Journal of Bodywork & Movement Therapies (2015) 19, 464–472

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/jbmt

ORIGINAL RESEARCH

Neuroscience education in addition to trigger point dry needling for the management of patients with mechanical chronic low back pain: A preliminary clinical trial☆

Mario Téllez-García, PT [a],
Ana I. de-la-Llave-Rincón, PT, PhD [a,b,c],
Jaime Salom-Moreno, PT, PhD [a,b,c],
Maria Palacios-Ceña, PT [a,b,c],
Ricardo Ortega-Santiago, PT, PhD [a,b,c],
César Fernández-de-las-Peñas, PT, PhD [a,b,c,*]

[a] *Department of Physical Therapy, Occupational Therapy, Physical Medicine and Rehabilitation, Universidad Rey Juan Carlos, Alcorcón, Spain*
[b] *Esthesiology Laboratory, Universidad Rey Juan Carlos, Alcorcón, Spain*
[c] *Cátedra de Investigación y Docencia en Fisioterapia, Terapia Manual y Punción Seca, Universidad Rey Juan Carlos, Alcorcón, Madrid, Spain*

Received 14 September 2014; received in revised form 12 November 2014; accepted 14 November 2014

KEYWORDS
Low back;
Spine;
Myofascial pain;
Dry needling;
Education

Summary The objective of the current study was to determine the short-term effects of trigger point dry needling (TrP-DN) alone or combined with neuroscience education on pain, disability, kinesiophobia and widespread pressure sensitivity in patients with mechanical low back pain (LBP). Twelve patients with LBP were randomly assigned to receive either TrP-DN (TrP-DN) or TrP-DN plus neuroscience education (TrP-DN + EDU). Pain intensity (Numerical Pain Rating Scale, 0–10), disability (Roland–Morris Disability Questionnaire-RMQ-, Oswestry Low Back Pain Disability Index-ODI), kinesiophobia (Tampa Scale of Kinesiophobia-TSK), and pressure pain thresholds (PPT) over the C5–C6 zygapophyseal joint, transverse process of L3 vertebra, second metacarpal, and tibialis anterior muscle were collected at baseline and 1-

☆ The protocol for this study was approved by the Institutional Ethics Committee of Universidad Rey Juan Carlos.

* Corresponding author. Facultad de Ciencias de la Salud, Universidad Rey Juan Carlos, Avenida de Atenas s/n, 28922 Alcorcón, Madrid, Spain. Tel.: +34 91 488 88 84; fax: +34 91 488 89 57.
E-mail address: cesar.fernandez@urjc.es (C. Fernández-de-las-Peñas).

http://dx.doi.org/10.1016/j.jbmt.2014.11.012

week after the intervention. Patients treated with TrP-DN + EDU experienced a significantly greater reduction of kinesiophobia ($P = 0.008$) and greater increases in PPT over the transverse process of L3 ($P = 0.049$) than those patients treated only with TrP-DN. Both groups experienced similar decreases in pain, ODI and RMQ, and similar increases in PPT over the C5/C6 joint, second metacarpal, and tibialis anterior after the intervention (all, $P > 0.05$). The results suggest that TrP-DN was effective for improving pain, disability, kinesiophobia and widespread pressure sensitivity in patients with mechanical LBP at short-term. The inclusion of a neuroscience educational program resulted in a greater improvement in kinesiophobia.

Introduction

Chronic low back pain (LBP) represents a significant health care burden resulting in substantial costs to society (Linton, 1998). The economic burden associated with the management of LBP represents the highest compensation costs for workers in the United States (Dagenais et al., 2008). It has been estimated that the 1-year prevalence for LBP ranges from 22% to 65% (Walker, 2000). A recent study found that the prevalence of LBP has remained consistent over the years in Spain (Fernández-de-las-Peñas et al., 2013) suggesting that a better understanding for proper management of this condition is needed.

Physical therapy is one conservative treatment used for patients with chronic LBP. Several therapeutic approaches including lumbar spinal manipulation (Rubinstein et al., 2011), exercises (Haladay et al., 2013), soft tissue manual therapy (Brosseau et al., 2012), and neurophysiology education (neuroscience education) (Clarke et al., 2011) are typically used for management of this condition; however, further studies investigating the effects of interventions targeted soft tissues and neurophysiology pain education are needed (Clarke et al., 2011). The relevance of soft tissue interventions is related to the hypothesis that muscle trigger points (TrP) can be involved in centrally mediated pain mechanisms observed in patients with chronic LBP (Roussel et al., 2013).

TrPs are defined as hypersensitive regions in taut bands of a skeletal muscle that are painful to palpation and elicit referred pain (Simons et al., 1999). Clinically, TrPs are classified as active or latent. Active TrPs are spontaneously painful and when palpated elicit a referred pain reproducing the patient's symptoms. Latent TrPs are not painful spontaneously but when palpated elicit a referred pain that does not reproduce any of the patient's symptoms. Different studies have demonstrated that the levels of chemical mediators, e.g., bradykinin, serotonin, or substance P, are higher in active muscle TrPs as compared to latent TrPs and muscle regions without TrPs (Shah et al., 2005; Shah and Gilliams, 2008).

TrP-dry needling (TrP-DN) is a proposed therapeutic approach (Dommerholt and Fernandez-de-las-Peñas, 2013) used to reduce TrP-associated pain (Tough and White, 2011). Preliminary evidence suggests that TrP-DN of active TrPs in patients with LBP may be effective (Itoh et al., 2004; Chen and Nizar, 2011); however, additional well-designed studies are needed to further determine treatment effectiveness (Furlan et al., 2005). The mechanisms as to how TrP-DN exerts its effects are not fully understood. However, it has been proposed that both mechanical and neuro-physiological theory mechanisms exist (Dommerholt, 2011). In fact, potential neurophysiological effects of TrP-DN include a decrease in metabolic mediators, reduction of peripheral nociceptive inputs, and activation of descending inhibitory pain pathways (Chou et al., 2012; Cagnie et al., 2013).

Neuroscience pain education consists of educational sessions describing the neurobiology and neurophysiology of pain and pain processing by the nervous system. This approach attempts to increase patients' understanding of their pain by explaining in detail the underlying neurophysiology of chronic pain (Butler and Moseley, 2003). It has been proposed that neuroscience education can modify the attitude of the patients and in combination with other therapy interventions improves pain and disability (Louw et al., 2011). It is possible that TrP-DN can be an effective complementary intervention to neuroscience education for the management of patients with chronic LBP.

To the best of the authors' knowledge, no previous study has investigated the clinical and neurophysiological effects of the combination of TrP-DN and neuroscience education in patients with chronic LBP. Potentially, both treatments may help decrease hyper-excitability of central nervous system. Therefore, the purpose of this preliminary clinical trial was to determine the short-term effects of TrP-DN alone versus TrP-DN combined with neuroscience pain education on pain intensity, disability, kinesiophobia and widespread pressure pain sensitivity in individuals with chronic mechanical LBP. We hypothesized that patients receiving TrP-DN combined with neuroscience education would experience greater improvement in all outcomes than patients receiving TrP-DN in isolation.

Methods

Participants

A preliminary single-blind randomized clinical trial was conducted. Consecutive patients with chronic non-specific LBP referred by their physician for physical therapy from January to June 2014 were screened for eligibility criteria.

In the current study, chronic non-specific low back pain was defined as pain symptoms localized below the costal margin and over the gluteus area persisting for ≥3 months. To be eligible patients had to exhibit: 1, age between 18 and 65 years; 2; history of non-specific LBP without referral into the lower extremity longer than 1 year; 3, score ≥4 points on the Roland Morris Disability Questionnaire; 4, have not received physical therapy within the last 6 months; and 5, exhibit at least 1 active TrP reproducing their symptom.

All of the following criteria were required for a patient to be diagnosed of active TrPs (Simons et al., 1999): 1, presence of a palpable taut band; 2, presence of a hypersensitive spot in the taut band; 3, palpable or visible local twitch on snapping palpation; and, 4, the referred pain elicited by palpation of the painful spot reproduced the symptoms, totally or partially, of the patient. These criteria have good inter-examiner reliability (kappa: 0.84—0.88) when applied by an experienced clinician (Gerwin et al., 1997). Participants were examined for the presence of active TrPs in the gluteus medius and quadratus lumborum muscles by a clinician with 8 years of clinical experience in the assessment and treatment of TrPs.

Patients were excluded if they exhibited any of the following: 1, lumbar stenosis; 2, clinical signs of radiculopathy; 3, diagnosis of lumbar spondylolisthesis; 4, diagnosis of fibromyalgia syndrome; 5, LBP with a specific underlying pathology such as tumor, infection, inflammatory disorder, herniated disc, prolapsed disc; 6, treatment with corticosteroid or oral medication in the past 6 months; 7, history of spinal surgery; 8, signs consistent with nerve root compression, e.g., positive straight-leg-raise test <45°, diminished lower-extremity force, or diminished reflexes; or, 9, fear to needles. The medical history from each patient was solicited from their primary care physician to assess the presence of the exclusion criteria. The local human research committee of the Universidad Rey Juan Carlos, Spain approved the protocol. The study was conducted following the declaration of Helsinki. All subjects signed an informed consent prior to their inclusion in the study.

Randomization

Following the baseline examination, patients were randomly assigned to receive either TrP-DN alone (TrP-DN group) or TrP-DN plus neuroscience education (TrP-DN + EDU group). Concealed allocation was performed by using a computer-generated randomized table of numbers created prior to the start of data collection by a researcher not involved in the recruitment or treatment of patients. Outcome measures were assessed before the intervention and 1-week after the last intervention by an assessor blinded to treatment allocation group. Patients were refrained from taking any medication or seeking additional treatments during the study period.

Pain and disability outcomes

Participants provided demographic and clinical data and also completed a number of measures of pain and disability including the Roland—Morris Disability Questionnaire (RMQ) (Roland and Morris, 1983), the Oswestry Low Back Pain Disability Index (ODI) (Fairbank et al., 1980), and a Numerical Pain Rate Scale (NPRS) (Jensen et al., 1999).

The RMQ is the most commonly used questionnaire for assessing disability due to LBP (Murphy and Lopez, 2013). It consists of 24 items reflecting limitations in different activities of daily living attributed to LBP including walking, bending over, sitting, lying down, dressing, sleeping, self-care and daily life activities. The patient marks each item that applies to his/her current status, and each item receives a score of 1. The total score ranges from 0 (no disability) to 24 (maximum possible disability) (Roland and Morris, 1983). The Spanish version of the RMQ has exhibited good test—retest reliability (ICC: 0.87) and good internal consistency (Cronbach α: 0.84—0.91) (Kovacs et al., 2002). It has been reported that a change of 2—3 points (Bombardier et al., 2001) or 30% from baseline scores (Jordan et al., 2006) represents Minimum Clinically Important Difference (MCID) for the RMQ.

The ODI consists of a questionnaire including 10 items referring to activities of daily living that might be disrupted by LBP. Each item is answered on a 6-point Likert scale ranging from "no problem at all" [0] to "not possible" [5]. The total score ranges from 0 to 50 (Fairbank et al., 1980). The Spanish version of the ODI has shown good test—retest reliability (ICC: 0.92) and favorable internal consistency (Cronbach α: 0.86) (Flórez et al., 1995). A change of 10 points has been recommended as the MCID for the ODI (Ostelo and de Vet, 2005).

A 10-point Numerical Pain Rating Scale (NPRS; 0: no pain, 10: maximum pain) was used to assess the patients' current level of LBP (Jensen et al., 1999). The MCID for the NPRS in patients with chronic LBP has been reported to be 2.5 points (Ostelo and de Vet, 2005).

Kinesiophobia assessment

The Tampa Scale of Kinesiophobia (TSK) was used to assess the fear of movement and (re) injury (Miller et al., 1991). It is a 17-item questionnaire where each item is scored on a 4-point Likert scale ranging from "strongly disagree" [1] to "strongly agree" [4]. Items 4, 8, 12, and 16 are negatively worded and reversed scored. Ratings are summed to yield a total score (ranging from 17 to 68 points) where higher values reflect greater fear of (re) injury (Miller et al., 1991). Test—retest reliability of the TSK ranges from 0.90 to 0.96 in individuals with chronic LBP (George et al., 2010). The Spanish version of this questionnaire has also demonstrated good reliability (internal consistency and stability) and validity (convergent and predictive) (Gómez-Pérez et al., 2011).

Pressure pain thresholds

Pressure pain threshold (PPT), defined as the amount of pressure applied for the pressure sensation to first change to pain (Vanderweeën et al., 1996), was assessed bilaterally (dominant and non-dominant sides) with an electronic algometer (Somedic AB©, Farsta, Sweden) over the C5—C6 zygapophyseal joint, the L3 transverse process, the second metacarpal, and the tibialis anterior muscle with the aim of

examining changes in widespread pressure pain sensitivity (Roussel et al., 2013). Patients were instructed to press a switch when the sensation first changed from pressure to pain. The mean of 3 trials was calculated and used for the analysis. Thirty seconds was provided between each trial. It has been reported that PPT conducted over the cervical spine exhibit excellent intra-rater reliability (ICC: 0.94—0.97) and good to excellent inter-rater reliability (ICC: 0.79—0.90) when performed on individuals with acute neck pain (Walton et al., 2011). The minimal detectable change MDC for PPT over the cervical spine and tibialis anterior muscle in individuals with acute neck pain has been reported to be 47.2 kPa and 97.9 kPa, respectively (Walton et al., 2011).

Trigger point-dry needling (TrP-DN)

Active TrPs located in the gluteus medius (see Fig. 1) and quadratus lumborum (see Fig. 2) muscles were treated with TrP-DN. These muscles were chosen because active TrPs are prevalent in patients with chronic mechanical LBP (Iglesias-González et al., 2013). The intervention was provided by a clinician with 10 years of experience in the treatment of TrPs using this technique. Patients in both groups received 3 sessions of TrP-DN over active TrPs with disposable stainless steel needles (0.3 mm * 30/50 mm, Novasan©, Madrid, Spain) that were inserted through the skin over the active TrP. In this study, the fast-in and fast-out technique described by Hong was applied (Hong, 1994). After locating an active TrP, the overlying skin was cleaned with alcohol, and the needle was subsequently inserted, penetrating the skin and muscle to a depth of approximately 20—25 mm into the TrP. The position of the patient was always side lying. Once inserted into the TrP, the needle was moved in multiple directions until the first local twitch response was obtained. It is suggested that multiple local twitch responses should be elicited during TrP-DN for successful treatment. Once the first local twitch response was obtained, the needling was performed in an up and down fashion, performing 5—8 mm vertical motions with no rotations (fast in and fast out technique), at approximately 1 Hz for 25—30 s with the aim of eliciting different local twitch responses. In such a manner, the needle performed multiple insertions into the active TrP without removing it from the skin. Since TrP-DN sometimes induces post-treatment soreness, patients were advised to report any increase in their symptoms after the intervention.

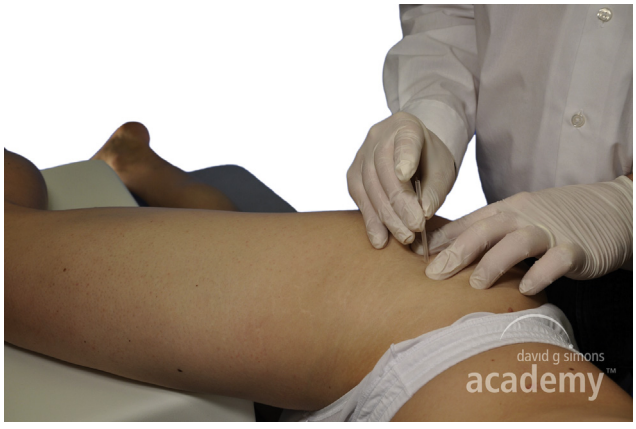

Figure 1 Trigger point dry needling (TrP-DN) applied over active TrPs in the gluteus medius muscle of a patient with mechanical low back pain. Copyright, David G Simons Academy™, Switzerland©, with permission.

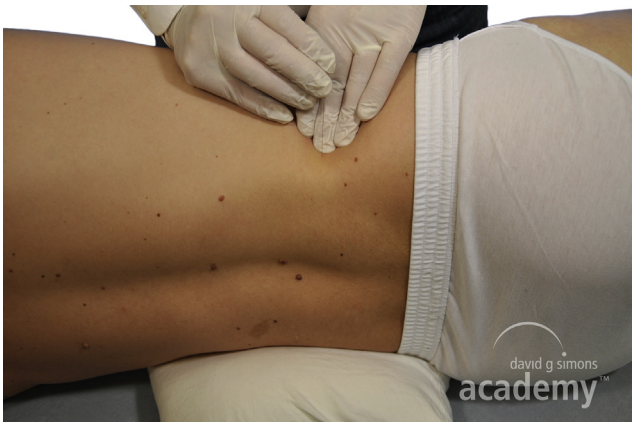

Figure 2 Trigger point dry needling (TrP-DN) applied over active TrPs in the quadratus lumborum muscle of a patient with mechanical low back pain. Copyright, David G Simons Academy™, Switzerland©, with permission.

All patients exhibited bilaterally active TrPs in the gluteus medius and quadratus lumborum muscles that referred pain reproducing their symptoms. Patients received 3 sessions of TrP-DN, once per week during the study period. TrP-DN was based on clinical findings. Active TrPs received dry needling in the first and second sessions in all patients (mean: 4). At the third and last treatment session a 40% reduced number of active TrPs were observed. Therefore, TrP-DN was only applied to the remaining active TrPs (mean: 2.6 points).

Neuroscience education

Participants allocated to TrP-DN + EDU received a 30 min education session, once per week for the last 2 weeks (treatment sessions number 2—3) after the application of TrP-DN. They received face-to-face individual educational sessions focused on neurophysiology of pain with no particular reference to the lumbar spine with a discussion of acute nociception versus chronic pain. In addition, patients were informed about the role of beliefs and attitudes towards their pain. A PowerPoint presentation based on the book "Explain Pain" (Butler and Moseley, 2003) was used for better understanding of the concepts. During the sessions patients were encouraged to ask potential questions and their input was used to individualize the information they received. Written information about pain physiology concepts discussed during the sessions was provided as homework between sessions (Nijs et al., 2011).

Statistical analysis

Data were analyzed with Statistical Package for Social Scientists (SPSS) version 18.0. Mean, standard deviations, and

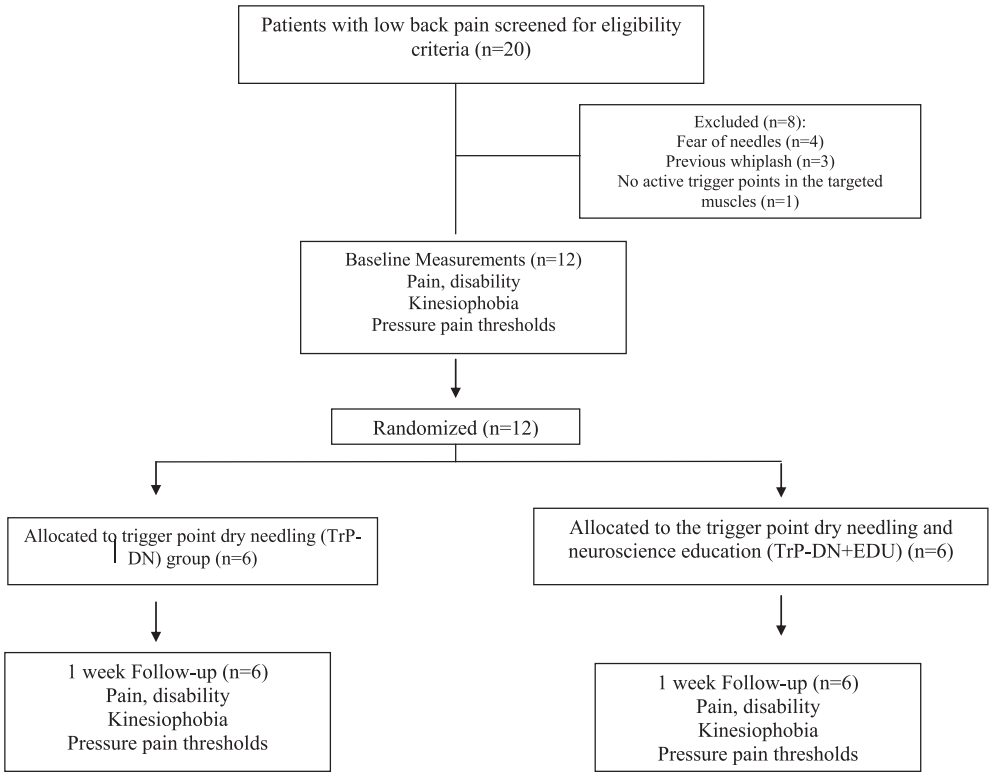

Figure 3 Flow diagram of patients throughout the course of the study.

95% confidence intervals (95% CI) were calculated for each variable. The Kolmogorov–Smirnov (KS-test) test showed normal distribution of quantitative data. Baseline demographic variables and self-report outcomes were compared between groups using independent *t*-tests for continuous data and χ^2 tests of independence for categorical data. Separate 2 × 2 mixed model analysis of variance (ANOVAs) with time (baseline, 1-week after) as the within-subjects factor and group (TrP-DN, TrP-DN + EDU) as the between-subjects factor were used for determine between-groups differences in pain (NPRS), disability (RMQ and ODI), and kinesiophobia (TSK). For PPT data, separate 2 × 2 × 2 mixed model ANOVAs with time (baseline, 1-week after) and side (dominant, non-dominant) as within-subjects factors and group (TrP-DN, TrP-DN + EDU) as the between-subjects factor were used. The main hypothesis of interest was the Group * Time interaction. To enable comparison of effect sizes, standardized mean score differences (SMDs) were calculated by dividing mean differences between both groups by the pooled standard deviation.

Results

Twenty consecutive patients with mechanical LBP were screened for eligibility criteria. Twelve patients (mean ± SD age: 36 ± 10 years old; 66% female) satisfied the eligibility criteria, agreed to participate, and were randomized to either the TrP-DN ($n = 6$) or TrP-DN + EDU ($n = 6$) group. The reasons for ineligibility are listed in Fig. 3 which provides a flow diagram of patient recruitment and retention. Baseline features between both groups were similar and comparable for all variables (Table 1).

The 2 × 2 mixed-model ANOVA revealed a significant Group * Time interaction for TKS ($F = 10.66$; $P = 0.008$): patients treated with TrP-DN + EDU experienced a greater reduction of kinesiophobia than those treated only with TrP-DN (Table 2). Between-group difference in change score was large (SMD: 3.01) in favor of the TrP-DN + EDU group. No significant Group * Time interaction was found for pain intensity ($F = 0.067$; $P = 0.801$), ODI ($F = 0.398$; $P = 0.542$) or RMQ ($F = 3.066$; $P = 0.111$), but there was a main effect for time with both groups experiencing a similar decrease in the intensity of pain ($F = 16.421$; $P = 0.002$), ODI ($F = 37.212$; $P < 0.001$), and RMQ ($F = 43.421$; $P < 0.001$), after the intervention (Table 2). Pre-post effect sizes were large (SMD > 1.01) for both groups in all outcomes. Between-group differences in change score was small (SMD: 0.25) for intensity of pain and moderate for RMQ and ODI (SMD < 0.65) in favor of the TrP-DN + EDU (pain and RMQ) or TrP-DN (ODI) group, respectively.

The 2 × 2 × 2 ANOVA revealed a significant Group * Time interaction for PPT over transverse process of L3 (Group * Time: $F = 4.396$, $P = 0.049$; Side * Time: $F = 1.162$, $P = 0.294$; Group * Time * Side: $F = 0.003$, $P = 0.956$), but not over the remaining locations: C5–C6 (Group * Time: $F = 0.058$, $P = 0.812$; Side * Time: $F = 0.345$, $P = 0.563$; Group * Time * Side: $F = 0.254$, $P = 0.620$), second metacarpal (Group * Time: $F = 0.296$, $P = 0.592$; Side * Time: $F = 0.054$, $P = 0.819$; Group * Time * Side: $F = 0.498$, $P = 0.488$), and tibialis anterior (Group * Time: $F = 0.179$, $P = 0.677$; Side * Time:

Table 1 Baseline demographics for both groups.

	TrP-DN group ($n = 6$)	TrP-DN + EDU group ($n = 6$)
Clinical features		
Gender (male/female)	2/4	2/4
Age (years)	37 ± 13	36 ± 5
Pain intensity (NPRS, 0–10)	4.8 ± 3.1	5.0 ± 2.1
Time with pain (months)	19 ± 8	17 ± 9
ODI (0–50)	30.0 ± 14.8	24.2 ± 9.7
RMQ (0–24)	8.3 ± 1.2	10.3 ± 3.4
TKS (17–68)	43.3 ± 5.9	41.5 ± 6.2
Pressure pain thresholds (kPa/cm^2)		
PPT C5–C6 dominant side	169.0 ± 28.2	176.1 ± 33.5
PPT C5–C6 non-dominant side	158.5 ± 30.2	174.7 ± 80.4
PPT transverse process L3 dominant side	242.6 ± 61.9	210.8 ± 60.1
PPT transverse process L3 non-dominant side	226.8 ± 20.3	217.8 ± 85.8
PPT second metacarpal dominant side	190.0 ± 38.2	251.1 ± 129.1
PPT second metacarpal non-dominant side	183.7 ± 44.8	199.5 ± 92.4
PPT tibialis anterior dominant side	321.1 ± 62.1	375.3 ± 71.8
PPT tibialis anterior non-dominant side	300.2 ± 44.4	347.6 ± 40.6

TrP-DN: trigger point dry needling; ODI: Oswestry low back pain disability index; RMQ: Roland–Morris disability questionnaire; NPRS: numerical pain rate scale; TSK: Tampa scale of kinesiophobia.

$F = 0.002$, $P = 0.963$; Group * Time * Side: $F = 0.437$, $P = 0.516$). Patients receiving TrP-DN + EDU exhibited large increases in PPT over the transverse process of L3 as compared to those receiving TrP-DN (between-group differences in change scores, SDM > 1.10, both sides). Again, there was a main effect for time with both groups experiencing similar increases in PPT over C5/C6 joint ($F = 35.709$; $P < 0.001$), the second metacarpal ($F = 5.885$; $P = 0.025$), and tibialis anterior ($F = 49.255$; $P < 0.001$), after the intervention (Table 3).

Ten of the 12 patients (83%) experienced some soreness after TrP-DN treatment but did not experience an increase in their symptoms. TrP-DN post-treatment soreness resolved spontaneously within 24–32 h in all patients without any intervention.

Discussion

The results of this preliminary randomized clinical trial suggest that TrP-DN was effective for improving pain, disability, kinesiophobia and widespread pressure pain sensitivity in subjects with mechanical LBP at short-term follow-up period. In addition, the inclusion of a neuroscience educational program resulted in a significantly greater reduction in kinesiophobia.

Patients in both groups experienced similar improvements in pain and disability. Although improvements in both groups exceeded the MCID of 2.5 points for pain intensity for this patient population (Ostelo and de Vet, 2005) the clinical relevance should be considered cautiously since the lower bound estimates of the 95% CI did not exceed this value (Table 2). However, within-group scores and their for both the ODI and RQM surpassed their respective MCID of 10 (Ostelo and de Vet, 2005) and 3 (Bombardier et al., 2001) points respectively, suggesting a clinically relevant effect on disability (Table 2). A similar reduction of pain and disability in both groups may suggest that TrP-DN was the main responsible for these changes. This finding could be related to the fact that a greater number of active TrPs was associated with higher intensity of the pain in patients with non-specific LBP (Iglesias-González et al., 2013).

Both groups also experienced a decrease in kinesiophobia, however changes were larger in those patients

Table 2 Baseline, final treatment session, and change scores for pain, disability and kinesiophobia.

Outcome group	Baseline	End of treatment	Within group change scores	Between-group difference in change scores
Pain intensity (NPRS, 0–10)				
TrP-DN group	4.8 ± 3.1	1.2 ± 1.1	−3.6 (−6.0, −1.1)	0.6 (−3.8, 4.8)
TrP-DN + EDU group	5.0 ± 2.1	0.8 ± 1.0	−4.2 (−6.6, −1.7)	
Oswestry low back pain disability index (ODI, 0–50)				
TrP-DN group	30.0 ± 14.8	6.0 ± 5.1	−24.0 (−39.1, −8.9)	4.5 (−11.3, 20.4)
TrP-DN + EDU group	24.2 ± 9.7	4.7 ± 3.2	−19.5 (−29.9, −9.0)	
Roland–Morris disability questionnaire (RMQ, 0–24)				
TrP-DN group	8.3 ± 1.2	2.2 ± 0.8	−6.1 (−8.0, 4.3)	3.6 (−1.0, 7.2)
TrP-DN + EDU group	10.3 ± 3.4	1.0 ± 1.1	−9.7 (−13.6, −5.1)	
*Tampa scale of kinesiophobia (TKS, 17–68)**				
TrP-DN group	43.3 ± 5.9	38.3 ± 5.1	−5.0 (−11.6, 1.6)	−12.7 (−21.3, −4.0)*
TrP-DN + EDU group	41.5 ± 6.2	23.8 ± 2.9	−17.7 (−25.1, −10.2)	

Values are expressed as mean ± standard deviation for baseline and final means and as mean (95% confidence interval) for within- and between-group change scores.
*Significant between-groups differences (Group * Time interaction, ANOVA $P < 0.05$).

Table 3 Baseline, final treatment session, and change scores for pressure pain thresholds.

Outcome group	Baseline	End of treatment	Within group change scores	Between-group difference in change scores
PPT C5–C6 dominant side (kPa/cm^2)				
TrP-DN group	169.0 ± 28.2	224.6 ± 46.8	55.6 (19.1, 92.3)	18.0 (−30.0, 65.7)
TrP-DN + EDU group	176.1 ± 53.5	249.7 ± 42.6	73.6 (32.4, 114.8)	
PPT C5–C6 non-dominant side (kPa/cm^2)				
TrP-DN group	158.5 ± 30.2	240.8 ± 42.4	82.3 (37.6, 126.3)	6.6 (−12.0, 15.6)
TrP-DN + EDU group	174.7 ± 80.4	250.3 ± 61.3	75.6 (25.5176.7)	
PPT transverse process L3 dominant side (kPa/cm^2)				
TrP-DN group	242.6 ± 61.9	335.7 ± 28.7	93.1 (38.4, 147.7)	61.8 (4.5, 128.1)*
TrP-DN + EDU group	210.8 ± 60.1	365.7 ± 95.1	154.9 (101.5, 208.3)	
PPT transverse process L3 non-dominant side (kPa/cm^2)				
TrP-DN group	226.8 ± 20.3	352.4 ± 42.1	125.6 (69.4, 181.8)	58.7 (50.9, 66.5)*
TrP-DN + EDU group	217.8 ± 85.8	402.1 ± 133.0	184.3 (111.7, 247.4)	
PPT second metacarpal dominant side (kPa/cm^2)				
TrP-DN group	190.0 ± 38.2	247.6 ± 57.7	57.6 (25.8, 89.4)	41.9 (−16.7, 75.3)
TrP-DN + EDU group	251.1 ± 129.1	266.8 ± 49.3	15.7 (7.6, 23.8)	
PPT second metacarpal non-dominant side (kPa/cm^2)				
TrP-DN group	183.7 ± 44.8	225.5 ± 52.4	41.8 (18.5, 72.1)	5.4 (−14.6, 25.8)
TrP-DN + EDU group	199.5 ± 92.4	246.7 ± 11.3	47.2 (23.7, 70.2)	
PPT tibialis anterior dominant side (kPa/cm^2)				
TrP-DN group	321.1 ± 62.1	444.3 ± 97.4	123.2 (32.6, 213.7)	8.1 (−13.8, 30.1)
TrP-DN + EDU group	375.3 ± 71.8	490.4 ± 103.1	115.1 (23.1, 207.1)	
PPT tibialis anterior non-dominant side (kPa/cm^2)				
TrP-DN group	300.2 ± 44.4	402.4 ± 63.0	102.2 (39.7, 164.7)	37.1 (−26.3, 100.5)
TrP-DN + EDU group	347.6 ± 40.6	486.9 ± 101.1	139.3 (37.7, 240.8)	

Values are expressed as mean ± standard deviation for Baseline and Final means and as mean (95% confidence interval) for within- and between-group change scores.
*Significant between-groups differences (ANOVA $P < 0.05$).

receiving TrP-DN plus neuroscience education. These findings seem expected since the objective of neuroscience education is decreasing the fear of movement or (re)injury. The current results are similar to previous studies supporting that neuroscience education programs are effective for the management of belief and attitudes of patients with LBP (Louw et al., 2011). We should recognize that the isolated application of TrP-DN also decreased kinesiophobia which may be attributed to the decrease in pain and disability. It is possible that both therapeutic interventions can be considered complementary for the management of patients with LBP.

This study also found a reduction of widespread pressure pain hypersensitivity in both groups as reflected by the increase in PPTs over both symptomatic-related areas (L3 vertebra and tibialis anterior) and non-symptomatic (C5/C6 and second metacarpal) areas. Within-group mean changes were larger than the MDC values of 47.2 kPa for the cervical spine and 97.9 kPa for the tibialis anterior muscle (Walton et al., 2011); but we should recognize that these changes exhibited broad 95%CIs and the lower bound did not always surpass. Other authors have established that differences between 20% and 25% are required to indicate a real change in PPTs (Prushansky et al., 2004). Changes in PPTs after the intervention in both groups were higher than 25% of pre-intervention scores in all areas (Table 3) supporting a meaningful effect for reducing widespread mechanical sensitivity. These results are similar to those previously found in patients with acute mechanical neck pain (Mejuto-Vázquez et al., 2014) supporting the hypothesis that TrP-DN can reduce widespread sensitivity.

The observed combined reduction in pain intensity and widespread pressure pain sensitivity after the TrP-DN application alone or combined with neuroscience education suggests possible segmental and central sensitization mechanisms (Dommerholt, 2011; Chou et al., 2012; Cagnie et al., 2013). In fact, it is hypothesized that TrP-DN provides a counter-irritant effect by removing nociceptive inputs resulting in inhibition of neuroplastic changes associated with central sensitization at the dorsal horn. Similarly, it is purported that therapeutic mechanisms of neuroscience education also involve a decrease of central nervous system hyper-excitability (Butler and Moseley, 2003; Louw et al., 2011) Therefore, both therapeutic interventions could be simultaneously applied for increasing their potential effects in patients with mechanical LBP.

Finally, the results need to be interpreted with caution based on some potential study limitations. First, we did not include a control group that received no treatment so we do not know if improvements observed in both groups could

also be partially due to the natural history of the condition; although this is unlikely since all patients had chronic symptoms. Second, we only collected outcomes at a short-term follow-up period. Third, we used TrP-DN alone or in combination with neuroscience education when in reality physical therapists usually treat patients with LBP using a multi-modal approach, so this may not truly reflect actual clinical practice. In fact, clinical guidelines suggest the use of manual therapy (including mobilization and manipulation) and exercises for the management of patients with mechanical LBP (Dagenais et al., 2010; Koes et al., 2010). Additionally, we applied a pragmatic and clinical approach where TrP-DN was applied based, on clinical findings. Future studies should continue to examine the effectiveness of multimodal approaches including TrP-DN and neuroscience education in combination with other accepted interventions such as spinal manipulative therapy or exercise for the management of patients with chronic non-specific LBP. Finally despite the small sample size of the current study, statistically significant and clinically relevant results were observed suggesting that a greater sample size would not alter the direction of the results. However, future trials may also benefit of greater sample size to increase the generalizability of the results.

Conclusion

The results of this preliminary randomized clinical trial suggest that TrP-DN was effective for improving pain, disability, kinesiophobia and widespread pressure pain sensitivity at short-term in individuals with mechanical LBP. The inclusion of neuroscience educational program exerts a greater impact for decreasing kinesiophobia. Additional large-scale clinical trials with a longer-term follow-up are needed to further confirm our results.

Conflicts of interest

The authors declare no conflicts of interest with the content of this article.

References

Bombardier, C., Hayden, J., Beaton, D.E., 2001. Minimal clinically important difference. Low back pain: outcome measures. J. Rheumatol. 28, 431—438.

Brosseau, L., Wells, G.A., Poitras, S., et al., 2012. Ottawa panel evidence-based clinical practice guidelines on therapeutic massage for low back pain. J. Bodyw. Mov. Ther. 16, 424—455.

Butler, D.S., Moseley, L., 2003. Explain the Pain. Noigroup Publications.

Cagnie, B., Dewitte, V., Barbe, T., et al., 2013. Physiologic effects of dry needling. Curr. Pain Headache Rep. 17, 348.

Chen, C.K., Nizar, A.J., 2011. Myofascial pain syndrome in chronic back pain patients. Korean J. Pain 24, 100—104.

Chou, L.W., Kao, M.J., Lin, J.G., 2012. Probable mechanisms of needling therapies for myofascial pain control. Evid. Based Complement. Alternat. Med. 705327.

Clarke, C.L., Ryan, C.G., Martin, D.J., 2011. Pain neurophysiology education for the management of individuals with chronic low back pain: a systematic review and meta-analysis. Man. Ther. 16, 544—549.

Dagenais, S., Caro, J., Haldeman, S., 2008. A systematic review of low back pain cost of illness studies in the United States and internationally. Spine J. 8, 8—20.

Dagenais, S., Tricco, A.C., Haldeman, S., 2010. Synthesis of recommendations for the assessment and management of low back pain from recent clinical practice guidelines. Spine J. 10, 514—529.

Dommerholt, J., Fernandez-de-las-Peñas, C., 2013. Trigger Point-Dry Needling: an Evidence and Clinical-Based Approach, first ed. Churchill Livingstone, Elsevier, London.

Dommerholt, J., 2011. Dry needling peripheral and central considerations. J. Man. Manip. Ther. 19, 223—237.

Fairbank, J.C., Couper, J., Davies, J.B., O'Brien, J., 1980. The Oswestry low back pain disability questionnaire. Physiotherapy 66, 271—273.

Fernández-de-las-Peñas, C., Alonso-Blanco, C., Hernández-Barrera, V., et al., 2013. Has the prevalence of neck and low back pain changed over the last 5 years? A population-based national study in Spain. Spine J. 13, 1069—1076.

Flórez, M., García, M.A., García, F., 1995. Adaptación transcultural a la población española de la escala de incapacidad por dolor lumbar de Oswestry. Rehabilitación 29, 138—145.

Furlan, A.D., Van Tulder, M., Cherkin, D., et al., 2005. Acupuncture and dry-needling for low back pain: an up-dated systematic review within the frame work of the Cochrane collaboration. Spine 30, 944—963.

George, S.Z., Valencia, C., Beneciuk, J.M., 2010. A psychometric investigation of fear-avoidance model measures in patients with chronic low back pain. J. Orthop. Sports Phys. Ther. 40, 197—205.

Gerwin, R., Shanon, S., Hong, C., Hubbard, D., Gevirtz, R., 1997. Interrater reliability in myofascial trigger point examination. Pain 69, 65—67.

Gómez-Pérez, L., López-Martínez, A.E., Ruiz-Párraga, G.T., 2011. Psychometric properties of the Spanish version of the Tampa scale for kinesiophobia (TSK). J. Pain 12, 425—435.

Haladay, D.E., Miller, S., Challis, J., Denegar, C., 2013. Quality of systematic reviews on specific spinal stabilization exercise for chronic low back pain. J. Orthop. Sports Phys. Ther. 43, 242—250.

Hong, C.Z., 1994. Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. Am. J. Phys. Med. Rehabil. 73, 256—263.

Iglesias-González, J.J., Muñoz-García, M.T., Rodrigues-de-Souza, D., Alburquerque-Sendín, F., Fernández-de-las-Peñas, C., 2013. Myofascial trigger points, pain, disability, and sleep quality in patients with chronic nonspecific low back pain. Pain Med. 14, 1964—1970.

Itoh, K., Katsumi, Y., Kitakoji, H., 2004. Trigger point acupuncture treatment of chronic low back pain in elderly patients: a blinded RCT. Acupunct. Med. 22, 170—177.

Jensen, M.P., Turbner, J.A., Romano, J.M., Fisher, L., 1999. Comparative reliability and validity of chronic pain intensity measures. Pain 83, 157—162.

Jordan, K., Dunn, K.M., Lewis, M., Croft, P., 2006. A minimal clinically important difference was derived for the Roland—Morris disability questionnaire for low back pain. J. Clin. Epidemiol. 59, 45—52.

Koes, B.W., van Tulder, M., Lin, C.W., et al., 2010. An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. Eur. Spine J. 19, 2075—2094.

Kovacs, F.M., Llobera, J., Gil del Real, M.T., et al., 2002. Validation of the Spanish version of the Roland—Morris questionnaire. Spine 27, 538—542.

Linton, S., 1998. The socio-economic impact of chronic back pain: is any one benefiting? Pain 75, 163—168.

Louw, A., Diener, I., Butler, D.S., Puentedura, E.J., 2011. The effect of neuroscience education on pain, disability, anxiety, and

stress in chronic musculoskeletal pain. Arch. Phys. Med. Rehabil. 92, 2041—2056.
Mejuto-Vázquez, M., Salom-Moreno, J., Ortega-Santiago, R., Truyols-Domínguez, S., Fernández-de-las-Peñas, C., 2014. Short term changes in neck pain, widespread pressure pain sensitivity and cervical range of motion after the application of trigger point dry needling in patients with acute mechanical neck pain: a randomized clinical trial. J. Orthop. Sports Phys. Ther. 44, 252—260.
Miller, R., Kori, S., Todd, D., 1991. The Tampa scale: a measure of kinesiophobia. Clin. J. Pain 7, 51—52.
Murphy, D.R., Lopez, M., 2013. Neck and back pain specific outcome assessment questionnaires in the Spanish language: a systematic literature review. Spine J. 13, 1667—1674.
Nijs, J., Paul van Wilgen, C., Van Oosterwijck, J., van Ittersum, M., Meeus, M., 2011. How to explain central sensitization to patients with "unexplained" chronic musculoskeletal pain: practice guidelines. Man. Ther. 16, 413—418.
Ostelo, R.W., de Vet, H.C., 2005. Clinically important outcomes in low back pain. Best Pract. Res. Clin. Rheumatol. 19, 593—607.
Prushansky, T., Dvir, Z., Defron-Assa, R., 2004. Reproducibility indices applied to cervical pressure pain threshold measurements in healthy subjects. Clin. J. Pain 20, 341—347.
Roland, M., Morris, R., 1983. A study of the natural history of back pain: part I. Spine 8, 141—144.
Roussel, N.A., Nijs, J., Meeus, M., Mylius, V., Fayt, C., Oostendorp, R., 2013. Central sensitization and altered central pain processing in chronic low back pain: fact or myth? Clin. J. Pain 29, 625—638.
Rubinstein, S.M., van Middelkoop, M., Assendelft, W.J., de Boer, M.R., van Tulder, M.W., 2011. Spinal manipulative therapy for chronic low-back pain: an update of a Cochrane review. Spine 36, E825—E846.
Shah, J.P., Gilliams, E.A., 2008. Uncovering the biochemical milieu of myofascial trigger points using in vivo microdialysis: an application of muscle pain concepts to myofascial pain syndrome. J. Bodyw. Mov. Ther. 4, 371—384.
Shah, J.P., Phillips, T.M., Danoff, J.V., Gerber, L.H., 2005. An in vitro microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. J. Appl. Physiol. 99, 1977—1984.
Simons, D.G., Travell, J., Simons, L.S., 1999. Myofascial pain and dysfunction In: The Trigger Point Manual, second ed., vol. 1. Williams & Wilkins, Baltimore.
Tough, E.A., White, A.R., 2011. Effectiveness of acupuncture/dry needling for myofascial trigger point pain. Phys. Ther. Rev. 16, 147—154.
Vanderweeën, L., Oostendorp, R.A., Vaes, P., Duquet, W., 1996. Pressure algometry in manual therapy. Man. Ther. 1, 258—265.
Walker, B., 2000. The prevalence of low back pain: a systematic review of the literature from 1966 to 1998. J. Spinal Disord. 13, 205—217.
Walton, D.M., Macdermid, J.C., Nielson, W., et al., 2011. Reliability, standard error, and minimum detectable change of clinical pressure pain threshold testing in people with and without acute neck pain. J. Orthop. Sports Phys. Ther. 41, 644—650.