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# **MUSCULOSKELETAL SECTION**

# Original Research Article Myofascial Trigger Points, Pain, Disability, and Sleep Quality in Patients with Chronic Nonspecific Low Back Pain

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## Abstract

Objective. To investigate the difference in the presence of trigger points (TrPs) between patients with chronic nonspecific low back pain (LBP) and healthy people, and to determine the relationship of TrPs with the intensity of ongoing pain, disability, and sleep quality.

Design. A cross-sectional study.

Setting. The role of TrPs in LBP has not been determined.

Patients. Forty-two patients with nonspecific LBP (50% women), aged 23–55 years old, and 42 age- and sex-matched controls participated.

Outcome Measures. TrPs were bilaterally explored within the quadratus lumborum, iliocostalis lumborum, psoas, piriformis, gluteus minimus, and gluteus medius muscles in a blinded design. TrPs were considered active if the subject recognized the local and referred pain as familiar symptoms, and TrPs were considered latent if the pain was not recognized as a familiar symptom. Pain measures were collected with a numerical pain rate scale, disability was assessed with the Roland–Morris questionnaire, and sleep quality was determined with the Pittsburgh Sleep Quality Index.

Results. Patients with nonspecific LBP exhibited a greater disability and worse sleep quality than healthy controls (P < 0.001). Patients with nonspecific LBP exhibited a mean of  $3.5 \pm 2.3$  active TrPs. Further, patients with nonspecific LBP showed a greater (P < 0.001) number of latent TrPs (mean:  $2.0 \pm 1.5$ ) than healthy controls (mean:  $1.0 \pm 1.5$ ). Active TrPs in the quadratus lumborum, iliocostalis lumborum, and gluteus medius muscles were the most prevalent in patients with nonspecific LBP. A greater number of active TrPs was associated with higher pain intensity ( $r_s = 0.602$ ; P < 0.001) and worse sleep quality ( $r_s = 0.338$ ; P = 0.03).

Conclusions. The local and referred pain elicited by active TrPs in the back and hip muscles contributes to pain symptoms in nonspecific LBP. Patients had higher disability and worse sleep quality than controls. The number of active TrPs was associated with pain intensity and sleep quality. It is possible that a complex interaction among these factors is present in patients with nonspecific LBP.

Key Words. Low Back Pain; Trigger Points; Myofascial Pain; Disability; Sleep

## Introduction

Nonspecific low back pain (LBP) is defined as pain between the costal margins and the inferior gluteal folds, usually accompanied by painful limitation of movement that may be associated with referred pain. The diagnosis of nonspecific LBP implies that the pain is not related to conditions such as fractures, spondylitis, trauma, tumor, infectious, vascular, metabolic, or endocrine-related processes. Nonspecific LBP is a major health problem in the modern society as 75% of the population will experience back pain at some time during the life [1]. The 1-year prevalence for LBP ranges from 22% to 65% [2]. Additionally, the economic burden associated with the management of LBP represents the highest compensation costs for workers in the United States [3,4].

As nonspecific LBP can be associated with referred pain, it has been proposed that muscle trigger points (TrPs) can be involved in pain processes in this condition [5]. Simons et al. defined a TrP as a hypersensitive spot in a taut band of a skeletal muscle that is painful on contraction, stretching, or stimulation of the muscle and elicits a referred distant pain [5]. From a clinical point of view, TrPs can be classified as active or latent. Active TrPs are those causing spontaneous pain or motor symptoms which stimulation reproduces the patient's symptom, and the pain is recognized as a familiar phenomenon. Latent TrPs do not cause spontaneous pain symptoms of the patient but referred pain when the muscle is stimulated [5]. Clinical distinction between active and latent TrPs has been substantiated by histochemical findings because higher levels of several chemical mediators, i.e., bradykinin, serotonin, or substance P, have been found in active TrPs compared with latent TrPs [6].

Few studies have examined the role of TrPs in the etiology and maintenance of LBP. Teixeira et al. [7] identified the presence of TrPs within the quadratus lumborum and gluteus medius muscles in 85.7% of patients with postlaminectomy pain syndrome. Chen and Nizar reported that 63.5% of patients with chronic back pain exhibited TrPs in the piriformis and lumbar paravertebral muscles [8]. In these patients, TrP dry needling involved a favorable outcome for pain. Samuel et al. found an association between the presence of TrPs in the muscles innervated by the corresponding segmental level, e.g., L4-L5 lesion and tibialis anterior muscle TrPs or L5-S1 lesion with gluteus medius and gastrocnemius TrPs, in individuals with lumbar disc prolapsed [9]. In addition, there is some evidence demonstrating that the treatment of active TrPs was effective for reducing symptoms in patients with LBP

[10]. Although these studies suggest that TrPs can be involved in the genesis of LBP, they focused on LBP caused by an underlying medical condition and did not include patients presenting with nonspecific LBP without any anatomical lesion. No previous study has investigated the relevance of active TrPs in patients with nonspecific LBP.

In addition to muscle impairments, sleep disturbances are considered an essential element in individuals with chronic LBP [11,12]. It has been proposed that addressing the ongoing cycle of pain and sleep disturbances is essential as an integral part of treatment of patients with chronic pain. We do not know if the presence of active TrPs is related to functional impairments such as sleep quality or self-rated disability in this population. Therefore, the aims of the current study were: 1) to investigate the presence of active TrPs in individuals with chronic nonspecific LBP; and 2) to determine the relationship between the presence of active TrPs, the intensity of ongoing pain, self-rated disability, and sleep quality in individuals with chronic nonspecific LBP.

## Methods

## Participants

In this study, patients with chronic nonspecific LBP recruited from a private clinic of physical therapy participated. To be included, they should suffer from a history of nonspecific LBP without referral into the lower extremity longer than 3 years with a pain-free period of at least 3 months preceding the current episode of LBP. Exclusion criteria included: 1) current pregnancy; 2) LBP with a specific underlying pathology such as tumor, infection, inflammatory disorders, hernia disc, and prolapsed disc; 3) signs consistent with nerve root compression, e.g., positive straight-leg-raise test <45°C, diminished lowerextremity force, or diminished reflexes; 4) prior lumbar spine surgery; 5) history of osteoporosis; or 6) spinal fracture. The medical history from each patient was solicited from their primary care physician to assess the presence of the exclusion criteria.

Additionally, healthy subjects without history of recurrent LBP and without any episode in the previous year, low back surgery or fracture, or neurological disorders, who responded to local advertisements, were also included. The control group was age- and sex-matched with the patient group. The protocol was approved by the local human research committee and conducted following the declaration of Helsinki. All subjects signed an informed consent prior to their inclusion in the study.

#### Demographic and Clinical Data

Demographic data including age, gender, weight, height, body mass index, past medical history, and location and nature of the symptoms was collected. An 11-point numerical pain rate scale (0: no pain; 10: maximum pain)

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was used to assess current intensity of pain, worst and lowest intensity of pain experienced in the preceding week [13].

## Self-Rated Disability

The Roland–Morris Activity scale contains 24 items reflecting the limitation in different activities of daily living attributed by the patient to LBP [14]. Patients were asked to mark whether the pain affects these functional activities the day of assessment. Only the "yes" items are counted for the score. Therefore, a higher score indicates a higher functional disability (0–24). In the current study, the Spanish version of the Roland–Morris Activity questionnaire was used [15].

# Sleep Quality Assessment

The Pittsburgh Sleep Quality Index (PSQI) is the most common used standardized questionnaire for assessment of sleep quality [16]. The PSQI consists of 19 self-reported questions and 5 questions answered by bedmates/ roommates. Items include recording usual bed time, usual wake time, number of actual hours slept, and number of minutes to fall asleep, as well as forced-choice Likert-type responses (0–3). The sum of the scores for the components yields one global score (0–21) where higher score indicates worse sleep quality [17]. A total PSQI score >8.0 indicates poor sleep quality [18].

# TrP Examination

TrPs were bilaterally explored in the quadratus lumborum, iliocostalis lumborum, psoas, piriformis, gluteus minimus, and gluteus medius muscles by an assessor with 9 years of experience in TrPs diagnosis. The order of evaluation was randomized between participants with a 2-minute rest period between muscles.

TrP diagnosis was performed following the criteria described by Simons et al. [5]: 1) presence of a palpable taut band in a skeletal muscle; 2) presence of a hyperirritable spot in the taut band; and 3) presence of referred pain in response to TrP compression. TrPs were considered active when local and referred pain reproduced any clinical pain symptom perceived by the subjects and the subject recognized the pain as familiar. TrPs were considered latent when the elicited local and referred pain did not reproduce any symptom familiar to the subject [5]. These criteria, when applied by a trained assessor, have shown a good interexaminer reliability (kappa) ranging from 0.84 to 0.88 [19].

TrP examination was conducted in a blinded fashion. After TrP assessment in all muscles, participants were asked for: "When I pressed each of these muscles, did you feel any pain locally and in other areas (referred pain). Please tell me whether the pain that you feel in the other area reproduced any symptom that you usually suffered from." Participants had to indicate whether the pain elicited by palpation reproduced their symptom (familiar pain) or another nonfamiliar pain.

## Analysis of Data

Data were analyzed with the SPSS statistical package (18.0 version; SPSS Inc., Chicago, IL, USA). Descriptive data were collected in all patients. The Kolmogorov-Smirnov test was used to analyze the normal distribution of the variables (P > 0.05). Quantitative data without a normal distribution (pain history, pain intensity, number of latent TrPs) were analyzed with nonparametric tests, and those data with a normal distribution (Roland-Morris, PSQI) were analyzed with parametric tests. Differences in the number of latent TrPs between groups were assessed with the nonparametric Mann-Whitney U-test. The chisquare  $(\chi^2)$  test was used to analyze differences in the distribution of TrPs for each muscle between both study groups. The unpaired student t-test was used to determine the differences in disability and quality of sleep between groups. The Spearman's rho (rs) test was used to determine the association between the number of TrPs and pain intensity, disability, and sleep quality. The statistical analysis was conducted at 95% confidence level, and a P value < 0.05 was considered statistically significant.

# Results

Forty-two individuals with chronic nonspecific LBP (50% women), aged 23–54 years old (mean age:  $45 \pm 10$  years), and 42 age- and sex-matched controls were included. No significant differences in any demographic data were observed between both groups (Table 1). Within the LBP group, 19 patients (45%) reported their right side as the most painful side, 17 (40%) reported their left side as the most painful side, and the remaining 6 (15%) reported bilateral pain. The mean duration of the history of pain was 9.1 years (95% confidence interval [CI] 6.8-11.3). The mean current intensity of LBP was 6.2 (95% CI 5.3-6.9), the worst intensity of pain experienced in the preceding week was 7.8 (95% Cl 7.2-8.4), whereas the lowest intensity of pain in the preceding week was 4.6 (95% CI 4.1-5.3). No significant association between clinical pain variables was found. As expected, individuals with nonspecific LBP exhibited a greater disability and worse sleep quality than controls (P < 0.001). Table 1 summarizes demographic and clinical data of patients with chronic nonspecific LBP and healthy controls.

The mean ± standard deviation (SD) number of TrPs for each patient with nonspecific LBP was  $5.5 \pm 1.9$  of which  $3.5 \pm 2.3$  were active TrPs and the remaining  $2.0 \pm 1.5$ were latent TrPs. Healthy controls only had latent TrPs (mean ± SD:  $1.0 \pm 1.5$ ). In fact, patients with nonspecific LBP showed a greater number of latent TrPs than healthy controls (z = -3.611; P < 0.001). The distribution of TrPs in the analyzed muscles was significantly different between patients with nonspecific LBP and controls for the quadratus lumborum, the iliocostalis lumborum, the piriformis, and the gluteus medius muscles (all, P < 0.001), but not for the psoas (P = 0.110) and gluteus minimus (P > 0.078) muscles. Active TrPs in the quadratus lumborum (45– 50%), iliocostalis lumborum, and gluteus medius (40– 45%) muscles were the most prevalent in patients with

	Nonspecific Low Back Pain (N = 42)	Healthy Controls $(N = 42)$	Significance
Gender (male/female)	21 / 21	21 / 21	NA
Age (years)	$45\pm8$	$45\pm9$	<i>t</i> = 0.105; <i>P</i> = 0.988
Weight (kg)	71 ± 12	72 ± 11	<i>t</i> = 0.182; <i>P</i> = 0.856
Height (cm)	$169 \pm 8$	170 ± 8	t = 0.311; P = 0.782
BMI (kg/cm <sup>2</sup> )	$24.5 \pm 3.2$	$24.9 \pm 3.4$	t = 0.365; P = 0.743
Roland-Morris (0-24)*	$12.9 \pm 2.5$	$0.8 \pm 0.2$	<i>t</i> = -27.950; <i>P</i> < 0.001
PSQI (0-21)*	$12.4\pm3.3$	$3.5\pm2.3$	<i>t</i> = -14.263; <i>P</i> < 0.001

 Table 1
 Demographic and clinical data of patients with nonspecific low back pain and healthy subjects

\* Indicated statistically significant difference between groups.

Data are expressed as means  $\pm$  standard deviation.

BMI = body mass index; PSQI = Pittsburgh Sleep Quality Index.

nonspecific LBP. Table 2 details the distribution of active TrPs for all muscles in patients with nonspecific LBP whereas Table 3 summarizes the presence of latent TrPs for all muscles in both patients with nonspecific LBP and healthy controls.

A significant positive association between the number of active TrPs and the mean intensity of pain episode

**Table 2** Number (N) and percentage of patients with nonspecific low back pain (N = 42) with active trigger points (TrPs) for each muscle

Muscle	More Painful Side	Less Painful Side
Quadratus lumborum	23 (55%)	19 (45%)
Iliocostalis lumborum	16 (38%)	14 (33%)
Psoas	4 (10%)	2 (5%)
Piriformis	15 (35%)	12 (28%)
Gluteus minimus	5 (12%)	2 (5%)
Gluteus medius	15 (35%)	16 (38%)

Note: None of the healthy controls had active trigger points.

( $r_s = 0.602$ ; P < 0.001) was found: the higher the number of active TrPs, the greater the intensity of the current pain episode. Additionally, the number of active TrPs was also positively associated ( $r_s = 0.338$ ; P = 0.03) with the PSQI: the higher the number of active TrPs, the worse the sleep quality. An interesting finding was that the number of active TrPs was negatively associated ( $r_s = -0.571$ ; P < 0.001) with the number of latent TrPs: the higher the number of active TrPs, the lower the number of latent TrPs. No significant association between the number of active TrPs and the Roland–Morris questionnaire was found.

## Discussion

The current study found that the local and referred pain elicited by active TrPs in back and hip muscles contributed to pain symptoms in individuals with chronic nonspecific LBP. When the assessor applied pressure to active TrPs, patients reported: "Yes, this is exactly the pain that I usually feel." In fact, a higher intensity of the pain was associated with a greater number of active TrPs. In addition, individuals with nonspecific LBP exhibited higher disability and worse sleep quality than controls. Sleep quality was associated with a higher number of active TrPs.

**Table 3** Number (N) and percentage of patients with nonspecific low back pain (N = 42) and matched controls (N = 42) with latent trigger points (TrPs) for each muscle

Muscle	Patients with Low Back Pain $(N = 42)$		Healthy Controls (N = 42)	
	More Painful Side	Less Painful Side	Dominant Side	Nondominant Side
Quadratus lumborum*	6 (14%)	7 (17%)	0 (0%)	4 (10%)
lliocostalis lumborum*	8 (19%)	8 (19%)	0 (0%)	2 (5%)
Psoas	11 (26%)	15 (36%)	8 (19%)	11 (26%)
Piriformis*	9 (22%)	8 (19%)	0 (0%)	3 (7%)
Gluteus minimus	3 (7%)	5 (12%)	5 (12%)	4 (10%)
Gluteus medius*	5 (12%)	7 (17%)	2 (5%)	2 (5%)

\* Significant differences between patients with nonspecific LBP and controls (all, P < 0.001).

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No association between disability and active TrPs was reported.

The presence of active TrPs in patients with chronic LBP has been previously reported [7-9]; however, these studies included patients with underlying pathologies explaining their LBP symptoms. Our study is the first one investigating the presence of active TrPs in patients with nonspecific LBP without an underlying medical condition explaining the symptoms. The current study found that the quadratus lumborum, the iliocostalis lumborum, and the gluteus medius muscles were the most affected by TrPs in patients with chronic nonspecific LBP, which agrees with previous findings [7,8]. It is interesting to note that patients with nonspecific LBP showed active TrPs in a similar pattern than individuals with postlaminectomy pain syndrome [7]. It is possible that similar nociceptive mechanisms are activated in individuals with LBP and, therefore, active TrPs are present in similar muscles. The presence of active TrPs in individuals with nonspecific LBP could perpetuate sensory and motor symptoms found in this population and hence contributing to sensitization mechanisms [20]. The fact that a greater number of active TrPs was associated with higher intensity of the pain episode supports a relevant role of the pain elicited by active TrPs in the development of pain symptoms in patients with nonspecific LBP. Obviously, other trigger factors can be also present at the same time.

Similarly than in previous studies conducted in individuals with tension-type headache [21,22], lateral epicondylalgia [23], or shoulder impingement [24], individuals with non-specific LBP also exhibited a greater number of latent TrPs than healthy controls. The presence of some latent TrPs in healthy asymptomatic people is expected, although in a lesser extent than in patients. In fact, the clinical relevance of latent TrPs has recently increased [25] as latent TrPs disturb normal pattern of motor recruitment and movement efficiency [26] and induce sensitization mechanisms [27]. It is possible that latent TrPs can also play a relevant role in motor symptoms found in individuals with nonspecific LBP.

An interesting finding was that the number of active TrPs was associated with a lower number of latent TrPs in patients with nonspecific LBP. As latent TrPs may become active with overload, stress, body posture, other somatic dysfunctions, fear to motion [28], it is possible that latent TrPs become active with time. In fact, our group of patients with nonspecific LBP has a history of pain episodes from 9 years. Future longitudinal studies are needed to confirm or refute this hypothesis.

We also showed that our group of patients exhibited moderate disability and that 88% (N = 37) patients reported poor sleep quality (PSQI > 8). These data are similar to those previously reported [11,12]. Disability was not associated with the number of active TrPs. This lack of association can be explained for the presence of high levels of pain associated with mild/moderate dis-

ability in our sample of patients with nonspecific LBP. It is also possible that individuals are habituated to the presence of pain, and their self-perceived disability was not associated with the intensity of the pain. In fact, we did not find such association between the intensity of the pain and disability. In addition, we observed an association between sleep quality and the number of active TrPs suggesting a possible interaction between these two factors. It has been previously reported a relationship between pain intensity and sleep quality in chronic LBP [12]. As the number of active TrPs was associated with the intensity of the pain and sleep quality, it is possible that a complex interaction among these factors is present in patients with nonspecific LBP. Current results suggest that sleep disorders and active TrPs can contribute to symptoms of individuals with nonspecific LBP by different and multiple mechanisms. Whether cause or consequence, sleep disorders must be taken into account in the overall management of these patients, in the same way as active TrPs, pain, and disability. It seems essential to address these aspects as an integral part of the evaluation and treatment of individuals with nonspecific LBP to achieve a positive outcome.

We should recognize potential limitations to the current study. First, the cross-sectional nature of the study does not permit to establish a cause and effect relationship between pain, TrPs, disability, and sleep quality. A randomized clinical trial where TrP interventions are applied to individuals with nonspecific LBP would help to clarify their etiological role in this pain syndrome. Second, we included patients with chronic nonspecific LBP, so our results should not be extrapolated to patients with acute nonspecific LBP. Third, we did not also collect data on catastrophizing, anxiety, fear, uncomfortable sleep position, medication side effects, or workplace features. All these variables are relevant in patients with LBP [29]. Future longitudinal studies with larger sample sizes and including these related outcomes are required to confirm a relationship between active TrPs, pain, and sleep quality in individuals with nonspecific LBP. Finally, we recognize that some TrPs can be more difficult or less reliable to palpation, at least in the low back [30]. For instance, manual palpation of the psoas muscle is more difficult than that of the gluteus medius muscle. It is interesting to note that active TrPs in the psoas muscles were the lowest prevalent.

## Conclusions

This study found that the local and referred pain elicited by active TrPs in back and hip muscles contributes to pain symptoms in individuals with nonspecific LBP. Higher pain intensity was associated with a greater number of active TrPs. Patients with nonspecific LBP exhibited higher disability and worse sleep quality than controls. Sleep quality, but not self-reported disability, was associated with a higher number of active TrPs. Our results suggest that active TrPs can contribute to sensory and motor symptoms in patients with chronic nonspecific LBP.

#### **Trigger Points in Low Back Pain**

## References

- 1 Andersson GB. Epidemiological features of chronic low back pain. Lancet 1999;354:581–5.
- 2 Walker B. The prevalence of low back pain: A systematic review of the literature from 1966 to 1998. J Spinal Disord 2000;13:205–17.
- 3 Dagenais S, Caro J, Haldeman S. A systematic review of low back pain cost of illness studies in the United States and internationally. Spine J 2008;8:8–20.
- 4 Wright A, Mayer TG, Gatchel RJ. Outcomes of disabling cervical spine disorders in compensation injuries: A prospective comparison to tertiary rehabilitation response to chronic lumbar spine disorders. Spine 1999;24:178–83.
- 5 Simons DG, Travell J, Simons LS. Myofascial Pain and Dysfunction. The Trigger Point Manual, Vol. 1, 2nd edition. Baltimore: Williams & Wilkins; 1999.
- 6 Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vitro microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. J Appl Physiol 2005;99:1977–84.
- 7 Teixeira M, Yeng LT, Garcia O, et al. Failed back surgery pain syndrome: Therapeutic approach descriptive study in 56 patients. Rev Assoc Med Bras 2011;57:282–7.
- 8 Chen CK, Nizar AJ. Myofascial pain syndrome in chronic back pain patients. Korean J Pain 2011; 24:100-4.
- 9 Samuel S, Peter A, Ramanathan K. The association of active trigger points with lumbar disc lesions. J Musculoskeletal Pain 2007;15(2):11–8.
- 10 Itoh K, Katsumi Y, Kitakoji H. Trigger point acupuncture treatment of chronic low back pain in elderly patients: A blinded RCT. Acupunct Med 2004;22: 170–7.
- 11 Marty M, Rozenberg S, Duplan B, et al. Quality of sleep in patients with chronic low back pain: A case-control study. Eur Spine J 2008;17:839–44.
- 12 Marin R, Cyhan T, Miklos W. Sleep disturbance in patients with chronic low back pain. Am J Phys Med Rehabil 2006;85:430–5.
- 13 Jensen MP, Turbner JA, Romano JM, Fisher L. Comparative reliability and validity of chronic pain intensity measures. Pain 1999;83:157–62.
- 14 Roland M, Morris R. A study of the natural history of back pain. Part I: Development of a reliable and sensitive measure of disability in low-back pain. Spine 1983;18:141–4.

- 15 Kovacs FM, Llobera J, Gil Del Real MT, et al. Validation of the Spanish version of the Roland-Morris questionnaire. Spine 2002;27:538–42.
- 16 Cole JC, Dubois D, Kosinski M. Use of patientreported sleep measures in clinical trials of pain treatment: A literature review and synthesis of current sleep measures and a conceptual model of sleep disturbance in pain. Clin Ther 2007;29:2580–8.
- 17 Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193–213.
- 18 Carpenter JS, Andrykowski MA. Psychometric evaluation of the Pittsburgh Sleep Quality Index. J Psychosom Res 1998;45:5–13.
- 19 Gerwin RD, Shanon S, Hong CZ, Hubbard D, Gevirtz R. Interrater reliability in myofascial trigger point examination. Pain 1997;69:65–7.
- 20 O'Neill S, Manniche C, Graven-Nielsen T, Arendt-Nielsen L. Generalized deep-tissue hyperalgesia in patients with chronic low-back pain. Eur J Pain 2007;11:415–20.
- 21 Fernández-de-las-Peñas C, Ge H, Arendt-Nielsen L, Cuadrado ML, Pareja JA. Referred pain from trapezius muscle trigger point shares similar characteristics with chronic tension type headache. Eur J Pain 2007;11: 475–82.
- 22 Fernández-de-las-Peñas C, Ge H, Arendt-Nielsen L, Cuadrado ML, Pareja JA. The local and referred pain from myofascial trigger points in the temporalis muscle contributes to pain profile in chronic tension-type headache. Clin J Pain 2007;23:786–92.
- 23 Fernández-Carnero J, Fernández-de-las-Peñas C, De-la-Llave-Rincón Al, Ge HY, Arendt-Nielsen L. Prevalence of and referred pain from myofascial trigger points in the forearm muscles in patients with lateral epicondylalgia. Clin J Pain 2007;23:353– 60.
- 24 Hidalgo-Lozano A, Fernández-de-las-Peñas C, Alonso Blanco C, et al. Muscle trigger points and pressure pain hyperalgesia in the shoulder muscles in patients with unilateral shoulder impingement: A blinded, controlled study. Exp Brain Res 2010;202: 915–25.
- 25 Ge HY, Arendt-Nielsen L. Latent myofascial trigger points. Curr Pain Headache Rep 2011;15:386– 92.
- 26 Lucas KR. The impact of latent trigger points on regional muscle function. Curr Pain Headache Rep 2008;12:344–9.

## Iglesias-González et al.

- 27 Xu YM, Ge HY, Arendt-Nielsen L. Sustained nociceptive mechanical stimulation of latent myofascial trigger point induces central sensitization in healthy subjects. J Pain 2010;11:1348–55.
- 28 Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. J Electromyogr Kinesiol 2004;14:95–107.
- 29 Roelofs J, Goubert L, Peters ML, Vlaeyen J, Crombez G. The Tampa Scale for Kinesiophobia: Further

examination of psychometric properties in patients with chronic low back pain and fibromyalgia. Eur J Pain 2004;8:495–502.

30 Hsieh CYJ, Hong CZ, Adams AH, et al. Inter-examiner reliability of the palpation of trigger points in the trunk and lower limb muscles. Arch Phys Med Rehabil 2000;81:258–64. Copyright of Pain Medicine is the property of Wiley-Blackwell and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.