The Effects of a Pain Psychology and Neuroscience Self-Evaluation Internet Intervention

A Randomized Controlled Trial

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Objectives: Many patients' chronic musculoskeletal pain is strongly influenced by central nervous system processes such as sensitization or amplification. Education about pain neuroscience can change patients' beliefs but has less consistent effects on pain outcomes. Patients may have greater clinical benefits if the educational intervention is personalized, and they evaluate various psychosocial risk factors with respect to their pain. We developed and tested a brief, internet-based Pain Psychology and Neuroscience (PPN) self-evaluation intervention.

Materials and Methods: From a patient registry, 104 adults reporting chronic musculoskeletal pain were randomized to the PPN intervention or a matched, active, education control condition. At baseline and 1-month (primary endpoint) and 10-month follow-ups, participants reported pain severity (primary outcome) and multiple secondary outcomes. Primary analyses compared the 2 experimental conditions using analyses of covariances; post hoc exploratory analyses compared the effects of PPN in subgroups of patients who met criteria for fibromyalgia (FM; n = 50) or who did not (n = 54; primarily spinal pain).

Results: At 1-month follow-up, compared with the control condition, PPN led to significantly lower pain severity ($\eta_p^2 = 0.05$) and interference ($\eta_p^2 = 0.04$), greater brain ($\eta_p^2 = 0.07$) and psychological ($\eta_p^2 = 0.07$) attributions for pain, and greater readiness for pain self-management ($\eta_p^2 = 0.08$). Effects on distress, pain catastrophizing, kinesiophobia, and life satisfaction were not significant. Exploratory analyses showed that the PPN intervention was especially beneficial for patients without FM but was of less benefit for those with FM. Most of the effects (except attributions) were lost at 10 months.

Discussion: A brief PPN self-evaluation intervention, presented online, can yield short-term improvements in musculoskeletal pain severity and interference, especially for people with spinal/localized pain rather than FM, perhaps because the psychology/neuroscience perspective is more novel for such patients.

Key Words: chronic back pain, fibromyalgia, centralized pain, neuroscience pain education, internet-based interventions

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Chronic musculoskeletal pain is substantially influenced by the central nervous system (CNS). Central sensitization, amplification, and even generation of pain are affected by processes that are cognitive (eg, appraisals and predictions), emotional (eg, psychosocial stress and regulation), and social/relational (eg, others' reactions, cultural expectations).[1–4] Although fibromyalgia (FM) is widely recognized as a central sensitization pain condition,[5,6] other types of musculoskeletal pain, such as back, neck, arm, and other localized pain problems also have a substantial centralized component.[7–11] Patients with musculoskeletal pain vary not only in the degree of centralization of their pain but also in their awareness of and willingness to adopt a "brain-based" rather than "peripheral injury" view of their pain.[12,13] The belief that one's pain is due primarily or solely to peripheral pain generators such as structural abnormalities, injured tissues, or disease—a belief that is often buttressed by anomalous laboratory or imaging findings—can be a barrier to effective psychological treatment of these pain conditions.[14]

Pain neuroscience education (also known as neurophysiological pain education or therapeutic neuroscience education) is a theoretical framework that has given rise to various educational interventions. Such interventions attempt to help patients shift their understanding of chronic pain from a traditional model—that their pain stems from structural or tissue damage—to a model of pain based on neurophysiological findings of the key role of the brain in chronic pain.[12,15,16] These education interventions are presented to patients in different formats, such as individual meetings, group workshops or seminars, booklets, and electronic modalities. Studies have tested the effects of such pain neuroscience education, and meta-analyses of these trials show that these interventions can shift patients' attributions of their pain's etiology and reduce their pain catastrophizing; however, effects on pain severity and pain interference are smaller and inconsistent.[13,15,16]

In most of these pain neuroscience educational interventions, patients are relatively passive recipients of the information, which is usually presented to them in a standardized, one-size-fits-all manner. The information is not tailored or personalized to each patient, nor are patients asked to self-evaluate or reflect deeply on the applicability of the information to their pain onset, course, and presentation. Yet research demonstrates that personalized learning is more powerful than standardized education,[17,18] suggesting that pain neuroscience education might be more effective if patients are asked to explore the relevance of the information to themselves. Furthermore, pain neuroscience education interventions usually focus on the neurophysiology of pain but provide much less information on important cognitive, emotional, social, and developmental processes that are known risk factors for chronic pain. In our clinical work, we have focused on 5 evidence-based risk factors or indicators that

patients' pain is relatively "centralized"; that is, that their brains play a primary role in their pain. These 5 indicators are: (1) the pain is widespread, and the patient has (or has had during their lives) other central sensitization syndromes[5,19]; (2) the patient has pain-related fearful beliefs including pain catastrophizing, kinesiophobia, and pain anxiety[20,21]; (3) the patient has stress-inducing or anxiety-inducing personality traits such as perfectionism, worry, or low self-esteem[22–24]; (4) stressful experiences in the patient's life were associated with pain onset and/or linked with pain exacerbations[25–27]; and (5) the patient has had adverse childhood experiences or developmental psychosocial trauma.[28,29]

We believe that the effects of pain neuroscience education might be enhanced if patients are presented information about such risk factors, asked to evaluate themselves on each, and encouraged to reflect on the relevancy of what they have learned to their pain experience. Therefore, we created a brief Pain Psychology and Neuroscience (PPN) intervention to accomplish these goals. Although pain neuroscience education is commonly provided in person, internet-based, self-guided education may be more efficient and have greater reach. Therefore, we placed this intervention on the internet and conducted a randomized controlled trial (RCT) to test its effects in people with chronic musculoskeletal pain. Importantly, very few or no RCTs of pain neuroscience education have included active, credible control conditions—thereby limiting conclusions about the specificity of the effects of pain neuroscience education. Thus, we compared PPN to an active, health-relevant, equivalent control exercise. Our primary hypothesis was that, compared with the control condition, PPN would reduce pain severity (primary outcome) and other secondary outcomes (eg, pain interference, distress, pain catastrophizing, and kinesiophobia) and increase psychological and brain-based attributions for pain and readiness for pain self-management. We hypothesized that such effects would occur at least in the short-term—at the primary endpoint of 1-month follow-up, but we also conducted a long-term follow-up (10 mo) to determine the duration of any short-term effects.

Our primary hypothesis was tested on the entire sample of patients with chronic musculoskeletal pain, but study participants varied in the extent to which they had widespread versus more localized pain. Nearly half of the sample met criteria for FM, whereas the other half did not. FM is the prototypical centralized condition,[5] and rarely is there evidence of peripheral disease or pathology to explain the widespread pain and other symptoms. Both professional and lay populations increasingly endorse the belief that FM stems from an "oversensitive" CNS and that psychosocial stress contributes to the condition.[30,31] In contrast, patients with localized pain, such as back or neck pain, can often point to initial injuries that caused acute pain as well as anomalous laboratory or imaging results of their spines, peripheral nerves, or other tissues that purportedly account for their chronic pain. Given these differences between FM and localized pain, we conducted post hoc exploratory analyses to determine whether the PPN intervention led to benefits in each of these 2 pain subgroups.

MATERIALS AND METHODS

Participants

Participants were English-speaking adults who had registered in an on-line research registry—the University of Michigan Health Research Volunteer Pool (umhealthresearch.org). Patient information on the registry was reviewed for inclusion and exclusion criteria by a research team member. Included patients reported a diagnosis of low back pain and/or FM for at least 3 months. Although additional information on the registry was somewhat limited and we were unable to conduct more detailed screening, we excluded patients from the study if their registry reported: (1) the presence of other serious disease or impairment (eg, cancer, systemic infection, serious vision impairment); (2) clear evidence of significant structural damage likely causing their pain (eg, vertebral compression fracture); (3) being considered for interventional spine procedures (eg, steroidal injections) or surgery; (4) use of illicit drugs; or (5) serious mental illness.

The study was approved by our Institutional Review Board and preregistered on Clinicaltrials.gov (NCT03391661). Recruitment occurred from December 2017 through April 2018; follow-ups were completed in May 2018 (1-mo) and February 2019 (10-mo). During recruitment, the patient registry automatically populated a list of potential participants, whose profiles were then manually screened to determine eligibility. Eligible participants were invited to participate via email, which contained a hyperlink to an internet-based survey platform (Qualtrics). Individuals choosing to participate provided informed consent and then completed baseline assessment measures in Qualtrics, which was followed immediately by their randomly assigned intervention condition.

Study Design and Randomization

This internet-based RCT compared the novel PPN intervention to an active, matched, control intervention that focused on health behaviors. At baseline, participants completed self-report measures of the outcomes and were immediately randomized to one of the 2 conditions in a 1:1 ratio by the built-in Qualtrics randomizer. Participants then proceeded to complete either the PPN intervention or control condition, as assigned; both conditions were a single session that typically lasted 20 to 25 minutes (based on Qualtrics recorded start and stop times). One month (ie, primary endpoint) and again 10 months (secondary endpoint) after completing the intervention, participants were sent a link to the Qualtrics survey that contained the follow-up measures. Participants were blind to assigned condition while completing baseline measures, and researchers were blind to condition assignment throughout data collection. Participants were compensated $30 in total for completing the baseline and 1-month follow-up assessments and an additional $10 for the 10-month follow-up.

Experimental Condition: PPN Intervention

We created the PPN condition for this study, drawing ideas and materials for the risk factors and exercises from several sources, including co-author Schubiner's *Unlearn Your Pain* manual,[32] widely used self-report measures, and a publicly available video. In this intervention, participants were first prepared for the subsequent exercises by viewing a 3-minute educational video (*Neural Pathways*; www.youtube.com/watch?v=D36yy63CHq4) about the role of the brain in chronic pain. The video is a narrated set of animations designed to educate people about the brain's neural pathways, pain perception, and how pain can be a learned response that is no longer driven primarily by injury or tissue damage but rather by expectations and fears. The video concludes by emphasizing that the brain can generate pain and that this process can be reversed or "unlearned."

After watching the video, participants completed 5 brief self-assessment exercises to explore evidence-based risk factors for "brain-based" pain. Two of these exercises used formal self-report scales, whereas the others presented lists

that we have used clinically or a subset of items from multiple published measures. The goal was not to obtain psychometrically sound data on patients from these exercises, but rather to briefly engage patients in self-evaluation and self-reflection with exercises that they could readily complete and score or tally. In addition, we arranged the 5 exercises in an order that we anticipated is most acceptable and validating to patients who might be unaware or dubious of the role of the CNS in their pain; that is, from most pain-focused/least psychosocial to least pain-focused/most psychosocial. The 5 self-assessment exercises were:

(1) A body map showing the 19 possible pain locations of the 2010 American College of Rheumatology (ACR) Preliminary Diagnostic Criteria for Fibromyalgia Survey,[33] and completion of a checklist of other central sensitization or stress-related syndromes or diagnoses;
(2) Items (modified in format to represent presence or absence) that we sampled from popular scales that assess pain catastrophizing,[34] kinesiophobia,[35] and pain anxiety[36];
(3) A checklist of personality traits related to stress and pain chronicity that we worded in lay terms (eg, "being a perfectionist," "not standing up for yourself," "often worrying");
(4) A checklist of stressful life events that "may have happened just before the pain started or at times when the pain got worse" (eg, an accident, a physical attack, sexual assault, divorce or separation, death of loved one, legal problems, etc.); and
(5) The 10-item Adverse Childhood Experiences Scale.[37]

Each exercise presented items that participants answered as present or absent with respect to their own experience. For each exercise, participants were asked to review their answers (eg, count the number of items endorsed) and then read a concise interpretation in lay language to help them explore how much the specific factor was relevant to their pain; for example, "Research shows that people who experience these events are more likely to have brain-based pain." At the end of each exercise, participants rated how much they think that risk factor contributes to their pain. The content of this intervention is available from the authors upon request.

Control Condition: Health Behaviors

We created the control condition to be parallel in structure, duration, and engagement to the PPN intervention; and to be health-relevant but not address pain or its potential causes. Thus, we developed a self-evaluation of participant's health-related behaviors. Participants were first shown a brief animated educational video (*Four Rules for a Healthy Lifestyle, animated for Harvard Medical School*; www.youtube.com/watch?v=jKikTtcqqzs) about behavioral influences on physical health. Following the video, participants were presented 5 exercises, each of which required participants to report on their health behaviors during the prior 24 hours. These 5 health behaviors were diet, exercise, sleep, hygiene, and social relationships. For each of these domains, participants recounted specific details of the behavior, read a brief educational statement of the role of this health behavior in overall health, and then rated their behavior. The content of this intervention is also available from the authors.

Background Measures

Participants provided sociodemographic information (eg, age, sex, ethnicity, education, marital status) and completed the modified 2010 ACR Preliminary Diagnostic Criteria for Fibromyalgia Survey, including pain body map.[33] This measure was used to classify patients as having (or not) FM, as determined by the modified 2010/2011 scoring algorithm, which captures both the number of pain locations over the body and other somatic symptoms. For this study, those participants who did not meet FM criteria on this measure were classified as having non-FM pain. The majority of these non-FM patients (85%) reported chronic low back pain, but most patients reported pain in multiple locations. From one third to half of these patients reported pain in each of these regions—neck, upper back, or hip/buttock—suggesting that "spinal pain" was the most common pain complaint.

Outcome Measures

At baseline and 1- and 10-month follow-up assessments, participants completed self-report measures of pain severity (predefined primary outcome), as well as secondary outcomes of pain interference, psychological distress, pain attributions, readiness for change, pain catastrophizing, kinesiophobia, and life satisfaction.

Pain Severity and Interference

The Brief Pain Inventory (BPI)[38]; assessed pain severity (mean of the 4, 0 to 10 ratings of current pain and highest, lowest, and average over the past week) and pain interference during the past week (mean of 7 items). Internal consistencies in our sample at baseline were $\alpha = 0.87$ (severity) and $\alpha = 0.93$ (interference).

Psychological Distress

Three measures from the Patient Reported Outcomes Measurement Information System (PROMIS)[39]; assessed depression (short form 8b), anxiety (short form 8a), and anger (short form 5a). For each measure, patients rated items regarding the last week on a 5-point scale ranging from 1 (*never*) to 5 (*always*). To reduce the number of analyses, and because these 3 measures converge on a single construct (the 3 measures correlated between $r = 0.58$ and 0.69 with each other in this sample), we created a composite measure of psychological distress by taking the mean of all items from all 3 scales. Internal consistency of the composite measure in our sample at baseline was very high ($\alpha = 0.96$).

Psychological and Brain Attributions

We developed 2 brief scales to assess attributions for pain targeted by our intervention. The 4-item psychological attribution scale assessed participants' beliefs that their thoughts and feelings and psychological treatment impact pain (eg, "Changing my thoughts, feelings, or relationships can change my pain."). The 3-item brain attribution scale assessed participant's beliefs that their pain is brain-based (eg, "My pain is coming primarily from my brain."). Items were rated from 0 (*strongly disagree*) to 4 (*strongly agree*) and averaged. Internal consistencies in our sample at baseline were $\alpha = 0.80$ (psychological attribution) and $\alpha = 0.71$ (brain attribution).

Readiness for Pain Self-Management

The Pain Stages of Change Questionnaire (PSOCQ)[40]; assessed readiness to engage in pain self-management in each of the 4 stages of change (precontemplation, contemplation, preparation, action). Participants rated items from 0 (*strongly disagree*) to 4 (*strongly agree*), and items were totaled for each stage. A *readiness* score was calculated using the method presented in the scoring manual for the University of Rhode Island Change Assessment (URICA),[41] which is the instrument upon which the PSOCQ was based.

The score is the sum of the ratings for contemplation (C), action (A), and maintenance (M), minus the score for precontemplation (PC): (Readiness = [C+A+M]−PC).

Kinesiophobia

The 11-item version of the Tampa Kinesiophobia Scale (TKS-11)[42]; assessed fear of movement or (re)injury related to pain. Items were rated from 1 (*strongly disagree*) to 4 (*strongly agree*) and summed. Internal consistency in our sample at baseline was $\alpha = 0.84$.

Pain Catastrophizing

The 13-item Pain Catastrophizing Scale (PCS)[34]; assessed 3 dimensions of catastrophizing when in pain: rumination, magnification, and helplessness. Items were rated from 0 (*not at all*) to 4 (*all the time*) and summed. Internal consistency in our sample at baseline was $\alpha = 0.93$.

Life Satisfaction

The 5-item Satisfaction with Life Scale (SWLS)[43]; measured self-reported satisfaction with one's life on a 7-point scale ranging from 1 (*strongly disagree*) to 7 (*strongly agree*); items were summed. Internal consistency in our sample at baseline was $\alpha = 0.90$.

Statistical Analyses

Variables were examined for distributions and outliers; variables met assumptions of parametric tests without transformation or correction. Analyses were conducted on the full randomized sample of 104 participants (intent-to-treat) at the primary endpoint of 1-month follow-up. Primary analyses were analyses of covariances (ANCOVA) on each outcome, comparing the 2 conditions while controlling for the baseline value of the dependent variable as well as baseline pain severity, which was selected as a covariate because it has prognostic value for outcomes. Similar ANCOVAs were conducted at the 10-month follow-up. Post hoc, exploratory analyses compared the effects of PPN versus control by repeating these ANCOVAs within the FM and non-FM subgroups separately. For these exploratory analyses, we focused on effect sizes for PPN versus control within each subgroup rather than directly comparing subgroups on PPN effects via significance testing, given that the trial was neither planned nor powered to test for such subgroup differences. Effect sizes (η_p^2) were calculated for each outcome measure to indicate the magnitude of differences between PPN and control conditions; a η_p^2 value of 0.01 is traditionally considered small, 0.06 is medium, and 0.14 is large.[44] Analyses were conducted in SPSS 25.0; α was set at 0.05 (2-tailed).

To determine sample size for the primary analyses, we estimated the effect of PPN to be slightly larger than medium in magnitude ($f = 0.30$) based on a recent meta-analysis of pain neuroscience education.[16] Power analysis (conducted with G-power) indicated that a sample of 90 participants would provide power of 0.80 to detect that effect using a 2-condition ANCOVA with 2 covariates and a 2-tailed α of 0.05.

RESULTS

Participant Characteristics and Flow Through the Trial

Figure 1 shows the CONSORT diagram of participant flow through the study. Of the 138 people who were eligible and invited to participate, 104 (75.4%) did so. As shown in Table 1,

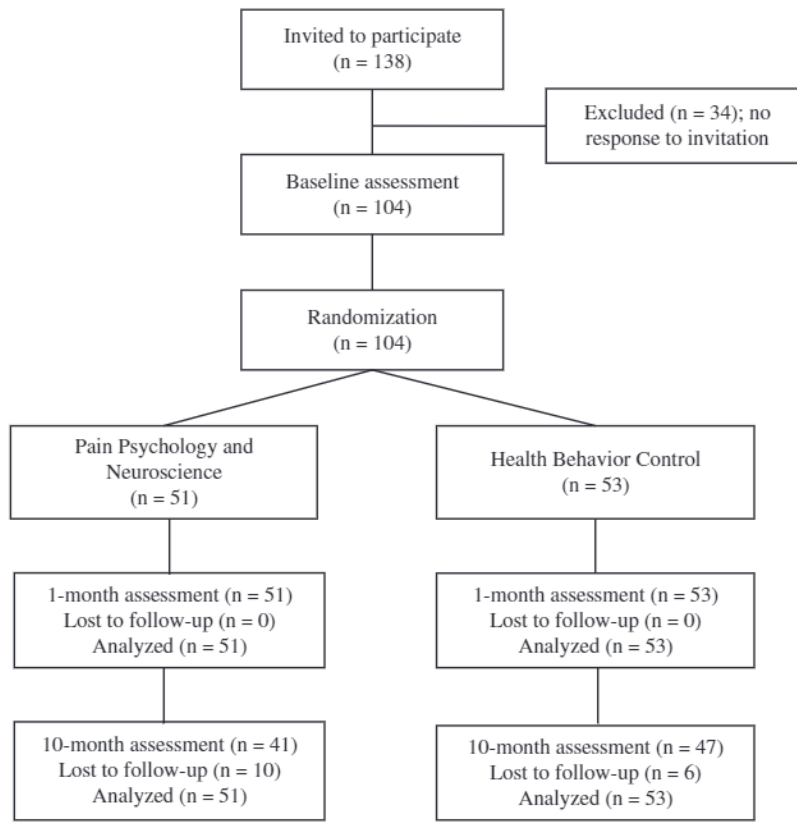

FIGURE 1. CONSORT flow diagram of participants through the trial.

TABLE 1. Sample and Condition Descriptive Data

Variables	Full Sample (N = 104)	Pain Psychology and Neuroscience (n = 51)	Health Behavior Control (n = 53)
Age, mean (SD) (y)	44.35 (14.71)	44.35 (14.87)	44.34 (14.69)
Pain duration, mean (SD) (y)	13.40 (9.20)	13.21 (8.99)	13.59 (9.48)
Sex, n (%)			
Male	26 (25.0)	15 (29.4)	11 (20.8)
Female	76 (73.1)	35 (68.6)	41 (77.4)
Other	2 (1.9)	1 (2.0)	1 (1.9)
Ethnicity, n (%)			
European American	87 (83.7)	43 (84.3)	44 (83.0)
African American	6 (5.8)	3 (5.9)	3 (5.7)
East Asian	1 (1.0)	0 (0.0)	1 (1.9)
South Asian	1 (1.0)	1 (2.0)	0 (0.0)
Middle Eastern	1 (1.0)	0 (0.0)	1 (1.9)
Pacific Islander	1 (1.0)	0 (0.0)	1 (1.9)
Other	7 (6.7)	4 (7.8)	3 (5.7)
Education, n (%)			
Doctoral degree	3 (2.9)	1 (2.0)	2 (3.8)
Master's degree	19 (18.3)	6 (11.8)	13 (24.5)
Bachelor's degree/4 y college	38 (36.5)	20 (39.2)	18 (34.0)
Associate's degree/2 y college	18 (17.3)	11 (21.6)	7 (13.2)
Some college	23 (22.1)	12 (23.5)	11 (20.8)
High school graduate	2 (2.9)	1 (2.0)	2 (3.8)
Marital status, n (%)			
Married	43 (41.3)	21 (41.2)	22 (41.5)
Committed relationship	17 (16.3)	5 (9.8)	12 (22.6)
Never married	26 (25.0)	13 (25.5)	13 (24.5)
Divorced/ separated	16 (15.4)	11 (21.6)	5 (9.4)
Widowed	2 (1.9)	1 (2.0)	1 (1.9)

the sample of 104 participants was comprised largely of women (73.1%), who were young to middle age on average ($M = 44.4$ y; SD = 14.7, range: 20 to 83 y), primarily white or European American (83.7%), and relatively educated (57.7% had completed a bachelors or higher degree). Just over half of the sample was married (41.3%) or in a committed relationship (16.3%).

As shown in Figure 1, all 104 participants completed baseline, their assigned intervention (PPN: n = 51; control: n = 53), and the 1-month follow-up assessment. At 10-month follow-up, 88 of the 104 participants (84.6%) provided follow-up data (PPN: n = 41; control: n = 47). The 16 patients lost to follow-up at 10 months did not reply to repeated email invitations to complete the on-line assessment. *t* Test and χ^2 analyses compared the 16 patients with incomplete data to the 88 who finished the 10-month follow-up. These 2 groups were comparable ($P > 0.20$) on experimental condition assignment, all baseline measures, and all medical/sociodemographic variables, except that non-completers were marginally less likely than completers to have a bachelor's or higher degree ($P = 0.08$).

Short-term (1-Month) Follow-up (Primary Endpoint)

Table 2 presents data (baseline *M*, SD, and the follow-up adjusted *M*, SE) for the outcome measures for both conditions. Also presented are ANCOVA *F* statistics, significance levels, and effect sizes comparing the 2 conditions.

Analyses first examined the primary outcome of pain severity at 1-month follow-up. The ANCOVA yielded a significant condition effect, $F_{(1,101)} = 5.24$, $P = 0.024$, $\eta_p^2 = 0.05$. The PPN condition reported lower pain severity than the control condition (near medium magnitude effect).

Analyses then tested secondary outcomes at 1-month follow-up. A significant condition effect was found for pain interference, $F_{(1,100)} = 4.80$, $P = 0.031$, $\eta_p^2 = 0.04$; the PPN condition had lower pain interference than the control condition (small/medium effect). For both attribution variables, there were significant condition effects: psychological attribution, $F_{(1,100)} = 7.44$, $P = 0.008$, $\eta_p^2 = 0.07$, and brain attribution, $F_{(1,100)} = 7.19$, $P = 0.009$, $\eta_p^2 = 0.07$. The PPN condition reported a greater belief that pain is brain-related and that pain is affected by psychological factors (medium effects). There also was a significant condition effect on readiness for pain-self management, $F_{(1,100)} = 8.22$, $P = 0.005$, $\eta_p^2 = 0.08$. The PPN intervention led to greater readiness to change than did the control condition (medium effect).

Finally, as shown in Table 2, the PPN condition did not differ significantly from the control condition on psychological distress, pain catastrophizing, kinesiophobia, or life satisfaction; although for all of these variables, the adjusted means were in the direction of better outcomes for the PPN than the control condition.

Long-term (10-Month) Follow-up

To conduct intent-to-treat analyses, missing follow-up data were imputed via multiple regression; the regression models included the variable's baseline and 1-month values as well as experimental condition, baseline pain severity, sex, and pain type (FM or non-FM).

As shown in Table 2, at the 10-month follow-up, the 2 conditions no longer differed significantly on pain severity, pain interference, or readiness for pain self-management. Compared with controls, however, the PPN condition continued to have higher attributions for the role of psychological factors, $F_{(1,84)} = 4.48$, $P = 0.04$, $\eta_p^2 = 0.05$ (near medium effect) and the brain, $F_{(1,84)} = 7.47$, $P = 0.01$, $\eta_p^2 = 0.08$ (medium effect). As at short-term follow-up, the 2 conditions did not differ on psychological distress, pain catastrophizing, kinesiophobia, or life satisfaction.

Intervention Effects for FM and Non-FM Subgroups

In this sample, 50 participants (48%) met criteria for FM, and 54 participants (52%) did not. These 2 subgroups were comparable on age ($P = 0.38$), pain duration ($P = 0.26$), education ($P = 0.65$), and marital status ($P = 0.39$); however, the FM group had a higher proportion of women (85.7%) than did the non-FM group (64.2%; $P = 0.01$). Participants with FM also reported greater pain severity, pain interference, catastrophizing, and distress than did participants without FM (all $P < 0.004$). Participants with FM also endorsed higher attributions of pain to their brain ($M = 2.03$, SD = 0.85) and to psychological processes ($M = 1.72$, SD = 0.90) than did those without FM ($M = 1.69$, SD = 0.75 and $M = 1.38$, SD = 0.88; $P = 0.03$ and 0.06, respectively).

Table 3 presents the outcome data for FM and non-FM subgroups separately. At the 1-month follow-up, among participants without FM, the PPN intervention (n = 28) led to significantly lower pain severity, $F_{(1,51)} = 8.51$, $P = 0.005$, $\eta_p^2 = 0.14$ (large effect) and pain interference, $F_{(1,50)} = 4.33$, $P = 0.042$, $\eta_p^2 =$

TABLE 2. Comparison of Pain Psychology and Neuroscience (PPN) Intervention and Health Behavior Control Condition at Follow-up

			ANCOVA		
Outcome/Timepoint	**PPN (n = 51)**	**Control (n = 53)**	***F***	***P***	**η_p^2**
Pain severity					
Baseline, mean (SD)	4.98 (1.54)	4.50 (1.73)			
1-mo adjusted, mean (SE)	4.03 (0.18)	4.60 (0.17)	5.24	**0.024**	0.05
10-mo adjusted, mean (SE)	4.46 (0.23)	4.21 (0.23)	0.62	0.434	0.01
Pain interference					
Baseline, mean (SD)	4.89 (2.44)	4.86 (2.64)			
1-mo adjusted, mean (SE)	3.91 (0.26)	4.71 (0.25)	4.80	**0.031**	0.04
10-mo adjusted, mean (SE)	4.36 (0.29)	4.58 (0.28)	0.28	0.601	0.00
Psychological distress					
Baseline, mean (SD)	1.41 (0.77)	1.40 (0.85)			
1-mo adjusted, mean (SE)	1.34 (0.07)	1.47 (0.07)	1.75	0.189	0.02
10-mo adjusted, mean (SE)	1.45 (0.08)	1.49 (0.08)	0.13	0.720	0.00
Psychological attribution					
Baseline, mean (SD)	1.70 (0.91)	1.39 (0.87)			
1-mo adjusted, mean (SE)	1.97 (0.10)	1.60 (0.09)	7.44	**0.008**	0.07
10-mo adjusted, mean (SE)	1.93 (0.10)	1.62 (0.10)	4.95	**0.028**	0.05
Brain attribution					
Baseline, mean (SD)	1.87 (0.80)	1.83 (0.83)			
1-mo adjusted, mean (SE)	2.14 (0.08)	1.83 (0.08)	7.19	**0.009**	0.07
10-mo adjusted, mean (SE)	2.22 (0.09)	1.85 (0.09)	9.21	**0.003**	0.08
Readiness to change					
Baseline, mean (SD)	8.28 (2.04)	8.02 (1.79)			
1-mo adjusted, mean (SE)	8.87 (0.19)	8.09 (0.19)	8.22	**0.005**	0.08
10-mo adjusted, mean (SE)	8.64 (0.24)	8.39 (0.23)	0.58	0.447	0.01
Pain catastrophizing					
Baseline, mean (SD)	17.94 (10.94)	18.47 (12.15)			
1-mo adjusted, mean (SE)	15.44 (1.09)	17.61 (1.07)	1.98	0.162	0.02
10-mo adjusted, mean (SE)	14.26 (1.03)	14.84 (1.01)	0.16	0.687	0.00
Kinesiophobia					
Baseline, mean (SD)	24.55 (7.63)	24.13 (5.72)			
1-mo adjusted, mean (SE)	22.95 (0.54)	24.28 (0.53)	3.08	0.082	0.03
10-mo adjusted, mean (SE)	23.77 (0.56)	24.16 (0.55)	0.24	0.623	0.00
Life satisfaction					
Baseline, mean (SD)	16.63 (7.08)	17.58 (8.43)			
1-mo adjusted, mean (SE)	17.71 (0.57)	17.30 (0.56)	0.25	0.618	0.00
10-mo adjusted, mean (SE)	18.36 (0.70)	17.73 (0.69)	0.41	0.524	0.00

Significant *P* values (2-tailed $P < 0.050$) are in bold.
ANCOVA indicates analyses of covariance.

0.08 (medium effect), than the control condition (n = 26). In addition, compared with controls, PPN led to greater brain attribution, $F_{(1,50)} = 5.10$, $P = 0.03$, $\eta_p^2 = 0.09$ (medium effect), and readiness to change, $F_{(1,50)} = 4.53$, $P = 0.038$, $\eta_p^2 = 0.08$ (medium effect), and lower pain catastrophizing, $F_{(1,50)} = 7.86$, $P = 0.007$, $\eta_p^2 = 0.14$ (large effect). In contrast, among participants with FM, PPN (n = 23) was not significantly different from controls (n = 27) on any outcome measure.

At 10-month follow-up, the effects of PPN on pain severity and most other outcomes were not significant for either the non-FM or FM subgroups. However, among those without FM, PPN led to a greater brain attribution for pain, $F_{(1,50)} = 12.36$, $P = 0.001$, $\eta_p^2 = 0.20$ (very large effect) than did control. For participants with FM, psychological attribution for pain was significantly higher after PPN than control, $F_{(1,46)} = 9.23$, $P = 0.004$, $\eta_p^2 = 0.17$ (large effect).

Although direct statistical comparisons of the FM and non-FM subgroups on the effects of PPN versus control were not planned and are substantially underpowered, we conducted such tests using the PROCESS macro of SPSS[45] to detect FM/non-FM subgroup moderation of the condition effects. Only one of the outcomes listed in Table 3 was significantly moderated by FM subgroup: the effect of PPN on reducing pain catastrophizing at 1-month follow-up was significantly stronger for the non-FM than the FM subgroup.

DISCUSSION

We developed a brief, internet-based PPN self-evaluation intervention in which participants learn about various risk factors for chronic centralized pain and evaluate the relevance of each risk factor to their pain. In this RCT, we compared the PPN intervention to a control condition that was health-relevant and equivalent in format and engagement, thereby controlling for a range of non-specific intervention factors. As hypothesized, we found that the PPN intervention decreased pain severity and pain interference 1 month later. Moreover, the PPN intervention changed participants' attributions—participants increased in attributions of their pain to their brain and psychological processes—and they became more ready to engage in pain self-management. Finally, post hoc exploratory analyses revealed that these effects occurred specifically among participants who did not have FM—who had primarily spinal pain—but the effects were generally absent among those with FM.

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TABLE 3. Comparison of Pain Psychology and Neuroscience (PPN) and Control Conditions for Participants With or Without Fibromyalgia

	Without Fibromyalgia					Fibromyalgia				
			ANCOVA					ANCOVA		
Outcome/Timepoint	PPN (n = 28)	Control (n = 26)	*F*	*P*	η_p^2	PPN (n = 23)	Control (n = 27)	*F*	*P*	η_p^2
Pain severity										
Baseline, mean (SD)	4.50 (1.04)	3.69 (1.53)				5.57 (1.84)	5.27 (1.56)			
1-mo adjusted, mean (SE)	3.32 (0.22)	4.25 (0.22)	8.51	**0.005**	0.14	4.77 (0.29)	5.03 (0.27)	0.44	0.513	0.01
10-mo adjusted, mean (SE)	3.96 (0.35)	3.95 (0.36)	0.00	0.981	0.00	4.97 (0.31)	4.55 (0.29)	0.95	0.334	0.02
Pain interference										
Baseline, mean (SD)	4.10 (2.27)	3.78 (2.24)				5.84 (2.35)	5.89 (2.62)			
1-mo adjusted, mean (SE)	2.97 (0.28)	3.85 (0.29)	4.33	**0.042**	0.08	4.87 (0.45)	5.71 (0.42)	1.85	0.181	0.04
10-mo adjusted, mean (SE)	3.67 (0.41)	4.05 (0.43)	0.40	0.532	0.01	5.15 (0.43)	5.14 (0.40)	0.00	0.990	0.00
Psychological distress										
Baseline, mean (SD)	1.28 (0.72)	1.08 (0.82)				1.57 (0.82)	1.71 (0.77)			
1-mo adjusted, mean (SE)	1.14 (0.09)	1.32 (0.10)	1.81	0.185	0.04	1.50 (0.11)	1.68 (0.10)	1.23	0.273	0.03
10-mo adjusted, mean (SE)	1.16 (0.10)	1.36 (0.11)	1.75	0.191	0.03	1.75 (0.13)	1.66 (0.12)	0.25	0.622	0.01
Psychological attribution										
Baseline, mean (SD)	1.54 (0.89)	1.21 (0.84)				1.89 (0.90)	1.56 (0.88)			
1-mo adjusted, mean (SE)	1.95 (0.14)	1.56 (0.14)	3.85	0.055	0.07	1.96 (0.14)	1.66 (0.13)	2.33	0.133	0.05
10-mo adjusted, mean (SE)	1.76 (0.14)	1.61 (0.15)	0.52	0.479	0.01	2.15 (0.13)	1.61 (0.12)	9.23	**0.004**	0.17
Brain attribution										
Baseline, mean (SD)	1.65 (0.64)	1.72 (0.86)				2.13 (0.91)	1.94 (0.80)			
1-mo adjusted, mean (SE)	2.18 (0.11)	1.81 (0.12)	5.10	**0.030**	0.09	2.07 (0.12)	1.88 (0.11)	1.16	0.287	0.03
10-mo adjusted, mean (SE)	2.27 (0.09)	1.79 (0.10)	12.36	**0.001**	0.20	2.21 (0.15)	1.86 (0.14)	2.84	0.098	0.06
Readiness to change										
Baseline, mean (SD)	7.82 (2.05)	8.06 (1.69)				8.84 (1.91)	7.98 (1.91)			
1-mo adjusted, mean (SE)	8.88 (0.28)	7.99 (0.29)	4.53	**0.038**	0.08	8.85 (0.28)	8.18 (0.25)	3.13	0.083	0.06
10-mo adjusted, mean (SE)	8.71 (0.32)	7.97 (0.33)	2.48	0.121	0.05	8.63 (0.36)	8.72 (0.33)	0.03	0.854	0.00
Pain catastrophizing										
Baseline, mean (SD)	14.57 (9.91)	14.38 (11.68)				22.04 (10.91)	22.41 (11.46)			
1-mo adjusted, mean (SE)	9.51 (1.26)	14.74 (1.31)	7.86	**0.007**	0.14	22.28 (1.81)	20.69 (1.67)	0.41	0.525	0.01
10-mo adjusted, mean (SE)	10.40 (1.52)	12.78 (1.58)	1.12	0.294	0.02	18.18 (1.42)	17.48 (1.31)	0.13	0.717	0.00
Kinesiophobia										
Baseline, mean (SD)	23.75 (7.38)	22.92 (5.75)				25.52 (7.99)	25.29 (5.55)			
1-mo adjusted, mean (SE)	21.37 (0.71)	22.91 (0.74)	2.13	0.151	0.04	24.80 (0.85)	25.66 (0.78)	0.55	0.461	0.01
10-mo adjusted, mean (SE)	22.75 (0.90)	23.30 (0.93)	0.17	0.680	0.00	24.84 (0.68)	25.14 (0.63)	0.10	0.756	0.00
Life satisfaction										
Baseline, mean (SD)	17.00 (7.72)	18.23 (8.47)				16.17 (6.36)	16.96 (8.51)			
1-mo adjusted, mean (SE)	18.54 (0.80)	17.27 (0.83)	1.16	0.287	0.02	16.96 (0.86)	17.11 (0.79)	0.02	0.894	0.00
10-mo adjusted, mean (SE)	19.56 (0.99)	17.07 (1.03)	2.90	0.095	0.06	17.16 (1.02)	18.14 (0.94)	0.50	0.483	0.01

Significant *P* values (2-tailed $P < 0.050$) are in bold.
ANCOVA indicates analyses of covariance.

One might be surprised that a self-guided, internet-based intervention that requires only 20 to 25 minutes reduced pain severity and interference 1 month later, especially when compared with an active, engaging control condition (similar to a placebo) rather than to no intervention, as is typical of pain neuroscience education trials. Pain neuroscience educational programs typically require a professional and more patient time and often do not reduce pain severity and interference.[15] We think that this PPN intervention is unique—rather than simply informing participants about pain neuroscience, the PPN intervention educates patients about key psychosocial risk factors for centralized pain and encourages patients to evaluate themselves and consider the relevance of each factor to their pain. In the PPN intervention, participants report and reflect on their experience of other central sensitization conditions, their thoughts and fears about their pain, their stress-inducing personality traits, their life events that triggered or have exacerbated their pain, and their history of adverse childhood events. Importantly, this intervention increased participants' attributions for the pain to their brain and to psychological factors, which are the specific shifts that the PPN intervention targets. One possible explanation of these findings, then, is that when patients reappraise the etiology of their pain "from body to brain" and consider the role played by emotions in their pain, the danger signal of bodily pain is reinterpreted as a "false alarm," and pain is consequently attenuated.[46] It also is noteworthy that the intervention increased participants' motivation for pain self-management rather than medical management; that is, according to the transtheoretical stages of change model, the PPN intervention reduced precontemplation and increased contemplation, preparation, and action. Such a shift is consistent with changed attributions and bodes well for the goal of shifting patients from biomedical to behavioral interventions.

It should be noted, however, that the effects of PPN on several other outcomes at 1-month follow-up were in the hypothesized direction but were smaller in magnitude and not statistically significant. In particular, the fact that pain catastrophizing and kinesiophobia did not decrease significantly after PPN tempers our proposal that PPN leads to a reattribution of pain as a false alarm of tissue damage. It should be noted,

however, that these 2 outcomes and 2 others that also did not change after PPN—psychological distress and life satisfaction—are "affect-laden." It is possible that PPN, which targets beliefs about the etiology of pain and the role of psychosocial factors, can change pain-related attributions and outcomes (intensity, interference) but has less impact on emotional processes. Affective outcomes may be more difficult to change than pain-related ones, as suggested in a review of several emotion-focused intervention trials for chronic pain that we have conducted.[47] Affect-related outcomes might need to be targeted directly in an intervention, perhaps with emotion regulation techniques.

Although our primary endpoint was at 1 month after the intervention, we were able to reassess nearly 85% of the sample at 10-month follow-up to evaluate whether any short-term effects were maintained. The intervention's short-term reductions in pain severity and interference and increased readiness to change were eliminated at long-term follow-up. The failure of this intervention to create long-lasting change in clinical outcomes is not surprising, given its brevity and lack of further intervention. It is possible that providing patients "booster sessions" or some other reminders about the PPN intervention over the subsequent months would have maintained its short-term benefits. Interestingly, PPN did lead to long-lasting shifts in brain and psychological attributions for pain. These findings indicate that the PPN intervention's attribution effects are robust, but they also suggest that changed attributions are not the sole contributor to the reduced pain, given that the pain effects were lost at 10 months despite continued changes in attributions.

About half of our sample met diagnostic criteria for FM, whereas the other half did not—they typically had more localized, usually spinal pain. These post hoc exploratory analyses revealed that, in participants without FM, the PPN intervention led to better outcomes than the control condition on multiple measures, including pain severity, interference, and catastrophizing, with medium to large effect sizes. In contrast, among participants with FM, the effects of PPN on outcomes were much smaller, and none were significant at 1-month follow-up.

There are several possible explanations for the finding of differential effects of the PPN intervention as a function of type of pain. Patients without FM may simply respond better to interventions than people with FM; indeed, FM has long been recognized as a challenging condition to treat, and meta-analyses suggest minimal improvement following psychological interventions for this population.[48] Relatedly, patients without FM in this sample had less pain, interference, distress, and pain catastrophizing than patients with FM. Some evidence suggests that patients who are less distressed and symptomatic at baseline respond better to behavioral interventions than do patients with more disturbances.[49,50]

More complex interpretations of the finding that only people without FM responded well to PPN pertain to patients' pre-existing beliefs about their pain. At baseline, participants with FM more strongly endorsed attributions that their pain was brain-based and psychologically influenced than did those without FM. This difference may stem from the observation that people with FM usually lack imaging or laboratory tests that suggest peripheral pathology that might drive their symptoms, and many patients have received the message that FM stems primarily from an oversensitized CNS. Thus, people with FM often acknowledge that their brains play a role in FM and that stress contributes to their symptoms. In contrast, people with more localized pain often recall a specific injury that triggered acute pain, and recurrence of this pain is interpreted as potential reinjury. In addition, unlike patients with FM, many patients with spinal and other localized pain can point to anomalous imaging or laboratory findings and subsequent medical diagnoses, buttressing their belief in peripheral causes of their pain.[51] Thus, in contrast to people with FM, those without FM likely found the PPN intervention to provide a novel alternative perspective on their pain. This new model may have led them to reappraise their pain's etiology and reduce their pain catastrophizing, which then attenuated their pain. A potential complementary perspective stems from the fact that people with FM experience increased invalidation from others due to the lack of a clear biomedical explanation for their symptoms.[52,53] It is possible that those with FM found the PPN intervention to be further stigmatizing and, as a result, did not respond as well to it as did those without FM. Finally, it also is possible that the increased readiness for pain self-management following PPN led the patients without FM to engage in cognitive and behavioral changes to reduce their pain and improve their functioning. Unfortunately, our study design precludes testing whether shifts in pain attributions or reduced pain catastrophizing preceded reductions in pain severity and interference, nor did we track changes in pain self-management over time. Future research should do so.

It should be acknowledged that our sample was small for these subgroup analyses, and we lacked the statistical power to properly test whether the PPN effect was significantly stronger in the non-FM than the FM subgroup. These subgroup analyses were, therefore, exploratory, and the finding that PPN is uniquely beneficial for non-FM musculoskeletal pain is, therefore, preliminary and needs replication in a larger sample to permit proper statistical comparisons. In addition, the non-FM subgroup was rather heterogeneous with respect to the location of their pain—although most of these patients had spinal pain, multiple pain locations were common. One might hypothesize that testing PPN on a sample that is more homogeneous with respect to localized pain, such as people with only chronic low back pain, might reveal even stronger effects of this intervention. Finally, it should be noted that the FM and non-FM groups differed on sex, pain severity and distress, and likely in other ways not assessed in this study. These other factors may account for subgroup differences on the PPN intervention and may predict outcomes of the PPN intervention better than the FM/non-FM subgrouping.

Additional limitations of this study should be noted. We relied on self-reports of participants' pain condition, engagement in the intervention, and outcome assessment; and we had no face-to-face contact with or oversight of the participants. Concerns about data quality, however, are somewhat attenuated by the enhanced study feasibility, including 100% retention through the 1-month follow-up. Two of the key outcome measures—psychological and brain attributions—were developed for this study, and although their internal consistencies were sound, their validity is unknown. Also, the exercises within the PPN intervention were created by us or extracted and modified from several sources; a future version of the PPN intervention might use only standardized measures for which patients' responses could be scored and compared with norms so that patients could be given evidence-based feedback. The use of an active, matched control condition is a strength of this study, controlling for confounding variables such as participation and rationale; however, in the absence of a no-intervention condition, we do not know how effective the PPN intervention is by itself, and we cannot rule out the possibility that some of the PPN effects are

due to unexpected responses to the control condition. It also would have been valuable to assess the credibility and acceptability not only of our control condition but especially of the PPN condition, given that the information presented may have conflicted with patients' prior beliefs about the etiology of their pain. Finally, the fact that we studied relatively well-educated people who had voluntarily joined a research registry and this particular education-oriented study suggests limitations to generalizability. It is important to test this intervention on other samples.

Despite these limitations, this brief, internet-based intervention helps patients learn about centralized pain and evaluate their risk factors for such pain. Patients' attributions shifted in the direction of brain and psychological processes accounting for the pain and in their readiness to engage in pain self-management. Moreover, this intervention resulted in some reduction in pain severity and interference for at least 1 month, although not for the longer-term. Exploratory analyses showed that these effects occurred in people who have more localized, largely spinal pain, but not patients with FM, perhaps because those with localized/spinal pain found this intervention to be more novel, challenging their higher baseline beliefs about the role of the brain and psychological factors in their pain.

Because it is brief and internet-based, the PPN intervention has the potential to reach a wide audience. Although the PPN intervention can stand alone, it may also serve to prepare patients with chronic musculoskeletal pain for subsequent engagement in more comprehensive psychological interventions. More generally, we encourage researchers and clinicians to consider the potential value of targeting patients' beliefs about the role of their body versus brain in their chronic pain and the importance of addressing emotional processes ranging from fears of one's pain to broader, more longstanding issues such as adverse life experiences.[54–57]

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