeedback training, relaxation training, and a psychoeducational group about CLBP on pain intensity and reduction of muscle tension (EMG). Their results showed that patients who received biofeedback training experienced an improvement in pain quality and intensity at follow-up compared to those who received relaxation training; effects of biofeedback did not significantly differ from effects of psychoeducation. These results suggest the importance of differentiating between relaxation and biofeedback training when examining effects of behavioral or respondent treatments. As the present meta-analysis used only studies that explicitly applied biofeedback training, our results suggest unique effects of (additional) biofeedback training, in line with Donaldson and colleagues’ study [38].

Overall, our meta-analysis of the existing literature suggests that biofeedback treatment, alone or in addition to other interventions, results in improvements in pain intensity, depression, cognitive coping, and reduction of muscle tension (EMG). Furthermore, moderator analyses indicated that longer biofeedback treatments were associated with greater reductions in pain-related disability. In addition, a greater proportion of biofeedback in the treatment was associated with larger effect sizes for reductions in depression.

However, the present results also show that despite high patient acceptance biofeedback treatment, the existing research is sparse. This is particularly true of methodologically rigorous studies, e.g., studies with control groups or studies assessing follow-up data. Despite the robust results, the results for long-term follow-up effects should be regarded with particular caution due to the small number of studies. In addition, due to the small numbers of studies and participants, it is not clear whether the statistically significant improvements also represent clinically significant improvements.

Strengths of the Current Study and Comparison with Previous Reviews

A strength of the present study is that we followed the methodological standards for conducting and reporting meta-analyses used by the Cochrane Reviews on chronic pain and recently recommended by QUORUM [10, 11, 25]. In addition, we adopted the IMMPACT criteria on outcome domains as encouraged by Morley et al. [39]. Previous reviews have focused on randomized controlled trials only (e.g., Cochrane Reviews; [40]); thus, some studies that were included in the present meta-analysis were omitted from previous reviews. To address this problem, we conducted moderator analyses, computed controlled effect sizes, and used sensitivity analyses to

test for publication bias. As a result, we were able to include these additional studies and also add to the literature by providing an up-to-date review. Given the sparse research on biofeedback treatment for chronic back pain, this approach appears to be important to provide more information about the efficacy of these interventions. Nevertheless, our results are comparable with previous findings. Morley and colleagues [40] also found small-to-medium effect sizes for biofeedback treatment compared to waitlist control conditions with respect to pain experience (as intensity, sensation, or unpleasantness), mood, and cognitive coping. In contrast to the present study, Morley and colleagues [40] reported on chronic pain in general, whereas the present analyses focused on chronic back pain patients.² Furthermore, in most previous studies, biofeedback was grouped as part of behavioral or respondent therapy along with interventions such as relaxation training, leading to difficulties in determining the specificity of the results to biofeedback treatment. Hoffman and colleagues [20] found medium-to-large effect sizes for self-regulatory treatments such as biofeedback or relaxation training for pain intensity ($d = 0.75$) and depression ($d = 0.81$) compared to a waitlist control group. Although this study showed self-regulatory treatments to be effective, the results rely on only three to four studies, respectively, and it is difficult to determine whether biofeedback, relaxation training, or another self-regulatory treatment accounted for the effects. As the current meta-analysis included only studies with biofeedback elements, a further strength of the meta-analysis is the higher specificity compared to previous reviews or meta-analyses.

Limitations

As observed by Hofmann and Smits [41], a limitation of meta-analyses in general is that the results are highly influenced by the selection of inclusion criteria, the quality of the studies included, and the outcome measures selected, in addition to the authors' expectations about the effects. We decided a priori to only include published studies. To obtain sufficient data, we selected relatively liberal inclusion criteria, resulting in heterogeneous study quality and some studies of unsatisfactory quality. Therefore, we computed the quality of the included studies using a validity rating scale based on modified Jadad criteria [31]. Our analyses revealed that study quality moderated the results for pain intensity, disability, and reduction of muscle tension (EMG). These findings underscore the importance of the quality of studies and suggest that the low quality of some of the included

studies represents a limitation of the present meta-analysis. Nevertheless, with the exception of disability, the results for all outcome variables remained significant when only controlled studies were included in the analysis.

Even though the outcome measures of this meta-analyses were followed by the IMMPACT recommendations, not all relevant outcome variables in the field of chronic back pain could have been assessed. For the core outcome domain "physical functioning," we chose outcome measures for disability. Besides disability, the construct of pain interference is an often investigated outcome in the field of chronic pain. Roughly, it describes the pain-related disruption with daily activities, but regarding a review by Wilson [29], the concept of pain interference "is not used or defined consistently or exclusively" (p. 500). Additionally, the measurement of pain interference includes a broad range of variables like quality of life with pain, pain-related task interference, pain disability and depression or functional disability. Maybe it is due to this assumption that the included studies mostly focused on reporting disability and only a few included pain interference as an outcome. Therefore, the reported data only focuses on disability as impairment of physical, psychosocial, and functional factors, but could also be seen as a kind of pain interference.

A further limitation of this meta-analysis is the heterogeneity of definitions of biofeedback and back pain in the studies, resulting in various combinations of biofeedback treatment and back pain not localized to a specific region. Thus, we are able to describe the general effect of biofeedback on back pain, but cannot make specific recommendations as to which biofeedback modality is best for which kind of back pain. We attempted to address this problem by comparing different biofeedback modalities, but results were not interpretable due to a small number of studies overall and highly unbalanced comparisons. Thus, one important finding of this meta-analysis is that there are only a few studies on the effectiveness of biofeedback treatment on chronic back pain, and only four of these studies [14, 21, 38, 42] were of high methodological quality. For effect sizes at follow-up after acute treatment, results should be considered preliminary for most of the outcomes, as only half of the studies reported follow-up data. The pool of studies with follow-up data was especially small for the outcome of reduction of muscle tension (EMG), with only three studies reporting follow-up data. To approach the problem of heterogeneity, we used the random-effects model for effect size analyses. There were no outliers, and our sensitivity analyses showed only small changes in effect sizes in both directions after adjustment for pre-post effect sizes.

² Our rationale for excluding studies on other pain syndromes such as fibromyalgia or headache was that these disorders show different symptom patterns, e.g., higher muscle tension in CLBP patients compared to fibromyalgia patients [59], and usually show different treatment effect sizes [60].

Clinical and Scientific Implications

Chronic back pain is often associated with depression, low cognitive coping (e.g., low self-efficacy expectations), high muscular tension, and disability in daily life (e.g., work absenteeism). The current results indicate that biofeedback treatment, whether as standalone treatment or as an additional feature in a psychological or physical therapy, can lead to improvements in pain intensity, muscular tension, emotional state, and cognitive coping among chronic back pain patients in the short term as well as in the long term. This is noteworthy, as previous analyses of long-term treatment effects for chronic back pain have been unable to demonstrate significant improvements. Hoffman et al. [20] found long-term treatment benefits for disability (e.g., return to work) for combined psychological and multidisciplinary treatments compared to an active control group, but did not find significant long-term effects on any other outcome variable, e.g., pain intensity. There are significant concerns about the long-term efficacy of some medication treatments for chronic back pain; as Martell et al. ([43], p. 123) observed in their meta-analysis, “opioids are commonly prescribed for but may only be efficacious for short-term treatment for chronic back pain (<16 weeks).” Given these concerns as well as high prevalence rates (up to 56 %) for side effects such as medication abuse or addiction, longer-term solutions are urgently needed. Another notable result of the present meta-analysis is that, consistent with Hofmann and colleagues’ [20] results, depression was reduced after acute treatment using biofeedback.

Clinicians should consider additional biofeedback treatment when treating patients with chronic back pain. Data could show that biofeedback is helpful in reducing a variety of pain-related symptoms. Thus, the low utilization of biofeedback as an intervention or therapy seems surprising. This discrepancy may be due to the fact that biofeedback not only requires expensive technology but also specific training to achieve satisfying effects. Biofeedback offers various possibilities for treatment, involving different sites, modalities (e.g., EMG, skin temperature, perspiration, heart rate), and procedures. Even if one modality is chosen, there is still a variety of dimensions to choose from, like “verbal instructions, focused attention, relaxation procedures, feedback, stress challenges, and motor skill learning” [38], p. 35]. Perhaps, this is a too high threshold for practitioners, especially against the background of the lack of clarity if the use of biofeedback justifies the additional expenses compared to more common CBT interventions. Patients and clinicians should keep in mind that the effects of biofeedback on various symptoms are small to medium. However, the possibility of long-term improvements suggests that it may be worthwhile for patients and clinicians to consider this treatment approach.

Unlike to literature about biofeedback treatments for headache which shows consistent findings that self-efficacy seems to be the main action of mechanism for biofeedback interventions, the data for chronic back pain is still unclear. The results for EMG-based measures for controlled studies show the greatest effect sizes in this meta-analysis. Therefore, it seems possible that for chronic back pain, another action of mechanism, e.g., reduction of muscle tension, is more important than self-efficacy. Further research should investigate in the action of mechanisms for biofeedback in chronic back pain using experimental designs and mediation analyses in treatment studies.

Scientifically, our study implies that more RCTs are needed in the field of biofeedback treatment for chronic back pain. Methodological quality of the studies was found to be a significant moderator for some outcome variables, e.g., pain intensity and reduction of muscle tension (EMG), indicating that better study quality resulted in smaller effects. RCTs are methodologically better performed and have higher data quality [44]; thus, more RCTs would allow for more confidence in the effects of biofeedback treatment. Additionally, an exact description of measures, participant flow, and procedure (which was missing in some of the included studies) should be regarded as essential for identifying important moderators or process variables. For example, Nestoriuc and Martin [16] found out that home training increased effects for biofeedback in migraine treatment up to 20 % compared to in-session biofeedback only. As the descriptions of the included studies for the current meta-analysis were often vague, we could not examine this variable as a moderator. The same problem applies to the exact back pain diagnosis. Researchers should be encouraged to provide detailed and accurate documentation of their studies on the basis of current standards. In addition, we recommend that further research on biofeedback treatment in chronic back pain, measure behavioral variables such as pain behavior or work absenteeism, to include another outcome dimension aside from questionnaires or self-ratings.

Conclusions

This is the first meta-analysis on the efficacy of biofeedback treatment for chronic back pain using the current standard recommendations to examine the following outcomes: pain intensity, reduction of muscle tension (EMG), depression, cognitive coping, and disability. The present results indicated that except for disability, (additional) biofeedback treatment led to improvements on all outcome measures in the short and long terms. Due to the sparse data and methodological flaws

of some of the included studies, these results should be regarded with caution, but suggest that biofeedback may be promising as a standalone or adjunctive intervention for chronic back pain.

Compliance with Ethical Standards

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Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval For this type of study, formal consent is not required.

References

*References marked with an asterisk indicate studies included in the meta-analysis

- Breivik H, Collett B, Ventafridda V, Cohen R, Gallacher D. Survey of chronic pain in Europe: prevalence, impact on daily life, and treatment. *Eur J Pain*. 2006;10(4):287–333. doi:10.1016/j.ejpain.2005.06.009.
- Bouhassira D, Lantéri-Minet M, Attal N, Laurent B, Touboul C. Prevalence of chronic pain with neuropathic characteristics in the general population. *Pain*. 2008;136(3):380–7. doi:10.1016/j.pain.2007.08.013.
- Johannes CB, Le TK, Zhou X, Johnston J a, Dworkin RH. The prevalence of chronic pain in United States adults: results of an Internet-based survey. *J Pain*. 2010;11(11):1230–9. doi:10.1016/j.jpain.2010.07.002.
- Andersson GB. Epidemiological features of chronic low-back pain. *Lancet*. 1999;354(9178):581–5. doi:10.1016/S0140-6736(99)01312-4.
- Hestbaek L, Leboeuf-Yde C, Kyvik KO, Manniche C. The course of low back pain from adolescence to adulthood: eight-year follow-up of 9600 twins. *Spine (Phila Pa 1976)*. 2006;31(4):468–72. doi:10.1097/01.brs.0000199958.04073.d9.
- Wolff R, Clar C, Lerch C, Kleijnen J. Epidemiology of chronic non-malignant pain in Germany. *Schmerz*. 2011;25(1):26–44. doi:10.1007/s00482-010-1011-2.
- Maniadakis N, Gray A. The economic burden of back pain in the UK. *Pain*. 2000;84(1):95–103. doi:10.1016/S0304-3959(99)00187-6.
- Pfingsten M, Schöps P, Wille T, Terp L, Hildebrandt J. Classification of chronic pain. Quantification and grading with the Mainz pain staging system. *Schmerz*. 2000;14(1):10–7. doi:10.1007/s004820000060.
- Scholic SL, Hallner D, Wittenberg RH, Hasenbring MI, Rusu AC. The relationship between pain, disability, quality of life and cognitive-behavioural factors in chronic back pain. *Disabil Rehabil*. 2012;34(23):1993–2000. doi:10.3109/09638288.2012.667187.
- Williams A, Eccleston C, Morley S. Psychological therapies for the management of chronic pain (excluding headache) in adults (review). *Cochrane Database Syst Rev*. 2012;11:CD007407. doi:10.1002/14651858.CD007407.pub3.
- Henschke N, Ostelo R, van Tulder M, et al. Behavioural treatment for chronic low-back pain (review). *Cochrane Database Syst Rev*. 2010;7:CD002014. doi:10.1002/14651858.CD002014.pub3.
- Schwartz NM, Schwartz MS. Definitions of biofeedback and applied psychophysiology. In: Schwartz MS, Andrasik F, editors. *Biofeedback: a practitioner's guide*, vol. 3. New York: Guilford Press; 2003. p. 27–42.
- Burns JW. Arousal of negative emotions and symptom-specific reactivity in chronic low back pain patients. *Emotion*. 2006;6(2):309–19. doi:10.1037/1528-3542.6.2.309.
- *Glombiewski JA, Hartwich-Tersek J, Rief W. Two psychological interventions are effective in severely disabled, chronic back pain patients: a randomised controlled trial. *Int J Behav Med*. 2010;17(2):97–107. doi:10.1007/s12529-009-9070-4.
- Jacobs JV, Henry SM, Jones SL, Hitt JR, Bunn JY. A history of low back pain associates with altered electromyographic activation patterns in response to perturbations of standing balance. *J Neurophysiol*. 2011;106(5):2506–14. doi:10.1152/jn.00296.2011.
- Nestoriuc Y, Martin A. Efficacy of biofeedback for migraine: a meta-analysis. *Pain*. 2007;128(1–2):111–27. doi:10.1016/j.pain.2006.09.007.
- Nestoriuc Y, Rief W, Martin A. Meta-analysis of biofeedback for tension-type headache: efficacy, specificity, and treatment moderators. *J Consult Clin Psychol*. 2008;76(3):379–96. doi:10.1037/0022-006X.76.3.379.
- Glombiewski JA, Bernady K, Häuser W. Efficacy of EMG- and EEG-biofeedback in fibromyalgia syndrome: a meta-analysis and a systematic review of randomized controlled trials. *Evid Based Complement Alternat Med*. 2013;2013. doi:10.1155/2013/962741.
- Hassett AL, Radvanski DC, Vaschillo EG, et al. A pilot study of the efficacy of heart rate variability (HRV) biofeedback in patients with fibromyalgia. *Appl Psychophysiol Biofeedback*. 2007;32(1):1–10. doi:10.1007/s10484-006-9028-0.
- Hoffman BM, Papas RK, Chatkoff DK, Kerns RD. Meta-analysis of psychological interventions for chronic low back pain. *Health Psychol*. 2007;26(1):1–9. doi:10.1037/0278-6133.26.1.1.
- *Flor H, Birbaumer N. Comparison of the efficacy of electromyographic biofeedback, cognitive-behavioral therapy, and conservative medical interventions in the treatment of chronic musculoskeletal pain. *J Consult Clin Psychol*. 1993;61(4):653–8. <http://psycnet.apa.org/journals/ccp/61/4/653/>.
- *Magnusson ML, Chow DH, Diamandopoulos Z, Pope MH. Motor control learning in chronic low back pain. *Spine (Phila Pa 1976)*. 2008;33(16):E532–8. doi:10.1097/BRS.0b013e31817df9a.
- *Nouwen A. EMG biofeedback used to reduce standing levels of paraspinal muscle tension in chronic low back pain. *Pain*. 1983;17(4):353–60. doi:10.1016/0304-3959(83)90166-5.
- *Stuckey SJ, Jacobs A, Goldfarb J. EMG biofeedback training, relaxation training, and placebo for the relief of chronic back pain. *Percept Mot Skills*. 1986;63(3):1023–36. doi:10.2466/pms.1986.63.3.1023.
- Liberati A, Altman DG, Tetzlaff J, et al. Annals of internal medicine academia and clinic the PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions. *Ann Intern Med*. 2009;151(4):W65–94. doi:10.1371/journal.pmed.1000100.
- Turk DC, Dworkin RH, Allen RR, et al. Core outcome domains for chronic pain clinical trials: IMMPACT recommendations. *Pain*. 2003;106(3):337–45. doi:10.1016/j.pain.2003.08.001.
- Dworkin RH, Turk DC, Farrar JT, et al. Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. *Pain*. 2005;113:9–19.
- Turk DC, Dworkin RH, McDermott MP, et al. Analyzing multiple endpoints in clinical trials of pain treatments: IMMPACT recommendations. *Pain*. 2008;139(3):485–93. doi:10.1016/j.pain.2008.06.025.

29. Wilson M. Integrating the concept of pain interference into pain management. *Pain Manag Nurs*. 2014;15(2):499–505. doi:10.1016/j.pmn.2011.06.004.
30. Glass GV. Primary, secondary, and meta-analysis of research. *Educ Res*. 1976;5(10):3–8. doi:10.3102/0013189X005010003.
31. Jadad AR, Moore RA, Carroll D, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials*. 1996;17(1):1–12. doi:10.1016/0197-2456(95)00134-4.
32. Borenstein M, Hedges L V., Higgins JPT, Rothstein HR. *Comprehensive meta-analysis version*. 2006.
33. Hedges LV, Olkin I. Nonparametric estimators of effect size in meta-analysis. *Psychol Bull*. 1984;96(3):573–80. doi:10.1037/0033-2909.96.3.573.
34. Cohen J. *Statistical power analysis for the behavioral sciences*, 2nd edn. Vol 2nd. 1988. doi:10.1234/12345678.
35. Rosenthal R. *Meta-analytic procedures for social research* (rev. Ed.). 1991.
36. Hedges LV, Vevea JL. Fixed- and random-effects models in meta-analysis. *Psychol Methods*. 1998;3(4):486–504. doi:10.1037/1082-989X.3.4.486.
37. Moses LE, Mosteller F, Buehler JH. Comparing results of large clinical trials to those of meta-analyses. *Stat Med*. 2002;21(6):793–800.
38. *Donaldson S, Romney D, Donaldson M, Skubick D. Randomized study of the application of single motor unit biofeedback training to chronic low back pain. *J Occup Rehabil*. 1994;4(1):23–37. doi:10.1007/BF02109994.
39. Morley S, Williams A, Eccleston C. Examining the evidence about psychological treatments for chronic pain: time for a paradigm shift? *Pain*. 2013;154(10):1929–31. doi:10.1016/j.pain.2013.05.049.
40. Morley S, Eccleston C, Williams A. Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain*. 1999;80(1–2):1–13. doi:10.1016/S0304-3959(98)00255-3.
41. Hofmann SG, Smits JAJ. Pitfalls of meta-analyses. *J Nerv Ment Dis*. 2008;196:716–717.
42. *Kapitza KP, Passie T, Bernateck M, Karst M. First non-contingent respiratory biofeedback placebo versus contingent biofeedback in patients with chronic low back pain: a randomized, controlled, double-blind trial. *Appl Psychophysiol Biofeedback* 2010;35(3):207–17. doi:10.1007/s10484-010-9130-1.
43. Martell BA, O'Connor PG, Kems RD, et al. Systematic review: opioid treatment for chronic back pain: prevalence, efficacy, and association with addiction. *Ann Intern Med*. 2007;146(2):116–27. doi:10.7326/0003-4819-146-2-200701160-00006.
44. Guyatt G, Oxman AD, Akl EA, et al. GRADE guidelines: 1. Introduction - GRADE evidence profiles and summary of findings tables. *J Clin Epidemiol*. 2011;64(4):383–94. doi:10.1016/j.jclinepi.2010.04.026.
45. *Adams J, Pearson SJ, Olson N. Innovative cross-modal technique of pain intensity assessment with lower back pain patients given biofeedback training. *Am J Clin Biofeedback* 1982;5(1):25–30.
46. *Asfour SS, Khalil TM, Waly SM, Goldberg ML, Rosomoff RS, Rosomoff HL. Biofeedback in back muscle strengthening. *Spine (Phila Pa 1976)*. 1990;15(6):510–13.
47. *Hallman DM, Olsson EMG, von Schéele B, Melin L, Lyskov E. Effects of heart rate variability biofeedback in subjects with stress-related chronic neck pain: a pilot study. *Appl Psychophysiol Biofeedback* 2011;36(2):71–80. doi:10.1007/s10484-011-9147-0.
48. *Huis in't Veld, RMH, Kosterink SM, Barbe T, Lindegård A, Marecek T, Vollenbroek-Hutten MMR. Relation between patient satisfaction, compliance and the clinical benefit of a teletreatment application for chronic pain. *J Telemed Telecare*. 2010;16(6):322–8. doi:10.1258/jtt.2010.006006.
49. *Keefe FJ, Block AR, Williams RB, Surwit RS. Behavioral treatment of chronic low back pain: clinical outcome and individual differences in pain relief. *Pain* 1981;11(2):221–31.
50. *Kröner-Herwig B, Beck A. An exploratory study of biofeedback for chronic low back pain. *Br J Ther Rehabil*. 2000;7:134–42.
51. *McLaughlin L, Goldsmith CH, Coleman K. Breathing evaluation and retraining as an adjunct to manual therapy. *Man Ther*. 2011;16(1):51–52. doi:10.1016/j.math.2010.08.006.
52. *Neblett R, Mayer TG, Brede E, Gatchel RJ. Correcting abnormal flexion-relaxation in chronic lumbar pain: responsiveness to a new biofeedback training protocol. *Clin J Pain*. 2010;26(5):403–9.
53. *Newton-John TR, Spence SH, Schotte D, Wing C, Mary S, Street P. Cognitive-behavioural therapy versus EMG biofeedback in the treatment of chronic low back pain. *Behav Res Ther*. 1995;33(6):691–7. doi:10.1016/0005-7967(95)00008-L.
54. *Nouwen A, Solinger JW. The effectiveness of EMG biofeedback training in low back pain. *Biofeedback Self Regul*. 1979;4(2):103–11.
55. *Santaella da Fonseca Lopes de Sousa K, Garcia Orfale A, Mara Meireles S, Roberto Leite J, Natour J. Assessment of a biofeedback program to treat chronic low back pain. *J Musculoskelet Pain* 2009;17(4):369–77. doi:10.3109/10582450903284828.
56. *Spence SH, Sharpe L, Newton-John T, Champion D. Effect of EMG biofeedback compared to applied relaxation training with chronic, upper extremity cumulative trauma disorders. *Pain* 1995;63(2):199–206. doi:10.1016/0304-3959(95)00047-V.
57. *Strong J, Cramond T, Maas F. The effectiveness of relaxation techniques with patients who have chronic low back pain. *Occup Ther J Res*. 1989;9(3):184–92.
58. *Vlaeyen JW, Haazen IW, Schuerman JA, Kole-Snijders AM, van Eek H. Behavioural rehabilitation of chronic low back pain: comparison of an operant treatment, an operant-cognitive treatment and an operant-respondent treatment. *Br J Clin Psychol*. 1995;34(Pt 1):95–118.
59. Thieme K, Rose U, Pinkpank T, Spies C, Turk DC, Flor H. Psychophysiological responses in patients with fibromyalgia syndrome. *J Psychosom Res*. 2006;61(5):671–9. doi:10.1016/j.jpsychores.2006.07.004.
60. Malone MD, Strube MJ. Meta-analysis of non-medical treatments for chronic pain. *Pain*. 1988;34(3):231–44. doi:10.1016/0304-3959(88)90118-2.

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