ABCDDV/1216

ABCD Arq Bras Cir Dig
2016;29(Supl.1):107-110
DOI: /10.1590/0102-6720201600S10026

Review Article

# TENDINOPATHY AND OBESITY

*Tendinopatia e obesidade*

Adham do Amaral e **CASTRO**, Thelma Larocca **SKARE**, Paulo Afonso Nunes **NASSIF**, Alexandre Kaue **SAKUMA**, Wagner Haese **BARROS**

From the Programa de Pós-Graduação em Princípios da Cirurgia, Faculdade Evangélica do Paraná/Hospital Universitário Evangélico de Curitiba/Instituto de Pesquisas Médicas (Postgraduate Program in Principles of Surgery, Evangelic Faculty of Paraná/ University Evangelic Hospital of Curitiba/ Medical Research Institute), Curitiba, PR, Brazil

**HEADINGS** - Tendinopathy. Obesity. Overweight. Adipokines.

**ABSTRACT – *Introduction*:** Tendinopathies and tendon tears account for over 30% of all musculoskeletal consultations. Obesity, which is becoming one of the world´s most prevalent public health issues, may be associated with this condition. ***Objective*:** To review the literature about tendinopathies and obesity association. ***Methods*:** This is a descriptive exploratory study using the portal Medline. Literature in English language from 2006 to 2014 were reviewed. ***Results*:** The pathogenesis of tendinopathies includes inflammatory, regenerative and degenerative processes that happen simultaneously from early to late phases of the disease. Mechanical stress upon tendons seems to be one of the most important factors to initiate the inflammatory response, but it´s not the only one that can deflagrate it: there are other extrinsic, genetic and metabolic factors that may be involved. Therefore, tendinopathies in obese patients can be due to tendon overload because of the excess of weight, but also because of increased production of pro-inflammatory mediators related to fat tissue such as adipokines. This pro-inflammatory state that obese people can suffer is known as adiposopathy, or sick fat syndrome. Weight loss is associated with decrease in adipokines and improvement of musculoskeletal symptoms. ***Conclusion*:** The relation of obesity and tendinopathies is supported by evidences of recent studies, exemplified in this review of literature.

**Correspondence:**
Adham do Amaral e Castro
E-mail: adham.castro@gmail.com

Financial source: none
Conflicts of interest: none

Received for publication: 14/01/2016
Accepted for publication: 17/05/2016

**DESCRITORES** - Tendinopatia. Obesidade. Sobrepeso. Adipócitos.

**RESUMO – *Introdução***: As tendinopatias e as fissuras em tendões respondem por 30% de todas as consultas médicas. A obesidade, que está se tornando um dos problemas de saúde pública mais prevalentes no mundo, pode estar associada com esta condição. ***Objetivo*:** Revisar a literatura acerca da associação entre obesidade e tendinopatias. ***Métodos***: Este é um estudo exploratório e descritivo utilizando artigos em língua inglesa do portal médico Medline, do período de 2006 a 2014. ***Resultados*:** Na patogênese das tendinopatias incluem-se elementos inflamatórios, regenerativos e degenerativos que aparecem de maneira simultânea em todos os estágios da doença. O estresse mecânico sobre os tendões parece ser um dos mais importantes na promoção do processo inflamatório inicial. Todavia não é o único. Existem fatores ambientais, genéticos e metabólicos atuando de maneira ativa. Portanto, as tendinopatias em indivíduos obesos podem se dever à sobrecarga mecânica pelo excesso de peso, mas, também, pelo aumento na produção de mediadores pró-inflamatórios relacionados ao tecido adiposo, como as adipocinas. O estado pró-inflamatório existente no indivíduo obeso é conhecido como adiposopatia ou síndrome da "gordura doente". A perda de peso está associada com decréscimo das adipocinas e diminuição da sintomatologia musculoesquelética. ***Conclusão*:** A associação da obesidade com tendinopatias tem sido fundamentada em estudos recentes como os desta revisão de literatura.

## INTRODUCTION

Tendinopathies and tendon tears are very common in medical practice, accounting for over 30% of all musculoskeletal consultations[6]. In this involvement, the tendinous portion of musculotendinous units loses the normal collagenous architecture that is replaced by an amorphous mucinous material[28]. It is hypothesized that an increase in the amount and duration of mechanical load supported by the tendon unleash programmed cell death or apoptosis[28].

Tendinopathies affect the physical functioning; cause pain and suffering. They may have economic implications for the patients with a negative impact in their quality of life[4,12,30,]. The prompt and accurate diagnosis is important for the correct treatment avoiding chronicity and disability[42]. The most vulnerable tendons are the Achilles, patellar, the rotator cuff and extensor carpis radialis brevis tendons[4].

There are two main types of tendon diseases: the enthesopathy and the tendinopathy. In the first, the inflammatory and mechanical injuries occur at the junction of tendon with the bone; in the second in the tendon midportion. These two types must be differentiated as they may have different etiologies[4].

It has been shown that obesity may be associated with tendinopathies. Besides the well-established health problems related to obesity such as vascular and heart diseases, the musculoskeletal implications of overweight have been more and more studied due to their huge economic burden. Obesity is becoming one of the most prevalent public health issues in the whole world [3,8,14,16,17,19,20,32,39,46,47,50-53]. The 2015 World Health Organization projection showed that 2.3 billion adults are overweight and more than 700 million are obese[52]. This

highlights the importance of studying all interferences of obesity in daily life.

The objective of this study was to do a literature review on the relationship of obesity and tendinopathies, first, focusing on clinical evidences and them focusing on its pathophysiology.

## METHODS

This study is a review of literature using the Pubmed Database. In October, 2015, it was accessed the portal using the following descriptors in English: "obesity"," overweight" and "body mass index" combined with the descriptors in English "tendinopathy", "tendinitis", "rotator cuff", "epicondylitis", "wrist", "patellar", "quadriceps", "Achilles", "plantar fascia" and "tendon".

The target studies of this review were the ones that had as the main objective or one of the main objectives the verification of the relationship between obesity or excess of weight and any kind of tendinopathy. After excluding repeated papers and considering only the clinical studies, the search resulted in 59 papers. But 49 of them were than excluded for the following reasons: studies that verified the relation of any tendinopathy with BMI, but in athletes instead of overweight or obese people; studies that did not made a statistic evaluation of BMI with tendinopathy and showed only frequency; study that evaluated BMI just as a marginal result of a tendinopathy, not being the main or one of the main objectives; studies that evaluated tendon tears instead of tendinopathy. After this process there were 10 articles, which formed the basis of this paper.

The 10 articles were classified according to the following variables: authors, year of publication, study design, number of patients, measure of tendinopathy, results and conclusions.

## RESULTS

Firstly, was sought the name of the first author and year of publication. Then, the study design was analyzed, number of patients, measure of tendinopathy and finally their results and conclusions.

All the 10 articles were published in English and the time of publication varied from 2006 to 2014. The tendinopathies analyzed were: rotator cuff, pattelar, medial and lateral epicondylitis, Achilles, trigger finger, posterior tibial, peroneal tendons, plantar fascia and pes anserinus. The type of studies was case control and cross sectional (some of them involving population).

Figure 1 shows the 10 studies characteristics.

## DISCUSSION

Obesity is a metabolic disease that has grown rapidly globally. The brake to this growth is one of the goals of the World Health Organization (WHO) to be achieved by 2025. WHO estimates in its latest data that more than one third of adults older than 18 years are now overweight, being 38% men and 40% women. In addition, it is estimated that the worldwide prevalence of obesity doubled between 1980 and 2014, being obese in 2014, 11% of men and 15% women, accounting for more than half a billion obese adults worldwide[53].

Tendinopathy is the most prevalent tendon disorder, and various preventive interventions have been investigated. Cook and Purdam (2009)[11] have proposed a model of tendinopathies, involving histological, clinical and imaging information aiming to understand their different presentation. In their model, characterized as a continuum of alterations, the tendinopathy starts as a result of an insult (generally an acute overload or compression) with a non-inflammatory proliferative tissue reaction and minimal collagen damage. In the following stage, the collagen splits up and abnormal tenocytes proliferation with neovascularization occurs. If the patient is treated in these two initial stages, the process may be reversible. In the last stage of this model, there is accentuated disruption of collagen fibers, diffuse cell death with appearance of neo-vessels and nerves in the substance of the tendon[11]. At this level the situation is irreversible. These three phases has been denominated as: 1) reactive tendinopathy; 2) tendon disrepair; and 3) degenerative tendinopathy[26].

Mc Creesh and Lewis (2013)[26] studying the pathogenic process of tendinopathies noted that inflammation and degeneration frequently occurs simultaneously. They defined that inflammatory, regenerative and degenerative processes happen in all stages, from early to late phases. The role of the inflammatory process in this context is not totally clear and may vary depending of which tendon is affected[24].

Mechanical stress upon tendons may be one of the main factors involved in the appearance of tendinopathies[26]. Healthy tendons have elastic properties being able to adapt to the tensions through changes in their mechanical properties and structure[26]. Overload or repetitive use may result in tendon disease and seems to be the dominant factor initiating the inflammatory response. It is believed that some of the damage caused by the overload is mediated through inflammatory process[2,25,33,38].

In addition to loading exposure, a great number of others extrinsic and intrinsic factors may interfere with development of tendinopathy[26]. Anatomical features, posture, occupational and sporting activities are found among the extrinsic factors. Some individuals may have a genetic predisposition[1]. Likewise, metabolic features play a role in tendinopathy, with diabetes mellitus being a well-known risk factor since long time ago[1]. Obesity is not so valued in this context, although this concept is changing as the knowledge in this field is growing.

So, tendinopathies in obese patients may be due not only to joint and tendon overload, but also because of increased production of pro-inflammatory mediators[7,15,17,40].

Adipose tissue is now recognized as a multifunctional organ. It plays an important role as an energy storage organ, but it also releases active pro- inflammatory molecules such as IL-6, TNF-α, and leptin that act on immune cells leading to local and systemic inflammation[27]. The inflammatory mediators elaborated in the fat tissue are generated by local macrophages that are increased in number in obese people; the percentage of these cells in adipose tissue ranges from less than 10% in lean individuals to 40-50% in the obese[30]. The inflammatory cytokines produced by adipocytes acts recruiting more macrophage, therefore perpetuating a vicious cycle of inflammation[27].

Not all obese patients suffer from chronic inflammation; there is a group of patients where the metabolic inflammatory syndrome predominates. Bays (2014)[7] defined adiposopathy, also known as sick fat syndrome, as the result of "a pathologic adipose tissue anatomic/functional disturbances promoted by positive caloric balance in genetically and environmentally susceptible individuals which results in adverse endocrine and immune responses that both directly and indirectly contribute to metabolic disease and increased cardiovascular disease risk"[7]. These people have increased rates of cancer, asthma, atherosclerosis, rheumatoid arthritis, diabetes, osteoporosis, Alzheimer's disease, osteoarthritis and depression[27]. Adiposopathy also leads to pain chronicity because the related non-resolving systemic inflammation that causes a pathophysiologic state that promotes nociception in dysfunctional musculoskeletal tissues and avoids healing and pain resolution[27,40].

Some studies have shown that overweight and obesity are related with disability affecting basic activities of daily living[22,34]; it also increases risk of chronic diseases with their secondary symptoms[23]. All of this impacts people´s quality of life. More recent studies have focused in quality of life changes before and after weight loss[10,21,31,35,36,39,40,43,48,49,53]. Weight loss is capable of improve quality of life, and to reduce risks of obesity associated diseases[43], even if the weight loss is modest[36].

Linkov et al. (2014)[25] demonstrated that the weight loss was associated not only with better quality of life, but also with decrease in adipokines; they also demonstrated a correlation between the level adipokines reduction and improvement of the physical quality of life[25].

Birn et al. (2015)[8] studied the association between musculoskeletal symptoms of obese patients before and after bariatric surgery. They found that patients with musculoskeletal disorders before the

| | Tendinopathy | Study design | Number of patients | Measure of tendinopathy | Results | Conclusions |
|---|---|---|---|---|---|---|
| Titchener et al.[45] (2014) | Rotator cuff | Case-control | 5000 cases and 5000 controls | Recorded diagnosis of rotator cuff disease within THIN database of National Health Service patient data coding system used in United Kingdom | BMI of 25.1–30 (overweight) and RC disease OR: 1.23 (1.10–1.38) BMI of 30.1–40 (obese) and RC disease OR: 1.25 (1.09–1.44) BMI > 40 (morbidly obese) no increased risk When the multivariate analysis was made the association was only observed in the BMI 25.1–30 (overweight) group OR: 1.15 (1.02–1.31) | Significant association observed only for overweight patients |
| Fairley et al.[16] (2014) | Patellar | Cross sectional | 297 | MRI | The prevalence of MRI defined patellar tendinopathy was 28.3%. Current weight (OR per kg = 1.04, 95% CI 1.01-1.06, P = 0.002), BMI (OR per kg/m2 = 1.10, 95% CI 1.04-1.17, P = 0.002), heaviest lifetime weight (OR per kg = 1.03, 95% CI 1.01-1.05, P = 0.007) and weight at age of 18-21 years (OR per kg = 1.03, 95% CI 1.00-1.07, P = 0.05) were all positively associated with the prevalence of patellar tendinopathy. | MRI defined patellar tendinopathy is associated with current and past history of obesity. |
| Titchener et al.[44] (2013) | Lateral epicondylitis | Case-control | 4998 cases and 4998 controls | Data were extracted using Read codes, which is a National Health Service (NHS) patient data coding system used by UK general practices. | BMI > 40 and lateral epicondylitis OR: 1.41 (1.01–1.97). When the multivariate analysis was made the association was no longer observed. | Obesity is not associated with lateral epicondylitis |
| Abate et al.[2] (2012) | Achilles | Case-control | 37 normal weight and 44 overweight | Power Doppler ultrasound. | Sonographic abnormalities were more frequently observed in tendons of overweight participants (21/88 (23.8 %) vs 6/74 (8.1 %), p = 0.007) and, among them, were significantly prevalent in runners (17/50 vs 4/38, p = 0.01). Intratendinous microvessels were found more frequently in tendons of overweight participants (15/88 (17 %) vs 2/74 (2.7 %), p = 0.003), and, in this latter group, were significantly prevalent in runners (12/50 vs 3/38, p = 0.04) | Overweight runners may precociously develop Achilles tendon abnormalities. |
| Rechardt et al.[32] (2010) | Rotator cuff | Cross sectional | 6,237 | Clinically. | In univariable analyses waist circumference was associated with chronic rotator cuff tendinitis in both men and women, while waist-to-hip ratio was associated only in men. Overweight and obese men had a high prevalence of chronic rotator cuff tendinitis; however a statistically significantly increased risk was observed only for increased waist circumference. In women weight-related factors were not statistically significantly associated with chronic rotator cuff tendinitis. However, the odds ratios were above unity for those with increased waist circumference. | There were associations of abdominal obesity with shoulder pain |
| Gaida et al[20] (2010) | Achilles | Cross sectional (population) | 298 | Ultrasound. | Asymptomatic Achilles tendon pathology was more evident in men (13%) than women (5%) (p = 0.007). Men with tendon pathology were older (50.9 ± 10.4, 36.3 ± 11.3, p < 0.001), had greater waist-hip-ratio (WHR) (0.926 ± 0.091, 0.875 ± 0.065, p = 0.039), higher android/ gynoid fat mass ratio (0.616 ± 0.186, 0.519 ± 0.142, p = 0.014) and higher upper-body/lower body fat mass ratio (2.346 ± 0.630, 2.022 ± 0.467, p = 0.013). Men older than 40 years with a waist circumference >83 cm had the greatest prevalence of tendon pathology (33%). Women with tendon pathology were older (47.4 ± 10.0, 36.0 ± 10.3, p = 0.008), had less total fat (17196 ± 3173 g, 21626 ± 7882 g, p = 0.009), trunk fat (7367 ± 1662 g, 10087 ± 4152 g, p = 0.003) and android fat (1117 ± 324 g, 1616 ± 811 g, p = 0.005). They had lower central/peripheral fat mass ratios (0.711 ± 0.321 g, 0.922 ± 0.194 g, p = 0.004) than women with normal tendons. | Men with Achilles tendon pathology had a central fat distribution. Women with tendon pathology had a peripheral fat distribution. An interaction between age and waist circumference was observed among men. |
| De la Parra-Márquez et al.[14] (2008) | Trigger finger | Case control | 250 cases and 250 controls | Cases were patients who were operated on for trigger finger. | Significant statistical relationship was found between trigger finger and obesity (OR 1.49, 95% CI 1.02-2.19). | Trigger finger may be related to body mass index (obesity). |
| Frey et al.[18] (2007) | Achilles, posterior tibial and peroneal tendons and plantar fascia. | Cross sectional | 1141 | Physical examination by an orthopaedic foot and ankle specialist, with the aid of special tests (such as MRI) when indicated. | One hundred and twenty–three (65.4%, standard residual 2.6) of the overweight/obese subjects had a diagnosis of tendinitis compared to 65 (34.6%, standard residual 2.7) normal subjects. Logistic regression comparing BMI and tendinitis demonstrated that it was almost twice as likely (1.923, p<0.0001, 95% CI 1.39 to 2.66) for an individual to have tendinitis of any tendon if overweight or obese. The relationship of BMI and plantar fasciitis demonstrated a 1.4 times increased probability (1.400, p< 0.040 , 95% CI 1.016 to 1.93) of plantar fasciitis being diagnosed in an overweight or obese patient. | Being overweight or obese significantly increased the chances of tendinitis and there was an increased likelihood of plantar fasciitis. |
| Alvarez-Nemegyei et al.[5] (2007) | Pes anserinus | Case control | 22 cases and 38 controls | Clinically. | There was no difference in prevalence of obesity between cases and controls. | No association was found with obesity. |
| Shiri et al.[41] (2006) | Lateral and medial epicondylitis. | Cross sectional (population) | 4783 | Diagnosis based on self-reported symptoms in the interview and clinical signs in the standardized health examination. | Body mass index, waist circumference, and waist:hip ratio were strongly associated with medial epicondylitis but not with lateral epicondylitis. The associations were statistically significant only for women and not for men. In the multivariable analyses after controlling for the effects of other covariates, medial epicondylitis was associated with the waist:hip ratio only in women. | Obesity is associated with medial epicondylitis. |

BMI = body mass index; RC = rotator cuff; OR = odds ratio; MRI = magnetic resonance imaging; CI = confidence interval.

**FIGURE 1** – Distribution of the 10 analyzed articles and their characteristics

surgery experienced a larger improvement of quality of life after weight reduction than participants without symptoms before the procedure[8]. These results indicate an important role of musculoskeletal symptoms in the quality of life of obese people.

Bout-Tabaku et al. (2015)[10] demonstrated that adolescents with severe obesity have musculoskeletal pain that limits their quality of life. They also demonstrated that c-reactive protein (CRP) levels were associated with higher BMI values and with musculoskeletal pain in crude analysis; but after adjustment, no significant association was observed between pain and CRP, suggesting that pain mechanisms of nociceptor activation are complex and related to cytokines, but it is not specific to CRP[10].

## CONCLUSION

Obesity is becoming one of the most important world´s health issues. Adding this to the facts that tendinopathies are one of the most frequent causes of musculoskeletal medical consultations and that obesity may be one of its cause, the scenario of the obese patient in the orthopedic doctors' office tends to become more frequent nowadays. So, it is important to be aware of the reviewed evidences that support the association between these two diseases to better manage these patients.

## REFERENCES


1. Abate M, Schiavone C, Di Carlo L, Salini V. Achilles tendon and plantar fascia in recently diagnosed type II diabetes: role of body mass index. Clin Rheumatol. 2012; 31(7):1109-1113.
2. Abate M, Oliva F, Schiavone C, Salini V. Achilles tendinopathy in amateur runners: role of adiposity (Tendinopathies and obesity). Muscles Ligaments Tendons J. 2012; 2(1):44-48.
3. Abate M. How obesity modifies tendons (implications for athletic activities). Muscles Ligaments Tendons J. 2014; 17; 4(3):298-302.
4. Ackerman PW, Renström P. Tendinopathy in sport. Sports Health. 2012; 4(3): 193-201.
5. Alvarez-Nemegyei J. Risk factors for pes anserinus tendinitis/bursitis syndrome: a case control study. J Clin Rheumatol. 2007;13(2):63-5.
6. Andarawis-Puri N, Flarow EL, Soslowsky LJ. Tendon basic Science: development, repair, regeneration and healing. J Orthop Res. 2015;33(6):780-784.
7. Bays H. Adiposopathy, "sick fat," Ockham's razor, and resolution of the obesity paradox. Curr Atheroscler Rep. 2014 ;16(5):409. doi: 10.1007/s11883-014-0409-1.
8. Birn I, Mechlenburg I, Liljensøe A, Soballe K, Larsen JF. The association between preoperative symptoms of obesity in knee and hip joints and the change in quality of life after laparoscopic Roux-en-y gastric bypass. Obes Surg. 2015 Aug 26. [Epub ahead of print]
9. Bischoff SC 37. Bischoff SC, Damms-Machado A, Betz C, Herpertz S, Legenbauer T, Löw T, et al. Multicenter evaluation of an interdisciplinary 52-week weight loss program for obesity with regard to body weight, comorbidities and quality of life--a prospective study. Int J Obes. 2012; 36(4):614-624.
10. Bout-Tabaku S, Michalsky MP, Jenkins TM, Baughcum A, Zeller MH, Brandt ML, et al. Musculoskeletal pain, self-reported physical function, and quality of life in the teen-longitudinal assessment of bariatric surgery (Teen-LABS) Cohort. JAMA Pediatr. 2015; 169(6):552-559.
11. Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. Br J Sports Med. 2009; 43(6):409-416.
12. Dall TM, Gallo P, Koenig L, Gu Q, Ruiz Jr D. Modeling the indirect economic implications of musculoskeletal disorders and treatment. Cost Eff Resour Alloc. 2013; 11(5). doi: 10.1186/1478-7547-11-5.
13. Danielson P. Reviving the "biochemical" hypothesis for tendinopathy: new findings suggest the involvement of locally produced signal substances. Br J Sports Med. 2009; 43: 265-8.
14. De la Parra-Márquez ML, Tamez-Cavazos R, Zertuche-Cedillo L, Martínez-Pérez JJ, Velasco-Rodríguez V, Cisneros-Pérez V. Risk factors associated with trigger finger. Case-control study. Cir Cir. 2008; 76(4):323-7.
15. Dean BJ, Gettings P, Dakin SG, Carr AJ. Are inflammatory cells increased in painful human tendinopathy? A systematic review. Br J Sports Med. 2015 Aug 5. pii: bjsports-2015-094754. doi: 10.1136/bjsports-2015-094754. [Epub ahead of print].
16. Fairley J, Toppi J, Cicuttini FM, Wluka AE, Giles GG, Cook J, O'Sullivan R, Wang Y. Association between obesity and magnetic resonance imaging defined pattelar tendinopathy in community-based adults: a cross-sectional study. BMC Musculoskelet Disord. 2014; 15:266. doi: 10.1186/1471-2474-15-266.
17. Franceschi F, Papalia R, Paciotti M, Franceschetti E, Di Martino A, Maffulli N, et al. Obesity as a risk factor for tendinopathy: a systematic review. Int J Endocrinol. 2014, Article ID 670262, 10 pages. Available at http://dx.doi.org/10.1155/2014/670262.
18. Frey C, Zamora J. The effects of obesity on orthopaedic foot and ankle pathology. Foot Ankle Int. 2007; 28(9):996-9.
19. Gaida JE, Ashe MC, Bass SL, Cook JL. Is adiposity an under-recognized risk factor for tendinopathy? A systematic review. Arthritis Rheum. 2009; 15; 61(6):8408-9.
20. Gaida JE, Alfredson H, Kiss ZS, Bass SL, Cook JL. Asymptomatic Achilles tendon pathology is associated with a central fat distribution in men and a peripheral fat distribution in women: a cross sectional study of 298 individuals. BMC Musculoskelet Disord. 2010;11:41. doi: 10.1186/1471-2474-11-41.
21. Gloy VL, Briel M, Bhatt DL, Kashyap SR, Schauer PR, Mingrone G, Bucher HC, et al. Bariatric surgery versus non-surgical treatment for obesity: a systematic review and meta-analysis of randomised controlled trials. BMJ. 2013; 347: f5934. Published online 2013 Oct 22. doi: 10.1136/bmj.f5934
22. Han TS, Tijhuis MA, Lean ME, Seidell JC. Quality of life in relation to overweight and body fat distribution. Am J Public Health. 1998; 88(12): 1814–20.
23. Lean MEJ, Han TS, Seidell JC. Impairment of health and quality of life using new US federal guidelines for the identification of obesity. Arch Intern Med. 1999 26 ;159(8):837-43.
24. Legerlotz K, Jones ER, Screen HR, Riley GP. Increased expression of IL-6 family members in tendon pathology. Rheumatology (Oxford). 2012; 51(7):1161-5.
25. Linkov F, Burke LE, Komaroff M, Edwards RP, Lokshin A, Styn MA, et al. An exploratory investigation of links between changes in adipokines and quality of life in individuals undergoing weight loss interventions: possible implications for cancer research. Gynecol Oncol. 2014; 133(1): 67-72.
26. McCreesh K, Lewis J. Continuum model of tendon pathology - where are we now? Int J Exp Pathol. 2013; 94(4):242-247.
27. Moulin CM, Marguti I, Peron JPS, Rizzo LV, Halpem A. Impact of adiposity on immunological parameters. Arq Bras Endocrinol Metab. 2009; 53(2):183-189.
28. Murrel GA. Understanding tendinopathies. Br J Sports Med. 2002;36:392-393.
29. Napoli N, Shah K, Waters DL, Sinacore DR, Qualls C, Villareal DT. Effect of weight loss, exercise, or both on cognition and quality of life in obese older adults." Am J Clin Nutr. 2014; 100(1):189-198.
30. Östör AJK, Richards CA, Prevost AT, Speed CA, Hazleman BL. Diagnosis and relation to general health of shoulder disorders presenting to primary care. Rheumatology. 2005; 44: 800-5.
31. Raoof M, Näslund I, Rask E, Karlsson J, Sundbom M, Edholm D, et al. Health-related quality-of-life (HRqoL) on an average of 12 years after gastric bypass surgery. Obes Surg. 2015;25 (7):1119-1127.
32. Rechardt M, Shiri R, Karppinen J, Jula A, Heliövaara M, Viikari-Juntura E. Lifestyle and metabolic factors in relation to shoulder pain and rotator cuff tendinitis: a population-based study. BMC Musculoskelet Disord. 2010 ;11:165. doi: 10.1186/1471-2474-11-165.
33. Rees JD, Stride M, Scott A. Tendons--time to revisit inflammation. Br J Sports Med. 2014; 48(21):1553-7.
34. Round J, Sampson EL, Jones L. A framework for understanding quality of life in individuals without capacity. Qual Life Res. 2014; 23(2): 477–484.
35. Rubin RR, Wadden TA, Bahnson JL, Blackburn GL, Brancati FL, Bray GA, et al; Look AHEAD Research Group. Impact of intensive lifestyle intervention on depression and health-related quality of life in type 2 diabetes: the Look AHEAD Trial. Diabetes Care. 2014; 37(6):1544-53.
36. Sarwer DB, Moore RH, Diewald LK, Chittams J, Berkowitz RI, Vetter M, Volger S, Wadden TA; POWER-UP Research Group. The impact of a primary care-based weight loss intervention on the quality of life. Int J Obes (Lond). 2013; 37 Suppl 1:S25-30.
37. Sarwer DB, Lavery M, Spitzer JC. A review of the relationships between extreme obesity, quality of life, and sexual function. Obes Surg. 2012; 22(4):668-76.
38. Scott A, Docking S, Vicenzino B, Alfredson H, Murphy RJ, Carr AJ, et al. Sports and exercise-related tendinopathies: a review of selected topical issues by participants of the second International Scientific Tendinopathy Symposium (ISTS) Vancouver 2012. Br J Sports Med. 2013; 47(9):536-44.
39. Scott A, Zwerver J, Grewal N, de Sa A, Alktebi T, Granville DJ, Hart DA. Lipids, adiposity and tendinopathy: is there a mechanistic link? Critical review. Br J Sports Med. 2015; 49(15):984-988
40. Seaman DR. Body mass index and musculoskeletal pain: is there a connection? Chiropr Man Therap. 2013;21(1):15. doi: 10.1186/2045-709X-21-15.
41. Shiri R, Viikari-Juntura E, Varonen H, Heliövaara M. Prevalence and determinants of lateral and medial epicondylitis: a population study. Am J Epidemiol. 2006;164(11):1065-74. .
42. Siena C, Helfenstein Jr M. Diagnostic mistakes involving tendonitis: medical, social, legal, and economic impact. Bras J Rheumatol. 2009; 49(6):712-25.
43. Styn MA, Wang J, Acharya SD, Yang K, Chasens ER, Choo J, et al. Health-related quality of life among participants in the SMART weight loss trial. Appl Nurs Res. 2012; 25(4):276-9.
44. Titchener AG, Fakis A, Tambe AA, Smith C, Hubbard RB, Clark DI. Risk factors in lateral epicondylitis (tennis elbow): a case-control study. J Hand Surg Eur. 2013;38(2):159-64.
45. Titchener AG, White JJ, Hinchliffe SR, Tambe AA, Hubbard RB, Clark DI. Comorbidities in rotator cuff disease: a case-control study. J Shoulder Elbow Surg. 2014; 23(9):1282-8.
46. van der Worp H, van Ark M, Roerink S, Pepping GJ, van den Akker-Scheek I, Zwerver J. Risk factors for patellar tendinopathy: a systematic review of the literature. Br J Sports Med. 2011;45(5):446-52.
47. Viikari-Juntura E, Shiri R, Solovieva S, Karppinnen J, Leino-Arjas P, Varonen H, Kalso E, Ukkola O. Risk factors of atherosclerosis and shoulder pain – Is there an association? A systematic review. Eur J Pain. 2008; 12(4):412-26.
48. Warkentin LM, Majumdar SR, Johnson JA, Agborsangaya CB, Rueda-Clausen CF, Sharma AM, et al. Weight loss required by the severely obese to achieve clinically important differences in health-related quality of life: two-year prospective cohort study. BMC Med. 2014;12:175. doi: 10.1186/s12916-014-0175-5.
49. Warkentin LM, Das D, Majumdar SR, Johnson JA, Padwal RS. The effect of weight loss on health-related quality of life: systematic review and meta-analysis of randomized trials. Obes Rev. 2014 ;15(3):169-182.
50. Wearing SC, Hennig EM, Byrne NM, Steele JR, Hills AP. Musculoskeletal disorders associated with obesity: a biomechanical perspective. Obes Rev. 2006; 7(3):239-50.
51. Wearing SC, Grigg NL, Hooper SL, Smeathers JE. Conditioning of the Achilles tendon via ankle exercise improves correlations between sonographic measures of tendon thickness and body anthropometry. J Appl Physiol (1985). 2011;110(5):1384-9.
52. World Health Organization. Preventing Chronic Diseases: a Vital Investment. Geneva: World Health Organization / Ottawa: Public Health Agency of Canada. 2005.
53. World Health Organization. Global status report on noncommunicable diseases 2014. Global target 7: Halt the rise in diabetes and obesity. ISBN 978 92 4 1564854.http://who.int/iris/bitstream/10665/148114/1/9789241564854_eng.pdf?ua=1
54. Yoon MA, Choi JY, Lim HK, Yoo HJ, Hong SH, Choi JA, Kang HS. High prevalence of abnormal MR findings of the distal semimembranosus tendon: contributing factors based on demographic, radiographic, and MR features. AJR Am J Roentgenol. 2014; 202(5):1087-1093.