



# The effects of a novel psychological attribution and emotional awareness and expression therapy for chronic musculoskeletal pain: A preliminary, uncontrolled trial



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## ABSTRACT

**Objective:** Current psychological and behavioral therapies for chronic musculoskeletal pain only modestly reduce pain, disability, and distress. These limited effects may be due to the failure of current therapies: a) to help patients learn that their pain is influenced primarily by central nervous system psychological processes; and b) to enhance awareness and expression of emotions related to psychological trauma or conflict.

**Methods:** We developed and conducted a preliminary, uncontrolled test of a novel psychological attribution and emotional awareness and expression therapy that involves an initial individual consultation followed by 4 group sessions. A series of 72 patients with chronic musculoskeletal pain had the intervention and were assessed at baseline, post-treatment, and 6-month follow-up.

**Results:** Participation and satisfaction were high and attrition was low. Intent-to-treat analyses found significant improvements in hypothesized change processes: psychological attributions for pain, emotional awareness, emotional approach coping, and alexithymia. Pain, interference, depression, and distress showed large effect size improvements at post-treatment, which were maintained or even enhanced at 6 months. Approximately two-thirds of the patients improved at least 30% in pain and other outcomes, and one-third of the patients improved 70%. Changes in attribution and emotional processes predicted outcomes. Higher baseline depressive symptoms predicted greater improvements, and outcomes were comparable for patients with widespread vs. localized pain.

**Conclusion:** This novel intervention may lead to greater benefits than available psychological interventions for patients with chronic musculoskeletal pain, but needs controlled testing.

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## Introduction

Chronic or persistent musculoskeletal pain is highly prevalent, a major source of morbidity, and a leading contributor to health care expenditures [1]. Many patients have pain that is localized to one or several bodily regions, such as the back, arms, neck, or legs; whereas others have chronic widespread pain (axial and in all four bodily quadrants) and are frequently diagnosed with fibromyalgia, which is estimated to occur in 2–4% of adults [2]. Medical treatments for musculoskeletal pain, such as opiates and other medications, spinal injections, and surgery, have reached staggering rates, but these interventions often have limited efficacy or troubling side effects or risks [3–5].

Psychological and behavioral interventions for chronic pain have been studied over the last several decades. The most popular treatment

program, cognitive behavior therapy (CBT), teaches patients that their pain should be viewed as a chronic condition that will not be cured medically but can be successfully self-managed using a variety of skills including relaxation training, goal setting, activity pacing, environmental changes, attention management, cognitive restructuring, behavioral experiments, and problem solving [6]. CBT has been widely tested in clinical trials, and meta-analytic reviews typically conclude that CBT is efficacious for a range of pain-related outcomes [7–9]. Yet, the actual effect sizes obtained indicate that the benefits of CBT are quite modest. Compared to no treatment (or treatment as usual), the effect of CBT ranges from about 0.25 to 0.50 standard deviations (“small to medium” effects), tend to weaken over time, and only a minority of patients show clinically significant improvement, such as a 30% reduction in pain. More recent approaches for chronic pain, such as mindfulness and acceptance-based interventions [10], have some conceptual overlap with CBT including a focus on accepting the chronic nature of the pain, but emphasize awareness of present experience beyond pain and engaging in value-based activities despite pain. Recent tests of these

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approaches suggest that they also surpass treatment as usual but appear no more beneficial than CBT [11–13].

Several changes in the field's conceptualization and treatment of chronic pain may lead to more powerful outcomes than provided by current approaches. First, current treatment models rarely differentiate among types of chronic pain and advocate the management of pain, regardless of its origin. Although the central nervous system (CNS) plays a role in all chronic pain, peripheral nociceptive afferent processes play the key role in some types of pain, such as that from joint degeneration (e.g., osteoarthritis), autoimmune disease (e.g., rheumatoid arthritis), genetic anomalies (e.g., sickle cell disease), neuropathy, or tumors. Individuals with these types of pain may benefit most from learning to manage, adapt to, or accept their pain. However, the CNS appears to be more important than peripheral nociception in other pain conditions, particularly those considered central sensitization or augmentation syndromes [2], such as fibromyalgia, pelvic pain, abdominal pain, some types of head pain, and some musculoskeletal pain conditions. Psychological stress or trauma, emotion dysregulation, interpersonal conflict, and learning processes such as conditioning, expectations, and attributions appear to play a primary role in predisposing, precipitating, and perpetuating pain. This is hypothesized to occur by creating, activating, and maintaining neural pain pathways [14], and because patients with these types of pain do not have significant peripheral or structural pathology, it may be possible to reduce or eliminate their pain, as opposed to manage it.

A second limitation of CBT and other current pain management approaches is that they do not directly address psychological trauma, victimization, or serious emotional and relational conflict even though such problems are at substantially elevated levels in patients with central sensitization pain conditions [15–21]. We acknowledge that acceptance and mindfulness-based pain management approaches can facilitate the experiencing of negative emotions, which may or may not impact on trauma and conflict, and that pain exposure therapies attempt to reverse behavioral avoidance of pain, which may activate emotional processes. Furthermore, some settings (e.g., the U.S. Veteran's Affairs system) separately treat post-traumatic stress disorder and teach pain management in different clinics. Yet current pain management or acceptance protocols do not directly target trauma and emotional and relational conflict through exposure and processing techniques; rather, techniques such as cognitive reappraisal or defusion, arousal reduction (e.g., relaxation), engaging in pleasant activities, and acceptance of current experience are used to attenuate negative emotions. However, a wealth of research indicates that a lack of awareness and expression of adaptive or primary emotions contribute to the presence and intensity of chronic pain, especially pain associated with central sensitization syndromes, whereas emotional awareness, expression, and processing, which usually involve exposure to avoided emotions and memories, are key mechanisms in reducing symptoms [22–26]. Indeed, focused and directive emotional processing approaches, such as emotion-oriented intensive psychodynamic therapy [22,27] and emotional disclosure (expressive writing) about stress [28], have been found to improve some somatic conditions, including musculoskeletal pain.

The reports of Sarno [29] and others [30] as well as the “explaining pain” model of Moseley and Butler [31] suggest that, at least for central sensitization types of pain, patients may benefit from an explanatory model that emphasizes that their pain is largely under the control of the CNS and learning experiences, and that one can change emotional and relational processes, potentially leading to pain remission or elimination. Thus, we developed and tested a treatment program for such patients with chronic musculoskeletal pain conditions. Our approach emphasizes re-attributing the primary source of pain to neural pathways rather than bodily injury or disease; understanding that pain is a signal of learned, usually emotional processes; recognizing that control over painful symptoms can be achieved through the power of the mind; engaging in emotional awareness and expression exercises; and re-

engaging in a full range of life activities to “unlearn” the pain. The intervention consists of an individual consultation for each patient followed by 4 sessions of group therapy.

We conducted an uncontrolled trial of this novel intervention on a series of patients seeking treatment for central sensitization-based chronic musculoskeletal pain. We hypothesized that the intervention would effectively reduce pain intensity (primary outcome) as well as interference, depression, and distress over 6 months. We also hypothesized that the treatment would change key theoretical processes: increasing patients' psychological attributions of pain, emotional awareness, and emotional approach coping, and decreasing alexithymia; and we hypothesized that improvements in these processes would be associated with improvements in pain-related outcomes. Finally, patients with emotional disturbances such as depression often show less benefit from CBT approaches than patients with less depression [32–34], and reviews indicate that patients with widespread pain or fibromyalgia achieve little or no significant pain reduction from CBT and mindfulness-based approaches [7,35]. Thus, we tested whether this novel therapy would be successful for patients with elevated depression and widespread pain, compared to less depressed patients and those with more localized pain.

## Method

### Participants

Participants were adults reporting musculoskeletal pain for at least 3 months who consulted an internal medicine physician (HS) at a hospital-based mind-body clinic. Patients were excluded if the physician's interview and history, review of medical records, or physical examination revealed that patients: a) had an autoimmune or other disease or structural pathological process that typically generates pain (e.g., rheumatoid arthritis, inflammatory bowel disease, systemic lupus, sickle cell disease, cancer); b) had serious mental illness or cognitive impairment; c) were suicidal or homicidal; or d) were non-literate in English. Also, patients who had the consultation but did not start the group phase of the treatment (described below) were excluded.

### Procedure

The trial was registered with [ClinicalTrials.gov](http://ClinicalTrials.gov) (NCT00861302). The study was a research evaluation of a clinical “mind-body” program, and patients were enrolled into the program from November 2008 through March 2011, and follow-up assessments were completed by October 2011. Self- or clinician-referred patients who contacted the clinic were sent routine “pre-consultation” baseline self-report measures of pain intensity, pain interference, and depression to be completed and brought to the consultation (described below). The consulting physician (HS) provided the independent research team with the contact information of each patient who finished the consultation and planned to enroll in the subsequent group phase of the program, and a researcher telephoned patients and requested their participation in an evaluation of the program. Interested patients were met in person at the clinic by a researcher before the group sessions started, provided written informed consent, and completed the “pre-group” baseline assessment, which included additional program evaluation outcome measures of pain and distress as well as process measures of pain attribution, emotional awareness, emotional approach coping, and alexithymia. Patients then participated in the group treatment (described below), after which they completed all measures (post-treatment). Patients then completed only the outcome measures 6 months after treatment. (An additional assessment was conducted 3 months after treatment, but the results for 3 and 6 months were nearly identical, so for simplicity of presentation, we give only the 6-month follow-up data.)

### Intervention program

The program consisted of an individual consultation followed by a 4-session group treatment. Both the consultation and group sessions were guided by a manual [36] and conducted by an internal medicine physician (HS) with specialized training in mind-body medicine.

The consultation occurred individually with each patient for 90–12 min and investigated the patient's medical and psychosocial history and identified linkages between life stressors, emotional processes, and the onset and exacerbation of symptoms including pain. A physical examination and review of imaging and laboratory studies, if available, were conducted to rule out specific peripheral structural causes for pain and to prompt discussion about the role of peripheral pathology versus CNS processes in pain. The physician then reviewed all findings with the patient and explained the medical decision-making process used to differentiate CNS-generated pain from peripheral pathology. Specific abnormal findings on MRI examinations were not considered to be causative of pain unless there were physical examination abnormalities, such as muscle weakness or alteration of deep tendon reflexes, to support them. The physician then discussed the program's model of chronic musculoskeletal pain, which holds that such pain is a “learned” mind-body syndrome. Emotionally difficult experiences during development as well as other learning processes (e.g., classical conditioning, modeling) create CNS pathways that are directly linked to pain pathways. Later in life, stress, exposure to eliciting stimuli, and fearful beliefs that the pain implies bodily damage then generate or amplify pain. Patients were then offered the group portion of the program and informed that a researcher would contact them and invite their participation in a program evaluation study.

The group portion occurred several weeks after the consultation, whenever a group of 6 to 10 patients could be formed. Patients attended four, 2-hour group sessions held at 1-week intervals. All patients were provided the manual [36], which guided them through the four sessions and included in-session exercises and homework. The curriculum consisted of four components: a) education about the mind-body neural pathway model of chronic pain; b) emotion awareness techniques; c) various expressive writing (emotional disclosure) exercises concerning past and current life stressors; and d) re-engagement in previously avoided activities. Education about the model, which had been introduced in the consultation, included research and case studies documenting the role of CNS processes in chronic pain, and patients were taught that their pain should be reinterpreted as a signal that emotional or other learning processes are activated. Emotion awareness techniques consisted of daily audio-recorded exercises that encouraged awareness of one's breath, body, and emotions; non-judgmental awareness of these emotions; and affirmations of self-acceptance and self-healing. Expressive writing was conducted in session and then as daily homework and consisted of writing about stress and emotions in free-writing prose, unsent letters, and imagined dialogues. Re-engagement in activity consisted of encouraging patients to remind themselves that their pain was central rather than peripheral and instructions to engage in physical activities while repeating positive affirmations about the health of their bodies. Patients were encouraged not to allow pain to dissuade them from engaging in important physical and relational experiences. Note that changes in pain medication or other treatments were not discussed or prescribed as part of the individual consultation or group program. After the fourth group session (and before the post-treatment assessment), the physician contacted each participant briefly by telephone to address any remaining concerns and encourage further practice.

### Process measures

To track hypothesized change processes, four measures were completed by patients at the “pre-group” baseline (after the initial consultation) and then again only at post-treatment.

Psychological attribution for pain was assessed with a set of 6 items that we created or modified from various scales. Items were rated from 0 (not at all) to 4 (completely) and included, “How much do you think that psychological factors such as stress or emotions cause your pain?” and “How much do you think that the source or cause of your pain is in your mind?” The six items were internally consistent ( $\alpha = .88$ ). Item ratings were averaged, and higher scores indicate a stronger attribution that pain is due to psychological factors.

Emotional awareness was assessed with the Levels of Emotional Awareness Scale (LEAS) [37], which is a performance-based measure of one's ability to generate specific and integrated emotional language in response to provocative scenarios. We used the two equivalent 10-scenario forms of the LEAS (Form A at pre-group baseline and B at post-treatment) to minimize practice or carry-over effects. Two trained, independent raters coded the patients' written responses according to the scoring manual, yielding values for “self,” “other,” and “total”; higher sums indicate greater emotional awareness. Exact agreement on individual scores between the two raters for a randomly selected 20% of the protocols was 93.3%, and no difference was greater than 1 point.

Emotional approach coping was assessed with the 8-item Emotional Approach Coping (EAC) Scale [38], which assesses both emotional processing (understanding, validating, and acknowledging one's emotions) and expression (valuing the expression of one's feelings). The scale was internally consistent (baseline  $\alpha = .90$ ). Items were rated from 1 to 4 and averaged; higher scores indicate greater emotional processing and expression.

Alexithymia was assessed with the Toronto Alexithymia Scale-20 (TAS-20) [39], a self-report scale that assesses people's difficulty identifying feelings, describing feelings, and a preference for externally-oriented thinking. This scale was internally consistent (baseline  $\alpha = .86$ ). Items were rated from 1 (strongly disagree) to 5 (strongly agree) and summed; higher scores indicated greater alexithymia.

### Pain-related outcome measures

We assessed the three major domains of adjustment in chronic pain: pain severity, pain interference, and psychological distress. For these measures, there were two different baseline assessments: patients completed the first two measures (three variables) listed below before the consultation (“pre-consultation”), and the last two measures (three variables) before the group sessions (“pre-group”). All four outcome measures were completed by patients at the post-treatment and follow-up time points. The study's pre-specified primary outcome was pain intensity, and the other variables were secondary outcomes.

Pain intensity and pain interference were assessed with the Brief Pain Inventory (BPI) [40], which includes four items, rated 0 to 10, for current pain and worst, least, and average pain over the past week. These ratings were internally consistent (baseline  $\alpha = .88$ ) and were averaged to yield a single pain intensity rating. The BPI also includes a set of items, rated 0 to 10, for how much the patient's pain interfered with activity, mood, mobility, work, sleep, and other functions during the past week. These items were internally consistent (baseline  $\alpha = .89$ ) and were averaged to yield a pain interference score.

Depressive symptoms during the past week were assessed with the 20-item Center for Epidemiologic Studies Depression scale (CESD) [41]. Items (rated 0 to 3) were summed to yield a total score. This measure was internally consistent (baseline  $\alpha = .90$ ), and scores of 16 or greater suggest clinically significant depressive symptoms.

Sensory and affective dimensions of current pain were assessed with the McGill Pain Questionnaire Short-Form (MPQ-SF) [42], which presents 11 sensory and 4 affective pain items rated from 0 (none) to 3 (severe). Both subscales were internally consistent (baseline  $\alpha = .83$  and  $.76$ ), and ratings were summed to yield sensory and affective pain scores.

Psychological distress during the past week was assessed with the 53-item Brief Symptom Inventory (BSI) [43], which assesses various symptoms, including depression, anxiety, hostility, and interpersonal sensitivity. Items were rated from 0 (not at all) to 4 (extremely), and we analyzed the global severity index (sum of all items), which is considered a measure of general psychological distress and was internally consistent in this sample (baseline  $\alpha = .95$ ).

### Data analyses

A power analysis based on a pre-post test of paired means indicated that a sample size of 67 patients would provide power of .80, using a 2-tailed alpha of .05, to detect a small to medium effect size (0.35 SD) change in the primary outcome, pain intensity, from baseline to post-treatment. Thus, we sought to enroll at least 70 patients. For the main analyses, paired samples t-tests examined whether the process measures changed significantly from baseline (pre-group assessment) to post-treatment, and whether the outcome measures changed from baseline to post-treatment and 6-month follow-up. Next, correlations examined whether the change in process measures (post-treatment minus baseline) predicted the change in outcomes (post-treatment and follow-up minus baseline). Finally, correlations and t-tests examined whether pre-consultation depressive symptoms and the diagnosis of widespread pain predicted change in outcomes.

In addition to statistical significance, we calculated effect sizes (ES) in two ways. At post-treatment and follow-up, ES was calculated with this formula:  $(\text{post } M - \text{baseline } M) / \text{baseline } SD$ . To determine the frequency of individual cases improving, we used the standard metric of 30% improvement from an individual's baseline score, as well as the moderately stringent criterion of 50% improvement, and the very stringent criterion of 70% improvement. Because some post-treatment and follow-up values were missing due to attrition, we used a conservative estimation approach (i.e., leading to lower effect size estimates) by assuming no change from the previous assessment; we replaced missing follow-up data with the last available data point, which was often the baseline value.

## Results

### Sample descriptive data and attrition

Table 1 presents baseline descriptive data on the sample of 72 patients in the trial. As can be seen, the sample averaged 49.3 years of age and was predominantly female, European American, married/partnered, and well-educated. The majority of the sample was depressed, reported moderate to high pain during the past week, and had pain in multiple locations (74% had back pain), including 26 who we classified with widespread pain (13 of whom reported a prior diagnosis of fibromyalgia). The MRIs of almost all patients with neck or back pain demonstrated abnormalities such as bulging or herniated discs, spinal stenosis, or degenerative disc disease; however, no patients were excluded due to those findings. None of the included patients had evidence of muscle weakness, loss of sensation, or alteration of deep tendon reflexes.

Fig. 1 shows the flow of patients through the trial. A total of 91 patients had the initial consultation, met study criteria, and were invited to participate in the program evaluation; 19 (21%) declined. Of the remaining 72 enrolled patients, most (81%) attended all four group sessions; 11% attended three sessions, and 8% attended only one or two sessions. Only four patients dropped from treatment and discontinued the study; thus, outcome data were provided by the other 68 patients (94%). A total of 8 patients did not provide 6-month assessment data, and these 8 patients were more educated ( $p = .05$ ) and tended to be less depressed (CESD:  $p = .052$ ) and less distressed (BSI:  $p = .062$ ) at baseline than the 64 patients who completed the 6-month assessment.

**Table 1**  
Sample descriptive data at baseline.

	n (%)	Mean (SD) range
Age		49.3 (15.6) 18–84
Gender		
Female	57 (79.2%)	
Male	15 (20.8%)	
Education		
High school graduate	10 (13.9%)	
Some college	13 (18.1%)	
College graduate	20 (27.8%)	
Graduate degree	29 (40.3%)	
Ethnicity		
European American	66 (91.7%)	
African American	4 (5.6%)	
Other	2 (2.8%)	
Marital status		
Married/partnered	45 (62.5%)	
Separated/divorced/widowed	13 (18.1%)	
Never married	14 (19.4%)	
Employment status		
Full-time	27 (37.5%)	
Part-time	11 (15.3%)	
Retired	12 (16.7%)	
Unemployed	14 (19.4%)	
Disabled	8 (11.1%)	
Depressed (CES-D $\geq 16$ )	53 (73.6%)	
Pain duration (years)		8.7 (9.1) 0.5–40
BPI worst pain last week (0–10)		7.5 (1.8) 2–10
Number of pain locations		
Localized	6 (8.3%)	
Multiple	40 (55.6%)	
Widespread pain	26 (36.1%)	
Medications at intake		
Opioids	17 (24.6%)	
Antidepressants	27 (39.1%)	
Anticonvulsants	17 (24.6%)	
Anxiolytics/sedatives	22 (31.9%)	
Muscle relaxants	2 (2.9%)	
NSAIDs/acetaminophen	18 (26.1%)	

### Change in pain attribution and emotional processes

As shown in Table 2 and as hypothesized, all four process measures changed significantly from before the group sessions to post-treatment. Psychological attribution for pain increased (medium effect). Emotional awareness on the LEAS also increased significantly, with a small effect for the total LEAS, but medium effect ( $ES = 0.48$ ) for the LEAS “other” component. Emotional approach coping significantly increased (small effect), and alexithymia significantly decreased (small to medium effect) over the group sessions.

### Changes in pain-related outcomes

Table 3 presents data on outcomes along with change scores and effect sizes. The top three variables show change from prior to the consultation, whereas the bottom three variables show change from prior to the group sessions, but after the consultation.

Pre-consultation pain intensity, pain interference, and depressive symptoms all decreased substantially to the end of treatment, with effect sizes of about 1.0 to 1.2 SD, and these effects were not only maintained but increased slightly after 6 months. Nearly two-thirds of the patients showed a 30% or greater reduction in pain after treatment and at 6 months, and about one-third met the very high criterion of 70% reduction in pain and depression at 6 months, and just below half showed 70% reduction in pain interference. Regarding “end-state functioning, that is, the absolute level of mean pain intensity ratings on the four 0 to 10-point BPI scales, only four patients (5.6%) had a BPI pain rating of 2.0 or less at baseline (pre-consultation), but at post-treatment, 30 patients (41.7%) had pain of 2.0 or less, and at 6-month follow-up, 36 patients (50.0%) reached this level.



## Flow of Patients through the Clinical Trial

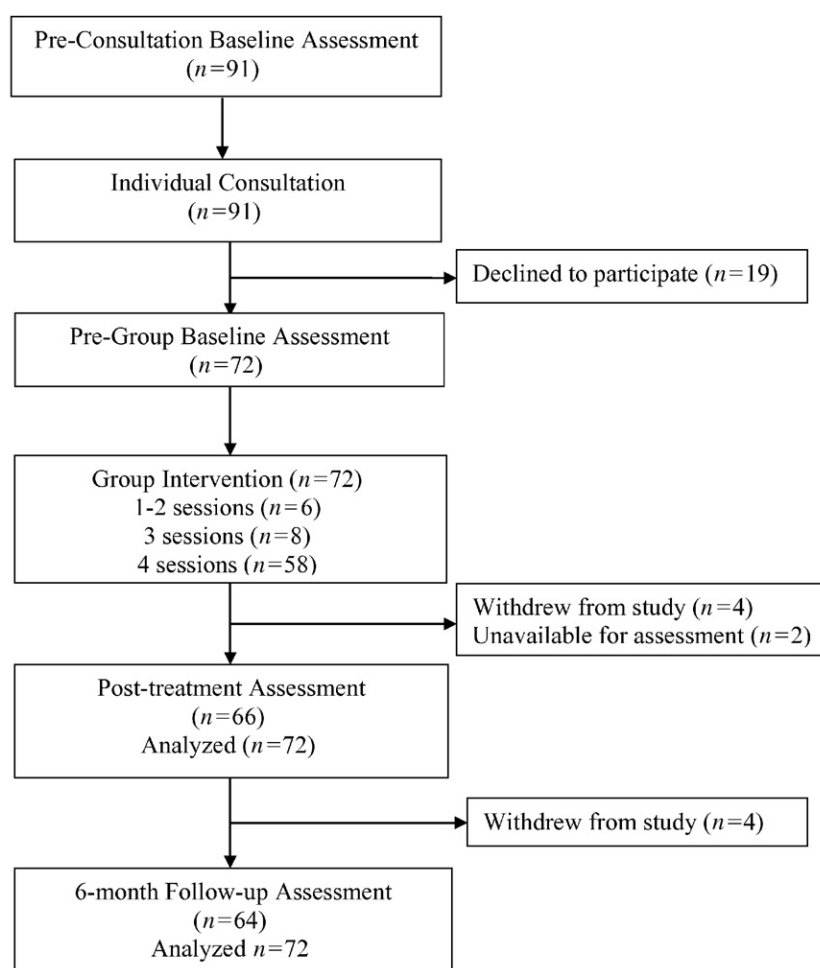


Fig. 1. Flow of patients through the clinical trial.

Pre-group sensory and affective pain and general distress all showed significant improvement to post-treatment, with effect sizes ranging from medium for sensory and affective pain to medium-large for

general distress; these improvements were maintained to 6 months. Two-thirds or more the patients showed 30% improvement on one or more of these outcomes at 6 months, and up to half of the patients improved at least 70% on affective pain at 6 months.

The 66 patients who provided post-treatment data rated their overall satisfaction with the program: 65.2% were “very much” satisfied, 25.8% were “quite a lot” satisfied, and 9.1% were “moderately” satisfied. None reported being less satisfied than “moderately.”

Table 2

Changes in treatment process measures over the course of the group sessions (N = 72).

Process measure	Mean (SD)	Change	t	Effect size
Psychological attribution				
Pre-group	2.60 (0.78)			
Post-treatment	2.98 (0.80)	0.38	4.04***	0.49
LEAS -total				
Pre-group	31.33 (5.16)			
Post-treatment	32.86 (4.66)	1.53	3.20**	0.30
LEAS -self				
Pre-group	28.24 (4.87)			
Post-treatment	29.90 (4.71)	1.67	3.40**	0.34
LEAS -other				
Pre-group	23.26 (6.08)			
Post-treatment	26.19 (5.07)	2.92	4.56***	0.48
EAC				
Pre-group	2.62 (0.76)			
Post-treatment	2.83 (0.75)	0.21	3.64***	0.28
TAS-20				
Pre-group	50.81 (12.25)			
Post-treatment	45.95 (13.13)	-4.85	-4.66***	-0.40

LEAS = Levels of Emotional Awareness Scale; EAC = Emotional Approach Coping Scale; TAS-20 = Toronto Alexithymia Scale-20.

\*\*  $p < .01$ .

\*\*\*  $p < .001$ .

## Relationships of process to outcome measures

Changes (post-treatment minus pre-group baseline) in the four process measures were correlated with changes in the outcomes measures. Consistent with hypotheses, increases in psychological attributions for pain correlated with decreases in BPI pain intensity, interference, and McGill sensory pain at post-treatment ( $r = -.27, r = -.26, r = -.30$ , respectively, all  $p < .05$ ) and 6 months ( $r = -.28, r = -.24, r = -.38$ , all  $p < .05$ ) and decreased affective pain at 6 months ( $r = -.26, p = .03$ ). Increases in emotional awareness (LEAS-other score only) correlated with decreases in BPI pain at post-treatment ( $r = -.30, p = .01$ ), and sensory pain at both post-treatment ( $r = -.35, p = .004$ ) and 6 months ( $r = -.28, p = .02$ ). Increases in emotional approach coping correlated with decreases in depressive symptoms ( $r = -.24, p = .04$ ) and psychological distress ( $r = -.27, p = .02$ ) at post-treatment. Finally, decreases in alexithymia were correlated with decreases in BPI pain at post-treatment ( $r = .25, p = .04$ ).

**Table 3**

Pain-related outcomes of the intervention: Baseline, post-treatment, and 6-month follow-ups and effect sizes (N = 72).

Outcome measure	Mean (SD)	Change from baseline	t	Effect size	30% improve n (%)	50% improve n (%)	70% improve n (%)
Pain intensity (BPI)							
Pre-consult	5.11 (1.87)						
Post-treatment	2.93 (2.00)	−2.18	−9.54***	−1.17	46 (63.9)	30 (41.7)	16 (22.2)
6-month	2.84 (2.14)	−2.27	−9.06***	−1.21	45 (62.5)	40 (55.6)	25 (34.7)
Pain interference (BPI)							
Pre-consult	5.49 (2.23)						
Post-treatment	2.83 (2.27)	−2.66	−9.23***	−1.19	49 (68.1)	39 (54.2)	24 (33.3)
6-month	2.60 (2.35)	−2.89	−8.98***	−1.30	47 (65.3)	38 (52.8)	33 (45.8)
Depressive sx (CESD)							
Pre-consult	25.82 (11.70)						
Post-treatment	15.26 (11.02)	−10.55	−8.05***	−0.90	45 (62.5)	31 (43.1)	16 (22.2)
6-month	14.53 (11.65)	−11.29	−8.48***	−0.99	45 (62.5)	37 (51.4)	23 (31.9)
Sensory pain (MPQ-SF)							
Pre-group	9.28 (6.10)						
Post-treatment	5.56 (5.50)	−3.72	5.81***	−0.61	41 (56.9)	36 (50.0)	20 (27.8)
6-month	6.09 (6.84)	−3.19	−3.61**	−0.52	41 (56.9)	36 (50.0)	26 (36.1)
Affective pain (MPQ-SF)							
Pre-group	3.49 (3.15)						
Post-treatment	1.68 (2.12)	−1.81	−5.57***	−0.57	51 (70.8)	45 (62.5)	33 (45.8)
6-month	1.85 (2.57)	−1.64	−4.77***	−0.52	52 (72.2)	49 (68.1)	38 (52.8)
Distress (BSI)							
Pre-group	56.08 (32.72)						
Post-treatment	34.68 (29.80)	−21.40	−7.75***	−0.65	47 (65.3)	28 (38.9)	15 (20.8)
6-month	31.16 (29.04)	−24.92	−8.40***	−0.76	50 (69.4)	38 (52.8)	25 (34.7)

BPI = Brief Pain Inventory; CESD = Center for Epidemiologic Studies-Depression Scale; MPQ-SF = McGill Pain Questionnaire (short form); BSI = Brief Symptom Inventory.

Effect size calculated with this formula: (post M − baseline M)/baseline SD.

All 72 patients included in all cells; missing values replaced with last value obtained from each patient.

\*\*  $p < .01$ .\*\*\*  $p < .001$ .

### Treatment predictors

Our final analyses examined how baseline (pre-consultation) depressive symptoms and the presence of widespread pain predicted outcomes of the treatment. As shown in Table 4, greater depressive symptoms significantly predicted greater reductions (i.e., more improvement) in multiple outcomes, including BPI pain intensity at 6 months; and pain interference, depressive symptoms, affective pain, and distress at both post-treatment and 6-month follow-ups. Widespread pain, however, was not predictive. The 26 patients with widespread pain did not differ significantly on any outcome measure at either post-treatment or 6-month follow-up from the 46 patients without widespread pain (all  $p > .20$ ); effect sizes were of comparable magnitude for widespread and no widespread pain sub-groups.

Finally, we examined patient age, gender, education, pain duration, and baseline opioid use as potential confounders of the above relationships. Of these five variables, only education had a significant relationship with any of the measures of change in outcomes to post-treatment or follow-up. Being more educated was related to a greater reduction in pain interference ( $r = -.23$ ,  $p = .052$ ) and depression ( $r = -.26$ ,  $p = .03$ ) at the 6-month follow-up; however, controlling for education did not eliminate the significant relationships noted above between baseline depression or changes in process measures and the various outcomes. Similarly, controlling for age, gender, pain duration, or opioid use did not change these relationships.

### Discussion

This study offers preliminary evidence for substantial efficacy of a novel psychological intervention for chronic musculoskeletal pain. In this non-randomized, uncontrolled case series, we found that a relatively brief intervention that focuses on attributing pain to psychological processes, emotional awareness and expression, and engaging in

desired activities despite pain leads not only to statistically but also clinically significant improvements in pain intensity, interference, depression, and distress, which last for at least 6 months. These improvements are considered “very large” by conventional standards (between 1.0 and 1.2 SD). Perhaps more persuasively, approximately two-thirds of the

**Table 4**

Association of baseline depressive symptoms and the presence or absence of widespread pain with the treatment outcomes (change scores).

Outcome measure time point change	Baseline depressive symptoms	Widespread pain (n = 26) M (SD)	No widespread pain (n = 46) M (SD)
	r		
Pain intensity (BPI)			
Post-treatment	−.20	−2.21 (2.04)	−2.15 (1.89)
6-month	−.23*	−2.18 (1.88)	−2.32 (2.26)
Pain interference (BPI)			
Post-treatment	−.28*	−2.51 (2.23)	−2.74 (2.58)
6-month	−.33**	−2.71 (2.12)	−3.00 (3.04)
Depressive sx (CESD)			
Post-treatment	−.54***	−9.86 (9.78)	−10.94 (11.89)
6-month	−.48***	−9.06 (9.12)	−12.55 (12.28)
Sensory pain (MPQ-SF)			
Post-treatment	−.18	−3.55 (5.22)	−3.95 (5.59)
6-month	−.06	−3.24 (6.23)	−3.16 (8.19)
Affective pain (MPQ-SF)			
Post-treatment	−.48***	−2.08 (2.84)	−1.73 (2.67)
6-month	−.39**	−1.92 (2.19)	−1.60 (3.32)
Distress (BSI)			
Post-treatment	−.39**	−29.20 (41.97)	−20.80 (22.07)
6-month	−.39**	−28.19 (28.08)	−23.08 (23.50)

Note: Outcome change scores calculated as post (or 6-month) minus baseline (i.e., lower values mean more improvement).

BPI = Brief Pain Inventory; CESD = Center for Epidemiologic Studies-Depression Scale; MPQ-SF = McGill Pain Questionnaire (Short Form); BSI = Brief Symptom Inventory.

The widespread pain and no widespread pain subgroups did not differ on change in any measure at any time point (all  $p > .25$ ).\*  $p < .05$ .\*\*  $p < .01$ .\*\*\*  $p < .001$ .

patients reached the standard clinical improvement criterion of 30% reduction in pain and other symptoms, and fully one-third or more of the patients reached the very stringent criterion of 70% improvement. Half of the patients had pain at or below 2.0 on a 0 to 10 scale after 6 months, and some (6.9%) were fully pain-free. After just five sessions—one individual consultation and four group sessions—these durable improvements appear to surpass substantially those obtained by standard CBT or acceptance and mindfulness-based psychological interventions for chronic musculoskeletal pain [7,9,12].

This study also demonstrated that this novel intervention is associated with changes in hypothesized processes or mechanisms. Over the course of the four group sessions, patients increased in their attribution that psychological processes cause their pain, which is consistent with our theoretical model. Patients also improved their emotional awareness, which was assessed with a performance-based measure of language use, the LEAS. A change in LEAS scores over a brief emotion-oriented treatment is noteworthy, because this measure is thought to be relatively free of the biases that influence self-report questionnaires, such as mood and social desirability effects. In support of the LEAS effect, we also found that self-reported emotional approach coping increased and alexithymia decreased over the four group sessions. That is, patients reported that they increased in their differentiation, understanding, and expression of their emotions. It is noteworthy that all of these measures have been studied almost exclusively as correlates or predictors of health, but rarely have studies demonstrated changes in these measures over the course of a brief therapy that targets emotional processes.

Moreover, we found that these changes in attributions and emotional processes were associated with changes in the outcome measures. Increases in patients' attribution of pain to psychological processes, emotional awareness (especially of emotions of others), and emotional approach coping; and decreases in alexithymia were linked to improvements in outcomes of treatment. These process-outcome links were particularly robust at post-treatment, but changes in attributions also predicted improvements at 6-month follow-up. This suggests that these cognitive and emotional changes may serve as the mechanisms by which this treatment improves pain and adjustment. Unfortunately, we were not able to assess the process measures frequently enough to determine their temporal relationship to changes in outcome, so it remains possible that changes in the process measures simply coincide with changes in pain and other outcomes.

There is some evidence from studies of CBT and other approaches that more depressed patients have relatively poor outcomes of CBT for pain management [32–34]. In contrast, we found that patients with greater depressive symptoms at baseline actually had more improvement in pain and other outcomes and were more likely to complete the study (6-month follow-up) than patients with lesser depressive symptoms. Similarly, patients with widespread pain or fibromyalgia often have relatively poor responses to CBT and mindfulness approaches [7,35], perhaps because such patients have, on average, elevated emotional and interpersonal disturbances, including trauma histories [44,45] that are not directly addressed by those interventions. We found, however, that those patients with widespread pain responded as well to our treatment as did patients without widespread pain. We think that a strong focus on psychological processes including emotional processing of stress and trauma through expressive writing, which has been found to be more effective for people with fibromyalgia than other types of pain [28], leads our treatment to be particularly helpful for those with more disturbed psychological profiles and widespread pain.

We do not believe, however, that our approach is appropriate for all patients with chronic pain. Indeed, many patients with chronic pain have peripheral disease processes or structural anomalies that generate pain, and CBT and acceptance/mindfulness approaches may be better suited to help such patients adapt to, cope with, or manage their pain. A model that attributes pain primarily to CNS and psychological processes may not apply in such cases, and the emotional and relational

problems in such patients are likely to be consequences of their pain rather than causes. Furthermore, the diagnostic framework used in this study has not been systematically applied to unselected patients presenting to a chronic pain program, although we suspect that a significant proportion of such patients are likely to fall into the category of CNS-generated pain.

In addition to the fact that our intervention applies only to a subset of people with chronic pain, this study has other limitations. Notably, it was an uncontrolled study of a case series of patients; therefore, we do not know how much of the effect is due to factors such as the passage of time, repeated assessments, attention from a professional, or engaging in any treatment. However, the average duration of pain in this sample was over 8 years, and the natural history of such long-standing chronic pain and widespread pain shows few remissions [46]. Nonetheless, the next step will be to conduct a randomized controlled trial comparing this approach against treatment as usual and then, ideally, against an alternative approach, such as CBT. We also did not track changes in the use of analgesic medication or other treatment approaches over the course of the intervention that might have contributed to improvements in outcomes. However, the effects were noted immediately after the rather brief intervention, and the program made no recommendations about analgesics or alternative treatments. Furthermore, patients taking opioids medication at baseline benefited as much as those not taking opioids. These observations reduce concerns that the benefits were due to engaging in alternative treatments or changing medications.

Another limitation is that the patients in this study were relatively well-educated, and they self-selected into treatment at a mind-body clinic rather than a clinic that uses medications or procedures. This intervention may be more successful for people who can readily understand mind-body links and attributions, and who are motivated to engage in such a change process. Indeed, we found some evidence that more educated patients had better outcomes. Thus, this treatment needs to be tested with a broader spectrum of patients, particularly those who are less educated; who are recruited from primary care, rehabilitation centers, or pain clinics; or who adhere to a medical model for pain management. The effects may be weaker for such patients. Given that this intervention was offered by a single, highly committed practitioner, it also is important to test the effects of this intervention in the hands of other professionals who are trained to offer it. The authors have developed a curriculum for training therapists, which can be delivered in 16–20 h, including supervision. It covers the initial consultative visit to discern linkages between stressful life events and the onset and exacerbation of pain and other symptoms, how to present the conceptual model, and how to conduct the various emotional processing interventions.

Our assessment strategy also had some limitations. Some measures were completed prior to the initial consultation because they were part of the standard clinical practice, and we added other measures after that consultation but before the group sessions, after patients consented to the study. It would have been ideal to repeat the same outcome measures (BPI and CESD) between consultation and group sessions, so that we could have determined how much change occurred due to the consultation alone. Furthermore, we assessed the process measures only before and after the four group sessions; thus, not only do we not know how much these processes changed out to 6 months, but the changes likely would have been larger had we been able to assess them prior to the consultation, rather than after it.

Despite these limitations, this study has important implications. A relatively brief psychological intervention for chronic musculoskeletal pain in which two-thirds of the patients have clinical improvements, and one-third have at least 70% improvement, is highly noteworthy and suggests that this intervention has much potential value. Substantial pain reduction is common in this program, and full pain remission is possible for some people. Current models of pain treatment appear to view all pain as similar and only able to be managed or accepted.

This study suggests that it may be useful to consider a model that affirms that some types of pain are CNS-based and primarily influenced by psychological factors, and that re-attribution and emotional awareness and expression can be adaptive and pain-reducing.

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## References

- [1] B.I. Martin, R.A. Deyo, S.K. Mirza, J.A. Turner, B.A. Comstock, W. Hollingworth, et al., Expenditures and health status among adults with back and neck problems, *JAMA* 299 (2008) 656–664.
- [2] M.B. Yunus, Fibromyalgia and overlapping disorders: The unifying concept of central sensitivity syndromes, *Semin. Arthritis Rheum.* 36 (2007) 339–356.
- [3] R.A. Deyo, S.K. Mirza, J.A. Turner, B.I. Martin, Overtreating chronic back pain: time to back off? *J. Am. Board Fam. Med.* 22 (2009) 62–68.
- [4] J.L. Friedly, B.A. Comstock, J.A. Turner, P. Heagerty, R.A. Deyo, S.D. Sullivan, et al., A randomized trial of epidural glucocorticoid injections for spinal stenosis, *N. Engl. J. Med.* 371 (2014) 11–21.
- [5] J.B. Staal, R.A. de Bie, H.C. de Vet, J. Hildebrandt, P. Nelemans, Injection therapy for subacute and chronic low back pain: an updated Cochrane review, *Spine* 34 (2009) 49–59.
- [6] S.P. Morley, A.P. Williams, New developments in the psychological management of chronic pain, *Can J Psych* 60 (2015) 168–175.
- [7] Bernardy K, Klose P, Busch AJ, Choy EHS, Häuser W. Cognitive behavioural therapies for fibromyalgia. *Cochrane Database Syst. Rev.* 2013, Issue 9. Art. No.: CD009796. DOI: <http://dx.doi.org/10.1002/14651858.CD009796.pub2>.
- [8] B.M. Hoffman, R.K. Papas, D.K. Chatkoff, R.D. Kerns, Meta-analysis of psychological interventions for chronic low back pain, *Health Psychol.* 26 (2007) 1–9.
- [9] Williams, AC de C, Eccleston C, Morley S. Psychological therapies for the management of chronic pain (excluding headache) in adults. *Cochrane Database Syst. Rev.* 2012, Issue 11. Art. No.: CD007407. DOI: <http://dx.doi.org/10.1002/14651858.CD007407.pub3>.
- [10] L.M. McCracken, K.E. Vowles, Acceptance and commitment therapy and mindfulness for chronic pain: Model, process, and progress, *Am. Psychol.* 69 (2014) 178–187.
- [11] S. Schmidt, P. Grossman, B. Schwarzer, S. Jena, J. Naumann, H. Walach, Treating fibromyalgia with mindfulness-based stress reduction: Results from a 3-armed randomized controlled trial, *Pain* 152 (2011) 361–369.
- [12] M.M. Veehol, M.J. Oskam, K.M.G. Schreurs, E.T. Bohlmeier, Acceptance-based interventions for the treatment of chronic pain: a systematic review and meta-analysis, *Pain* 152 (2011) 533–542.
- [13] J.L. Wetherell, N. Afari, T. Rutledge, J.T. Sorrell, J.A. Stoddard, A.J. Petkus, et al., A randomized, controlled trial of acceptance and commitment therapy and cognitive-behavioral therapy for chronic pain, *Pain* 152 (2011) 2098–2107.
- [14] M.A. Lumley, J.L. Cohen, G.S. Borszcz, A. Cano, A.M. Radcliffe, L.S. Porter, et al., Pain and emotion: A biopsychosocial review of recent research, *J. Clin. Psychol.* 67 (2011) 942–968.
- [15] R.F. Anda, V.J. Felitti, J.D. Bremner, J.D. Walker, C. Whitfield, B.D. Perry, et al., The enduring effects of abuse and related adverse experiences in childhood: a convergence of evidence from neurobiology and epidemiology, *Eur. Arch. Psychiatry Clin. Neurosci.* 256 (2006) 174–186.
- [16] J.C. Beckham, A.L. Crawford, M.E. Feldman, A.C. Kirby, M.A. Hertzberg, J.R. Davidson, et al., Chronic posttraumatic stress disorder and chronic pain in Vietnam combat veterans, *J. Psychosom. Res.* 43 (1997) 379–389.
- [17] D.A. Davis, L.J. Lueken, A.J. Zautra, Are reports of childhood abuse related to the experience of chronic pain in adulthood? A meta-analytic review of the literature, *Clin. J. Pain* 21 (2005) 398–405.
- [18] R.J. Gatchel, P.B. Polatin, T.G. Mayer, The dominant role of psychosocial risk factors in the development of chronic low back pain disability, *Spine* 20 (1995) 2702–2709.
- [19] R.T. Goldberg, W.N. Pachas, D. Keith, Relationship between traumatic events in childhood and chronic pain, *Disabil. Rehabil.* 21 (1999) 23–30.
- [20] B. Van Houdenhove, E. Neerinx, R. Lysens, H. Vertommen, L. van Houdenhove, P. Onghena, et al., Victimization in chronic fatigue and fibromyalgia in tertiary care: a controlled study on prevalence and characteristics, *Psychosom* 42 (2001) 21–28.
- [21] M.B. Yunus, Central sensitivity syndromes: a new paradigm and group nosology for fibromyalgia and overlapping conditions, and the related issue of disease versus illness, *Semin. Arthritis Rheum.* 37 (2008) 339–352.
- [22] A. Abbass, S. Kisely, K. Kroenke, Short-term psychodynamic psychotherapy for somatic disorders, *Psychother. Psychosom.* 78 (2009) 265–274.
- [23] J.W. Burns, P. Quartana, W. Gilliam, E. Gray, J. Matsuura, C. Nappi, et al., Effects of anger suppression on pain severity and pain behaviors among chronic pain patients: evaluation of an ironic process model, *Health Psychol.* 27 (2008) 645–652.
- [24] L.S. Greenberg, S.C. Paivio, Working with the Emotions in Psychotherapy, Guilford, New York, 1997.
- [25] M.A. Lumley, J.L. Cohen, R.A. Stout, L.C. Neely, L.M. Sander, A.J. Burger, An emotional exposure-based treatment of traumatic stress for people with chronic pain: preliminary results for fibromyalgia syndrome, *Psychother* 45 (2008) 165–172.
- [26] P.J. Quartana, K.L. Yoon, J.W. Burns, Anger suppression, ironic processes and pain, *J. Behav. Med.* 30 (2007) 455–469.
- [27] A. Abbass, S. Campbell, K. Magee, I. Lenzer, G. Hann, R. Tarzwell, Cost savings of treatment of medically unexplained symptoms using intensive short-term dynamic psychotherapy (ISTDP) by a hospital emergency department, *Arch Med Psychol* 2 (2010) 34–44.
- [28] M.A. Lumley, E.R. Sklar, J.N. Carty, Emotional disclosure interventions for chronic pain: from the laboratory to the clinic, *Translational Behav Med* 2 (2012) 73–81.
- [29] J.E. Sarno, The mindbody prescription: Healing the body, healing the pain, Warner Books, New York, 1998.
- [30] N. Selfridge, F. Peterson, Freedom from fibromyalgia: The 5 week program proven to conquer pain, Three Rivers Press, New York, 2001.
- [31] G.L. Moseley, D.S. Butler, Fifteen years of explaining pain: The past, present, and future, *J. Pain* 16 (2015) 807–813.
- [32] D.A. Drossman, B.B. Toner, W.E. Whitehead, N.E. Diamant, C.B. Dalton, S. Duncan, et al., Cognitive-behavioral therapy versus education and desipramine versus placebo for moderate to severe functional bowel disorders 1, *Gastroenterology* 125 (2003) 19–31.
- [33] L.M. McCracken, D.C. Turk, Behavioral and cognitive-behavioral treatment for chronic pain: outcome, predictors of outcome, and treatment process, *Spine* 27 (2002) 2564–2573.
- [34] J.A. Turner, S. Holtzman, L. Mancl, Mediators, moderators, and predictors of therapeutic change in cognitive-behavioral therapy for chronic pain, *Pain* 127 (2007) 276–286.
- [35] R. Lauche, H. Cramer, G. Dobos, J. Langhorst, S. Schmidt, A systematic review and meta-analysis of mindfulness-based stress reduction for the fibromyalgia syndrome, *J. Psychosom. Res.* 75 (2013) 500–510.
- [36] H. Schubiner, Unlearn your pain, Mind Body Publishing, Pleasant Ridge, MI, 2010.
- [37] R.D. Lane, D.M. Quinlan, G.E. Schwartz, P.A. Walker, S.B. Zeitlin, The Levels of Emotional Awareness Scale: a cognitive-developmental measure of emotion, *J. Pers. Assess.* 55 (1990) 124–134.
- [38] A.L. Stanton, S.B. Kirk, C.L. Cameron, S. Danoff-Burg, Coping through emotional approach: scale construction and validation, *J. Pers. Soc. Psychol.* 78 (2000) 1150–1169.
- [39] R.M. Bagby, J.D. Parker, G.J. Taylor, The twenty-item Toronto Alexithymia Scale–I. Item selection and cross-validation of the factor structure, *J. Psychosom. Res.* 38 (1994) 23–32.
- [40] C. Cleeland, K. Ryan, Pain assessment: global use of the brief pain inventory, *Ann. Acad. Med.* 23 (1994) 129–138.
- [41] L. Radloff, The CES-D Scale: a self-report depression scale for research in the general population, *Appl. Psychol. Meas.* 1 (1977) 385–401.
- [42] R. Melzack, The short-form McGill Pain Questionnaire, *Pain* 30 (1987) 191–197.
- [43] L.R. Derogatis, M. Melisratos, The brief symptom inventory: an introductory report, *Psychol. Med.* 13 (1983) 595–605.
- [44] B. Van Houdenhove, U. Egle, P. Luyten, The role of life stress in fibromyalgia, *Curr. Rheumatol. Rep.* 7 (2005) 365–370.
- [45] E.A. Walker, D. Keegan, G. Gardner, M. Sullivan, D. Bernstein, W.J. Katon, Psychosocial factors in fibromyalgia compared with rheumatoid arthritis II: sexual, physical, and emotional abuse and neglect, *Psychosom. Med.* 59 (1997) 572–577.
- [46] B. Walitt, M.A. Fitzcharles, A.L. Hassett, R.S. Katz, W. Häuser, F. Wolfe, The longitudinal outcome of fibromyalgia: a study of 1555 patients, *J. Rheum* 38 (2011) 2238–2246.