Association between muscle trigger points, ongoing pain, function, and sleep quality in elderly women with bilateral painful knee osteoarthritis

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Abstract

Objectives: 1, to investigate if the referred pain elicited by active TrPs reproduces the symptoms in individuals with painful knee osteoarthritis (OA); and, 2, to determine the relationship between the presence of active TrPs, intensity of ongoing pain, function, quality of life and sleep quality in individuals with painful knee OA.

Methods: Eighteen women with bilateral painful knee OA, aged 79 to 90 years, and 18 matched controls participated. Muscle TrPs were bilaterally explored in several muscles of the lower extremity. Trigger points were considered active if the elicited referred pain reproduced knee symptoms, and TrPs were considered latent if the elicited pain did not reproduce symptoms. Pain was collected with a numerical pain rate scale (NPRS, 0-10), function was assessed with Western Ontario and McMaster Universities (WOMAC), quality of life was assessed with the Medical Outcomes Study Short Form 36 (SF-36) questionnaire, and sleep quality was determined with the Pittsburgh Sleep Quality Index (PSQI).

Results: Women with knee OA exhibited a greater number of active TrPs (mean: 1±1, $P<0001$) but similar number of latent TrPs (mean: 4±2) than healthy women (mean: 4±3, $P=0.613$). A greater number of active TrPs was associated with higher intensity of ongoing pain ($r=0.605$; $P=0.007$). Higher intensity of ongoing knee pain was associated with lower physical function ($P<0.05$).

Conclusions: The referred pain elicited by active TrPs in the lower extremity muscles contributed to pain symptoms in painful knee OA. A higher number of active TrPs was associated with higher intensity of ongoing knee pain.

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Introduction

Osteoarthritis (OA) is the most common form of arthritis, affecting a higher number of people in ageing populations.[1] The estimated incidence of symptomatic knee OA ranges from 0.37% per year for males to 1.02% per year for females in the USA, and about 9.3% of the American population is diagnosed with symptomatic knee OA by age 60.[2]

OA is mainly featured by cartilage joint degradation, change in subchondral and marginal bone, synovial inflammation, and capsular thickening.[3] Individuals at different stages present with pain, but it is estimated that up to 40% of patients with radiological damage do not report pain and that there is poor relationship between imaging findings and sensory symptoms.[4]

Recent studies have reported that spreading sensitization is particularly apparent in individuals with knee OA with high levels of pain in absence of moderate to severe radiographic findings.[5,6] The presence of nociceptive changes in OA-related pain can be one of the many mechanisms explaining discrepancies between imaging and clinical features.

One feature of spreading sensitization is the presence of muscle hyperalgesia.[7] Bajaj et al. reported that patients with knee OA exhibited larger pain areas and higher muscle referred pain intensity after infusion of hypertonic saline in the leg muscles in comparison with healthy controls.[8] The presence of larger muscular referred pain areas after experimental stimulation has been related to spreading sensitization mechanisms in

other local pain disorders, e.g., lateral epicondylalgia,[9] temporomandibular disorders,[10] or tension type headache.[11] In addition, the presence of referred pain areas elicited by trigger points (TrPs) has been also associated to sensitization mechanisms in the same conditions.[12-14] In fact, it has been previously proposed that TrPs can be involved in pain processes in subjects with knee pain.[15] Trigger points (TrPs) are usually defined as hypersensitive tender spots within taut bands of skeletal muscles that are painful on muscle stimulation and that usually elicit referred pain.[16] Trigger points are clinically classified as active or latent. Active TrPs reproduce the patient's symptoms and the elicited referred pain during stimulation is recognized as a familiar phenomenon for the patient. Latent TrPs are not responsible of any symptom of the patient and the pain is not recognized as a familiar phenomenon. This clinical distinction between active and latent TrPs is substantiated by histochemical findings because higher levels of several chemical mediators and algogenic substances (i.e. bradykinin, serotonin, or substance P) were found in active TrPs as compared to latent TrPs and non-TrPs.[17]

There is scarce scientific information related to the presence of TrPs in patients with knee OA. An old case series found that treatment of TrPs in the semimembranosus muscle resulted in relief of persistent knee pain but without clarifying the cause of the pain.[18] Bajaj et al analyzed the presence of TrPs in a small number of patients with hip, knee and multiple joint OA and reported that OA-related pain was associated to latent, but not active, TrPs.[19] Further, there is preliminary evidence showing that treatment of active TrPs is effective for reducing symptoms in patients with knee OA.[20,21] However, no previous study has systematically investigated the presence of active TrPs in patients with knee OA and their relationship with other outcomes including knee pain, function, quality of life or sleep quality.

Therefore, the primary aims of the current study were: 1, to investigate if the referred pain elicited by active TrPs reproduces the symptoms in subjects with painful knee OA; and, 2, to determine the relationship between the presence of active TrPs, the intensity of ongoing pain, function, quality of life and sleep quality in individuals with painful knee OA.

Methods

Participants

For this study, women diagnosed with painful knee OA from a clinic of physical therapy associated to a geriatric center participated. Knee OA was diagnosed according to American College of Rheumatology (ACR) classification.[22] Clinical data including intensity and duration of the symptoms, radiological evaluation, and medication were collected. All patients had bilateral weight bearing, fixed flexion posterior-anterior and lateral X-rays of both knee regions. Degenerative profiles of the tibio-femoral joint were verified for each patient. Patient should exhibit moderate pain intensity with a numerical pain rate scale (NPRS, 0-10) score ≥ 3points and bilateral symptoms.

Participants were excluded if presented any of the following: 1) previous lower extremity surgery; 2) diagnosis of lower extremity radiculopathy or myelopathy; 3) any sensory dysfunction including nerve damage; 4) if they had received any therapeutic intervention in the past 6 months before the study; or, 5) if they were mentally impaired (Mini-Mental State Examination, MMSE<20/35).[23]

In addition, age-and gender-matched volunteers who had no knee pain or other lasting pain problems in the past year were included as healthy controls and recruited from the general population.

Participants were requested not to take any analgesic medication 24h before the examination. The study protocol was approved by human research committee of the Universidad Salamanca, Spain (registry 201200003354). Subjects signed an informed consent prior to participation in the study.

Demographic and clinical data

Demographic data including age, gender, weight, height, body mass index, past medical history and location of the symptoms were collected. An 11-point numerical pain rate scale (NPRS, 0: no pain; 10: maximum pain) was used to determine the mean intensity of knee pain experienced in the preceding 24h.[24,25] Since patients exhibited bilateral knee pain, the mean of both sides was used for the analysis.

The functional status of the patients was evaluated using the Western Ontario and McMaster Universities (WOMAC) index. The WOMAC consists of a self-administered questionnaire reflecting 3 dimensions: pain (5 items), stiffness (5 items), and physical function (17 items) in patients with OA in the lower extremity.[26] Each subscale ranges from 0 to 20 (pain), 0-8 (stiffness), and 0-68 (function) points. A higher WOMAC score represents greater limitation. This questionnaire has shown high reliability (ICC: 0.92-0.97) in patients with knee OA.[27] In the current study we used the validated Spanish version of the WOMAC which is a valid, reliable and responsive instrument in patients with knee OA.[28]

The health-related quality of life was assessed with the Medical Outcomes Study Short Form 36 (SF-36) questionnaire.[29] This questionnaire assesses 8 domains: physical functioning, role physical, bodily pain, general health, vitality, social functioning, role-emotional, and mental health. Each domain ranges from 0 (lowest level of functioning) to

100 (highest level of functioning) points.[30] The SF-36 questionnaire has shown the best ability to discriminate between subjects with health problems and healthy people.[31]

Sleep quality assessment

The Pittsburgh Sleep Quality Index (PSQI) is the most common used standardized questionnaire for the comprehensive assessment of sleep quality.[32] It consists of 19 self-rated questions and 5 questions answered by bedmates/roommates, if available. PSQI items use varying response categories that include recording usual bed time, usual wake time, number of hours slept, and number of minutes to fall asleep. The sum of the scores for the components yields one global score (0-21 point) where higher scores indicate worse sleep quality.[33] Buysse et al reported that the PSQI has good internal consistency (α: 0.83) and test-retest reliability (r: 0.85). A total score > 8.0 points has been found to be indicative of poor sleep quality.[34]

Trigger point (TrP) examination

Trigger points were bilaterally explored in the tensor fasciae latae, sartorius, rectus femoris, vastus lateralis, vastus medialis, gracilis, biceps femoris, semitendinosus, gastrocnemius and tibialis anterior muscles by an assessor with 9 years of experience in TrP diagnosis. The order of evaluation was randomized between subjects with a 2 minutes rest period between muscles.

Trigger Points (TrP) diagnosis was performed following the criteria described by Simons et al:[16] 1, presence of a hypersensitive tender spot within a palpable taut band (if possible); 2, local twitch response elicited by the snapping palpation of the taut band (when possible to palpation); and, 3) presence of referred pain in response to manual compression. Trigger points were considered active when the local and referred pain reproduced any pain symptom perceived by the subject and the subject recognized this

evoked pain as familiar. In the current study, TrPs were considered active when their elicited referred pain reproduced knee pain symptoms. Trigger points were considered latent when the elicited local and the referred pain did not reproduce any symptom experienced by the subject, in our study, in the knee area.[16] These criteria, when are applied by trained and experienced assessors have shown to exhibit good inter-examiner reliability (kappa: 0.84-0.88).[35]

Trigger point (TrP) examination was conducted as follows. After palpation of TrPs in all muscles, participants were asked for: "When I palpated this muscle, did you feel pain locally and/or in another distant area?" When the answer was positive, the subject was asked for: "Please tell me whether the pain that you feel during the manual examination reproduced any symptom that you usually suffered from in the knee area." Participants had to indicate whether the pain elicited during manual examination of the musculature reproduced or not any symptom around the knee. Since patients included in this study were elderly, particular attention was given to a proper understanding of the procedure.

Statistical analysis

Data were analyzed with the SPSS statistical package (18.0 Version). Descriptive data was collected on all patients. The Kolmogorov-Smirnov test revealed that all data showed a normal distribution ($P>0.05$); therefore, parametric tests were used in the main analyses. Differences in the number of TrPs (total, active or latent TrPs) between groups were assessed with the unpaired student t-test. The Pearson correlation test (r) test was used to determine the association between the number of TrPs, pain intensity, function (each subtest of the WOMAC), quality of life (each dimension of the SF-36), and sleep quality (PSQI). The unpaired student t-test was used for assess differences in clinical features, function (WOMAC), quality of life (SF-36), and sleep quality (PSQI) between

groups. The statistical analysis was conducted at 95% confidence level. A P value less than 0.05 was considered statistically significant.

Results

Eighteen women with painful knee OA, aged 79 to 90 years old, and 18 matched controls, aged 79 to 90 years were included. No differences in any demographic data were observed between groups (**Table 1**). The mean intensity of knee pain, based on the NPRS, experienced the previous 24 hours of the OA group was 4.0 ± 1.2.

The mean ± SD number of TrPs for each women with painful knee OA was 5 ± 3, 1±1 were active TrPs and the remaining 4±2 were latent TrPs. Healthy controls only exhibited latent TrPs (mean ± SD: 4 ± 3). Therefore, the number of TrP between both groups was significantly different for active TrPs ($t = 3.796$; $P<0.001$), but not for latent TrPs ($t=-0.510$; $P=0.613$): women with painful knee OA showed a greater number of active TrPs and similar number of latent TrPs than healthy women. Active TrPs in the vastus lateralis, vastus medialis and gastrocnemius muscles were the most prevalent in women with painful knee OA. **Table 2** summarizes the distribution of active and latent TrPs for all muscles in both women with painful knee OA and healthy women.

A significant positive association between the number of active TrPs and the intensity of pain was found in women with painful knee OA ($r=0.605$; $P=0.007$): the greater the number of active TrPs, the higher the intensity of ongoing pain. No other significant association between the number of latent or active muscle TrPs and function, quality of life or sleep quality was found.

Women with painful knee OA exhibited worse physical function (WOMAC and SF-36 domains, $P<0.01$) and greater stiffness (WOMAC, $P<0.001$) than healthy women. No differences in the remaining domains of the SF-36 questionnaire and quality of sleep

(PSQI) were found between groups. **Table 1** summarizes scores of the WOMAC, SF-36 and PSQI questionnaires of women with knee OA and healthy women. Significant correlations between the intensity of ongoing knee pain and function were found in both WOMAC (r=0.608, P=0.008) and SF-36 (r =-0.654, P=0.03) questionnaires: the greater the intensity of knee pain, the lower the physical function.

Discussion

The current study found that women with painful knee OA exhibited latent and active TrPs and that the referred pain elicited by some TrPs, those which were active, reproduced the knee pain symptoms. No differences in the presence of latent TrPs were found between women with painful knee OA and healthy women. The presence of active TrPs was associated with the intensity of ongoing knee pain. In addition, women with knee OA exhibited worse physical function than healthy women and it was negatively associated with the intensity of knee pain.

Some authors have previously suggested the relevance of muscle TrPs in knee pain;[15,16,18-21] however, no previous study has systematically investigated the presence of active and latent TrPs in women with painful knee OA. Our study found that active TrPs in the vastus lateralis, vastus medialis and gastrocnemius muscles were the most prevalent in women with painful knee OA. The referred pain elicited during manual examination by these TrPs reproduced the knee symptoms and the number of active TrPs was associated with the intensity of ongoing knee pain. Our results agree with a previous study where all individuals with knee OA waitlisted for total knee arthroplasty showed TrPs within the vastus lateralis and gastrocnemius muscles.[21] The presence of active TrPs in these muscles seems expected since the vastus medialis, vastus lateralis and the gastrocnemius muscles are intrinsically related to knee joint load and stabilization. It is

possible that patients with knee OA adapt their dynamic loads during walking or daily life activities inducing a muscle overload and promoting the activation of TrPs.[36] The fact that experimental muscle pain induced in knee muscles exerts generalized muscle inhibition supports this hypothesis.[37] Our findings would support the relevance of TrPs referred pain in OA-related knee pain; nevertheless, the presence of active TrPs was scarce. This hypothesis is partially supported by the fact that inactivation of active TrPs is effective for pain relief in these patients.[20,21] Future studies should investigate the etiologic role of muscle referred pain elicited by active TrPs in the development of knee OA pain symptoms.

An interesting finding was that women with painful knee OA exhibited a similar number of latent TrPs than healthy women. In fact, the number of latent muscle TrPs was unexpected high as compared to the number of active TrPs. These findings have been previously reported in published studies conducted in individuals with mechanical neck pain[38,39] but contrary to others conducted in individuals with whiplash injury,[40] tension type headache,[41,42] lateral epicondylalgia[43,44] or shoulder impingement[45] where healthy controls exhibited lower number of latent TrPs than the patient populations. The presence of some latent TrPs in asymptomatic people should be expected since muscles can experience repetitive overload during normal daily live activities[46]; however, it is also expected that this prevalence would be lower than in the symptomatic population. Discrepancies can be related to specific areas of the body, muscles investigated or the elder age of the sample. The presence of latent, but not active, TrPs in our control group supports that we were able to find an appropriate comparison group considering the age of our sample. It is important to note that the clinical relevance of latent TrPs has increased with recent studies[46] demonstrating that latent muscle TrPs can disturb normal

pattern of motor recruitment and movement efficiency[47] and may induce sensitization mechanisms.[48] Therefore, it is possible that motor disturbances induced by latent muscle TrPs also contributed to motor symptoms experienced by patients with knee OA; although this should be investigated in future studies.

We also showed that our group of women with painful knee OA exhibited lower physical function than healthy women which is expected and highly documented in the literature.[49,50] Surprisingly, we did not find differences in sleep quality or quality of life, probably because the small sample size. In fact, previous studies including higher sample sizes found that sleep disturbances are related to pain mechanisms in subjects with knee OA.[51,52] Further, we also found an association between physical function and the intensity of knee pain experienced the previous 24h. Since the intensity of knee pain was positively associated with the presence of higher number of active TrPs and worse physical function, it is plausible a complex clinical interaction between all these factors. Current results would suggest that it seems essential to address these different aspects as an integral part of the evaluation and treatment of individuals with painful knee OA.

We should recognize some limitations to the current study. Firstly, the sample size was small which may explain the lack of significance in some outcomes; however, it is difficult to get a proper sample size of elder women with painful knee OA without other comorbid conditions. Second, the inclusion of a control group is highly difficult in elder people. Although we included age-and gender-matched volunteers without knee pain or other lasting pain problems in the past year, this group also showed scores in the WOMAC, which can be attributed to the old age of the participants. Nevertheless, based on the results we believe that we were able to find an appropriate comparison group considering the age of our sample. Third, the cross-sectional nature of the study does not permit to establish a cause and effect relationship. Fourth, we did not also collect data on

catastrophizing, anxiety, fear, or medication side effects. In addition, we should consider that we assessed pain experienced in the preceding 24 hours; which can be a short period of time considering that patients were chronic. Future longitudinal studies with larger sample sizes and including all these related outcomes are required to further confirm a relationship between active TrPs, physical function and OA-related knee pain.

Conclusions

This study found that the referred pain elicited by active TrPs reproduced knee pain symptoms in women with painful knee OA. The presence of the number of active TrPs was associated with the intensity of ongoing pain. No differences in the presence of latent TrPs were found between women with painful knee OA and healthy women. Women with knee OA also exhibited worse physical function than healthy women and it was negatively associated with the intensity of ongoing knee pain.

References

1. Hochberg MC, Hunter DJ, Felson DT. Osteoarthritis. Br Med J 2006; 332: 640-2
2. Losina E, Weinstein AM, Reichmann WM, Burbine SA, Solomon DH, Daigle ME et al. Lifetime risk and age of diagnosis of symptomatic knee osteoarthritis in the US. Arthritis Care Res 2013; 65(5): 703-11
3. Kidd BL. Osteoarthritis and joint pain. Pain 2006; 123: 6-9.
4. Bedson J, Croft PR. The discordance between clinical and radiographic knee osteoarthritis: a systematic search and summary of the literature. BMC Musculoskelet Disord 2008, 9:116-126.
5. Finan PH, Buenaver LF, Bounds SC, Hussain S, Park RJ, Haque UJ et al. Discordance between pain and radiographic severity in knee osteoarthritis: findings from quantitative sensory testing of central sensitization. Arthritis Rheum 2013; 65(2): 363-72.
6. Suokas AK, Walsh DA, McWilliams DF, Condon L, Moreton B, Wylde V et al. Quantitative sensory testing in painful osteoarthritis: a systematic review and meta-analysis Osteoarthritis Cartilage 2012; 20: 1075-85.
7. Arendt-Nielsen L, Nie H, Laursen MB, Laursen BS, Madeleine P, Simonsen OH et al. Sensitization in patients with painful knee osteoarthritis. Pain 2010; 149: 573-81.
8. Bajaj P, Bajaj P, Graven-Nielsen T, Arendt-Nielsen L. Osteoarthritis and it association with muscle hyperalgesia: an experimental controlled study. Pain 2001; 93: 107-14.
9. Slater H, Arendt-Nielsen L, Wright A, Graven-Nielsen T. Sensory and motor effects of experimental muscle pain in patients with lateral epicondylalgia and controls with delayed onset muscle soreness Pain 2005; 114: 118-130.

10. Svensson P, List T, Hector G. Analysis of stimulus-evoked pain in patients with myofascial temporomandibular pain disorders. Pain 2001; 92: 399-409.
11. Schmidt-Hansen PT, Svensson P, Bendtsen L, Graven-Nielsen T, Bach F. Increased muscle pain sensitivity in patients with tension-type headache. Pain 2007; 129; 113-21.
12. Fernández-Carnero J, Fernández-de-las-Peñas C, De-la-Llave-Rincón AI, 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.
13. Fernández-de-las-Peñas C, Galán-del-Río F, Alonso-Blanco C, Jiménez-García R, Arendt-Nielsen L, Svensson P. Referred pain from muscle trigger points in the masticatory and neck-shoulder musculature in women with temporomandibular disorders. J Pain 2010; 11: 1295-304.
14. Fernández-de-las-Peñas C, Cuadrado ML, Arendt-Nielsen L, Simons DG, Pareja JA. Myofascial trigger points and sensitization: an updated pain model for tension type headache. Cephalalgia 2007; 27: 383-93.
15. Galletti R, Obletter G, Giamberardino MA, Formica LM, Cicchitti G, Vecchiet L et al. Pain in osteoarthritis of the knee. Adv Pain Res Ther 1990; 13: 183-90.
16. Simons DG, Travell J, Simons LS. Myofascial pain and dysfunction. The trigger point manual. Volume 1 (2nd edition). Baltimore: Williams & Wilkins, 1999.
17. 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.
18. Weiser HI. Semimembranosus insertion syndrome: a treatable and frequent cause of persistent knee pain. Arch Phys Med Rehabil 1979; 60: 317-9.

19. Bajaj P, Bajaj P, Graven-Nielsen T, Arendt-Nielsen L. Trigger Points in patients with lower limb osteoarthritis. J Musculosketal Pain 2001; 9 (3): 17-33.
20. Itoh K, Hirota S, Katsumi Y, Ochi H, Kitakoji H. Trigger point acupuncture for treatment of knee osteoarthritis: a preliminary RCT for a pragmatic trial. Acupunct Med 2008; 26: 17-26.
21. Henry R, Cahill CM, Wood G, Hroch J, Wilson R, Cupido T, Vandenkerkhof E. Myofascial pain in patients waitlisted for total knee arthroplasty. Pain Res Manag 2012; 17: 321-7.
22. Altman R, Asch E, Bloch D, Borenstein D, Brandt K, Christy W et al. Development of criteria for the classification and reporting of osteoarthritis: Classification of osteoarthritis of the knee: Diagnostic and Therapeutic Criteria Committee of the American Rheumatism Association. Arthritis Rheum 1986; 29: 1039-49.
23. Crum RM, Anthony JC, Bassett SS, Folstein MF. Population based norms for the Mini-Mental State Examination by age and educational level. JAMA 1993; 269: 2386-91
24. McCaffery M, Beebe A. Pain: Clinical manual for nursing practice, Mosby St. Louis, MO; 1989
25. Brunelli Z, Zecca E, Martini C et al. Comparison of numerical and verbal rating scales to measure pain exacerbations in patients with chronic cancer pain. Health Qual Life Outcomes 2010; 8: 42.
26. McConnell S, Kolopack P, Davis AM. The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC): a review of its utility and measurement properties. Arthritis Rheum 2001; 45:453-61.

27. Williams VJ, Piva SR, Irrgang JJ, Crossley C, Fitzgerald GK. Comparison of reliability and responsiveness of patient-reported clinical outcome measures in knee osteoarthritis rehabilitation. J Orthop Sport Phys Ther 2012; 42: 716-23.
28. Escobar A, Quintana JM, Bilbao A, Azkárate J, Güenaga JI. Validation of the Spanish version of the WOMAC questionnaire for patients with hip/knee osteoarthritis: Western Ontario and McMaster Universities Osteoarthritis Index. Clin Rheumatol 2002; 21:466-71.
29. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Med Care 1992; 30: 473-83.
30. Ware JE, Snow KK, Kosinski M, Gandek B. SF-36 Health Survey: manual and interpretation guide, Boston, MA: New England Medical Center, The Health Institute, 1993.
31. McHorney CA, Ware JE, Raczek AE. The MOS 36-item short-form health survey (SF-36). II. Psychometric and clinical tests of validity in measuring physical and mental health construct. Med Care 1993; 31: 247-63.
32. Cole JC, Dubois D, Kosinski M. Use of patient-reported 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.
33. 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.
34. Carpenter JS, Andrykowski MA. Psychometric evaluation of the Pittsburgh Sleep Quality Index. J Psychosom Res 1998; 45: 5-13.
35. Gerwin RD, Shanon S, Hong CZ, Hubbard D, Gevirtz R. Interrater reliability in myofascial trigger point examination. Pain 1997; 69: 65-7.

36. Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. J Electromyogr Kinesiol 2004; 14: 95-107.
37. Henriksen M, Rosager S, Aaboe J, Graven-Nielsen T, Bliddal H. Experimental knee pain reduces muscle strength. J Pain 2011; 12: 460-7.
38. Fernández-de-las-Peñas C, Alonso-Blanco C, Miangolarra JC. Myofascial trigger points in subjects presenting with mechanical neck pain: a blinded, controlled study. Man Ther 2007; 12: 29-33.
39. Muñoz-Muñoz S, Muñoz-García MT, Alburquerque-Sendín F, Arroyo-Morales M, Fernández-de-las-Peñas C. Myofascial trigger points, pain, disability and sleep quality in individuals with mechanical neck pain. J Manipul Physiol Ther 2012; 35: 608-13.
40. Fernández-Pérez A, Villaverde-Gutiérrez C, Mora-Sánchez A, Alonso-Blanco C, Sterling M, Fernández-de-las-Peñas C. Muscle trigger points, pressure pain threshold, and cervical range of motion in patients with high level of disability related to acute whiplash injury. J Orthop Sports Phys Ther 2012; 42: 634-41.
41. 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.
42. 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.
43. Fernández-Carnero J, Fernández-de-las-Peñas C, De-la-Llave-Rincón AI, 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.

44. Fernández-Carnero J, Fernández-de-las-Peñas C, De-la-Llave-Rincón AI, Ge HY, Arendt-Nielsen L. Bilateral myofascial trigger points in the forearm muscles in chronic unilateral lateral epicondylalgia: A blinded controlled study. Clin J Pain 2008; 24: 802-7.
45. Hidalgo-Lozano A, Fernández-de-las-Peñas C, Alonso Blanco C, Ge HY, Arendt-Nielsen L, Arroyo-Morales M. 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.
46. Ge HY, Arendt-Nielsen L. Latent myofascial trigger points. Curr Pain Headache Rep 2011; 15: 386-92.
47. Lucas KR. The impact of latent trigger points on regional muscle function. Curr Pain Headache Rep 2008; 12: 344-9.
48. 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.
49. van Dijk GM, Dekker J, Veenhof C, van den Ende CH; Carpa Study Group.Course of functional status and pain in osteoarthritis of the hip or knee: a systematic review of the literature. Arthritis Rheum 2006; 55: 779-85
50. van Dijk GM, Veenhof C, Schellevis F, Hulsmans H, Bakker JP, Arwert H, Dekker JH, Lankhorst GJ, Dekker J. Co-morbidity, limitations in activities and pain in patients with osteoarthritis of the hip or knee. BMC Musculoskelet Disord 2008; 9: 95.
51. Hawker GA, French MR, Waugh EJ, Gignac MA, Cheung C, Murray BJ. The

multidimensionality of sleep quality and its relationship to fatigue in older adults with painful osteoarthritis. Osteoarthritis Cartilage 2010; 18: 1365-71.

52. Murphy SL, Lyden AK, Phillips K, Clauw DJ, Williams DA. Subgroups of older adults with osteoarthritis based upon differing comorbid symptom presentations and potential underlying pain mechanisms. Arthritis Res Ther 2011; 13: R135

Table 1: Demographic and clinical data of women with painful knee osteoarthritis (OA) and healthy women

	Knee osteoarthritis (n=18)	Healthy controls (n=18)	Significance
Age (years)	85 ± 4	85 ± 6	t = 0.914; P = 0.367
Weight (kg)	68.8 ± 9.1	64.3 ± 12.0	t = 1.207; P = 0.213
Height (cm)	152 ± 7	153 ± 7	t = -0.500; P = 0.620
BMI (kg/cm^2)	29.7 ± 3.8	27.4 ± 4.8	t = 1.610; P = 0.117
MMSE	26.1 ± 5.0	26.7 ± 4.4	t = -0.333; P = 0.741
WOMAC pain*	8.3 ± 3.9	3.9 ± 5.8	t = 2.650; P = 0.012
WOMAC stiffness*	3.9 ± 1.2	2.4 ± 1.6	t = 3.875; P < 0.001
WOMAC physical function*	43.2 ± 9.8	31.2 ± 13.0	t = 3.137; P = 0.004
SF36 physical function*	12.5 ± 12.5	33.1 ± 16.1	t = -4.262; P < 0.001
SF36 physical role	56.9 ± 40.0	73.6 ± 33.7	t = -1.351; P = 0.186
SF36 bodily pain	56.3 ± 22.7	61.5 ± 26.5	t = -0.640; P = 0.527
SF36 social function	79.9 ± 23.1	79.2 ± 18.6	t = 0.099; P = 0.922
SF36 mental health	55.8 ± 20.9	52.0 ± 22.5	t = 0.521; P = 0.606
SF36 emotional role	68.5 ± 37.0	57.4 ± 40.9	t = 0.854; P = 0.399
SF36 vitality	54.4 ± 17.9	53.6 ± 24.7	t = 0.116; P = 0.909
SF36 general health	60.6 ± 16.1	59.2 ± 21.8	t = 0.217; P = 0.830
PSQI	11.1 ± 5.0	9.5 ± 4.8	t = 0.939; P = 0.354

Data are expressed as means ± standard deviation; BMI: body mass index; MMSE: Mini-Mental State Examination; WOMAC: Western Ontario and McMaster Universities Arthritis Index; SF36: 36-Item Short Form Health Survey; PSQI: Pittsburgh Sleep Quality Index
* Indicated statistically significant difference between groups

Table 2: Number (n) of women with knee osteoarthritis and healthy women with muscle trigger points (TrPs).

	Women with painful knee osteoarthritis (n=18)									
	Tensor fasciae latae		**Sartorius muscle**		**Rectus femoris**		**Vastus lateralis**		**Vastus medialis**	
	Right Side	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**
Active TrPs (n)	0	0	1	2	1	1	2	2	2	1
Latent TrPs (n)	2	3	6	6	3	4	5	3	4	4
No TrPs (n)	16	15	11	10	14	13	11	13	10	13
	Gracilis muscle		**Biceps femoris muscle**		**Semitendinosus**		**Tibialis anterior**		**Gastrocnemius**	
	Right Side	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**
Active TrPs (n)	0	1	0	0	0	0	1	0	2	1
Latent TrPs (n)	5	5	0	1	2	3	3	5	5	3
No TrPs (n)	13	12	18	17	16	15	14	13	11	14
	Healthy women (n=18)									
	Tensor fasciae latae		**Sartorius muscle**		**Rectus femoris**		**Vastus lateralis**		**Vastus medialis**	
	Right Side	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**
Active TrPs (n)	0	0	0	0	0	0	0	0	0	0
Latent TrPs (n)	3	5	4	3	3	3	9	6	5	4
No TrPs (n)	15	13	14	15	15	15	9	12	13	14
	Gracilis muscle		**Biceps femoris**		**Semitendinosus**		**Tibialis anterior**		**Gastrocnemius**	
	Right Side	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**	**Right Side**	**Left Side**
Active TrPs (n)	0	0	0	0	0	0	0	0	0	0
Latent TrPs (n)	5	4	2	3	0	2	6	4	4	8
No TrPs (n)	13	14	16	15	18	16	12	14	14	10