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SYSTEMATIC REVIEW

Tensor Fascia Latae Muscle Structure and Activation in Individuals With Lower Limb Musculoskeletal Conditions: A Systematic Review and Meta-Analysis

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Abstract

Background Dysfunction of the tensor fascia latae (TFL) muscle is often clinically implicated in many musculoskeletal disorders.

Objective To systematically review the literature of the TFL muscle to determine whether there are differences in its structure and activation between individuals with and without lower limb musculoskeletal conditions.

Data sources A comprehensive search in MEDLINE, EMBASE, CINHAL, and LILACS was undertaken from year of inception to 9 July 2019.

Eligibility criteria for selecting studies Studies that directly investigated the structure or activity of the TFL muscle between individuals with a lower limb musculoskeletal condition and a pain-free control group.

Results Seventeen studies were included ($n = 556$ participants), eight reporting structure and ten activation of the TFL muscle. Conditions included lateral hip pain, hip joint pathology, ACL injury, iliotibial band syndrome, and patellofemoral joint osteoarthritis. Meta-analysis identified with low confidence (p value = 0.07) a small tendency towards hypertrophy in the affected side of participants with hip joint diseases (SMD 0.37, 95% CI [− 0.02, 0.77]). Moderate effect sizes were found for a higher cross-sectional area of the TFL/sartorius ratio in abductor tendon tear (SMD 0.74; 95% CI [0.05, 1.43, p value = 0.04), and for a smaller body mass normalized TFL volume in patellofemoral joint osteoarthritis (SMD − 0.61; 95% CI [− 1.23, 0.00], p value = 0.05). Normalised electromyography (EMG) amplitude did not differ between groups for any condition, but when EMG was analysed as linear envelopes or synergies, some differences in pattern of TFL activation were observed between individuals with lateral hip pain and controls. Timing of TFL activation did not differ between individuals with knee conditions and controls.

Conclusions and implications Common clinical assumptions of the role of TFL muscle in lower limb musculoskeletal conditions are not well investigated and poorly supported by current research. There are contradictory findings on the muscle size of TFL. Differing methodology in muscle activation studies precludes a clear interpretation for comparison between groups.

PROSPERO registration number CRD42017076160.

1 Introduction

The tensor fascia latae (TFL) muscle has been clinically implicated in the pathomechanics of several lower limb musculoskeletal conditions, such as iliotibial band (ITB) syndrome [1, 2] and patellofemoral pain [3], and the extent to which data support this clinical reasoning has not yet been determined. This muscle has contributed to tasks involving single leg support (e.g., stance phase of gait) [4], but also has actions in the sagittal and transverse planes [5]. Because of its anatomical attachment to the ITB, it has been suggested that augmented activation of the TFL may have effects such as modification of movement patters (e.g., increased hip internal rotation), enhancement of ITB tension, and inhibition [2] leading to subsequent weakening of gluteal muscles [6], thereby contributing to suboptimal patellofemoral joint loading [7] and ITB irritation [8]. This has led to treatments that aim to recruit gluteal muscles while minimising TFL activity [9, 10], and passive therapeutic interventions (e.g.,

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Key points

There are contradictory findings regarding the size of the tensor fascia latae (TFL) muscle between individuals with and without lower limb musculoskeletal conditions

Different methodologies of EMG studies preclude a clear interpretation for comparison between individuals with and without lower limb musculoskeletal conditions

Clinicians have limited consistent evidence on differences in structure and activation that are specific to the TFL on which to base management of lower limb musculoskeletal conditions

massage and stretching of the ITB) [1]. The evidence upon which these programs are based requires consideration. From an aetiological point of view, an increasing strain rate of the ITB (an indirect measure of the viscoelastic tissue tension) has been suggested as a causative factor for development of ITB syndrome in female runners [11]. The potential role of the TFL muscle in this biomechanical model is also relevant for the understanding of such conditions.

Muscle output depends on both morphology/structure of the muscle (which determines the potential to generate force) and activation characteristics. These features provide direct measurements of a specific muscle, and may be altered in musculoskeletal conditions. Muscle structure is typically quantified as size (cross-sectional area or volume), architecture, composition of muscle fibres, and fat area/content [12, 13]. Activation is generally measured using electromyography (EMG) [14], which records the amplitude and timing of muscle activity during tasks or functional movements. Although impairments in structure and activation of the gluteal muscles have been studied in several musculoskeletal conditions [15–17], and measurements of the abductor muscle function (which included contributions from TFL and other muscles, and thus cannot be attributed specifically to TFL) have been studied in clinical populations [7, 8, 15], the role of TFL structure and activation warrants further evaluation.

This study aimed to systematically review the literature on the TFL muscle to determine whether: (1) there are any structural differences (e.g., muscle size, fat area/content), and (2) there are any differences in muscle activation (e.g. amplitude, timing, etc.) between individuals with and without lower limb musculoskeletal conditions.

2 Methods

2.1 Protocol and Registration

This systematic review was performed according to the PRISMA guidelines for reporting systematic reviews [18], and registered at the international prospective register of systematic reviews (PROSPERO) (CRD42017076160).

2.2 Eligibility Criteria

Studies were included if they compared a direct measure of TFL muscle structure (e.g., muscle architecture, size, fibre composition and fat content/area) or activity (e.g., electrical activity, shear wave elastography, magnetic resonance elastography, muscle functional magnetic resonance imaging), between individuals with and without lower limb musculoskeletal conditions. A direct measure was considered to be any method that provides a measuring that is specific to TFL muscle, and thus can be discriminated from other muscles. For this reason, hip strength or ITB tightness were not included in this review as they include contributions from other muscles and tissues. Participants of any age and setting were included. Studies were eligible if they: (1) involved individuals with a lower limb musculoskeletal condition; (2) reported a measure of TFL structure or activity; (3) included a control group; and (4) were reported as a full-text peer-reviewed paper. Musculoskeletal conditions of the lower limb were considered as any condition, injury or disorder affecting the musculoskeletal system (i.e. muscles, tendons, ligaments, bones, cartilage, joints or any connective tissue). Studies were excluded if they: (1) reported non-musculoskeletal diseases; (2) assessed cadavers; (3) employed biomechanical models only.

2.3 Search Strategy

Studies were identified by searching electronic databases (MEDLINE, EMBASE, CINHAL and LILACS) from year of inception to 9 July 2019, regardless of language, and by scanning reference lists of the included articles. The search strategy was divided into two parts: the condition of interest (e.g., physical impairments, injury or disorders of the lower limb) and the muscle of interest (e.g., tensor fascia latae, hip abductor or hip muscle) using a combination of MeSH terms and keywords. The MEDLINE search strategy is presented as a supplementary file (see Online Resource 1).

2.4 Study Selection

The search results from each database were combined and duplicates removed. Titles and abstracts of studies

collected through the search strategy and those from additional sources were screened by one reviewer (MB), then checked by a second reviewer (RM) who reviewed a randomly selected sub-sample (10%) of the excluded studies to confirm selection decisions. Full text review followed a similar process. Clarification on matters of eligibility was discussed with two other investigators (PWH and BV).

2.5 Quality Assessment

Quality of eligible studies was assessed using a modified tool (ROBINS-I) for non-randomised studies, developed by the Cochrane Collaboration group [19] and adapted from a previous systematic review on EMG data [16]. Each item was independently rated by two reviewers (MB, LM) using a scale of 'yes/probably yes' (score = 1) for those adequately meeting the criteria, 'partially/somewhat described' (score = 0.5), and 'no/probably no' (score = 0) for those in which the quality measure was not addressed adequately or not reported clearly (see Online Resource 2). Disagreements over methodological quality were resolved through consensus by a third reviewer. For each study, a quality score—expressed as percentage—was calculated by summing the corresponding item scores and dividing this by the maximum score achievable.

2.6 Data Extraction

A standardised form was used to extract data from the included studies for assessment of study quality and evidence synthesis. Extracted information included: study setting; study design; sample characteristics (age, sex and BMI for both groups, and intensity and duration of symptoms for the condition); diagnostic criteria of the condition (clinical or imaging); sample size; outcome measures (including all quantitative and qualitative muscle structure and activation measures) and protocol (data acquisition, method and instrument employed, task, and signal processing). One reviewer (MB) extracted all data, and any query was discussed and resolved by all investigators. Missing data were requested from study authors. All muscle structure and activity measures that compared participants with lower limb musculoskeletal pain and pain-free individuals were included.

2.7 Statistical Methods

2.7.1 Reliability of Quality Assessment

Data were collected and described as measures of muscle structure (e.g., muscle volume, cross sectional area, fat area/content) or muscle activity (e.g., EMG amplitude, temporal measures). Inter-rater reliability of quality assessment between the two assessors (MB, LM) was reported using Cohen's kappa statistics analysis (STATA version 14.0). Reliability was considered as slight (0.00–0.2), fair (0.21–0.4), moderate (0.41–0.6), substantial (0.61–0.8) or almost perfect (0.81– 1.0) [20].

2.7.2 Meta-analysis of Study Data

Mean and standard deviation were extracted for each outcome measure and the standardized mean differences (SMD) with 95% confidence intervals (CI) were calculated (Cohen's *d*) [21]. When authors reported median and interquartile range, previously described equations were used to estimate the mean (scenario 2) [22], and the sample standard deviation (scenario 3) [23] for the calculation of the SMD. The effect size was considered as small (0.2–0.6), moderate (0.61–1.2), large (1.21–2.0) and very large (2.01–4.0) according to the SMD value [24]. For analysis of heterogeneity, a random effects model was used, as data from different conditions were pooled for meta-analysis, using Review Manager 5.3 software.

3 Results

3.1 Study Selection

The search retrieved a total of 4072 studies of which 114 progressed to full-text assessment. Of these, 17 were eligible with a measure of TFL for analysis (Fig. 1). One of the eligible studies was excluded [25] as it reported on duplicate data [26]. Seven authors were contacted [6, 27–32] to provide additional information. Only one author was not able to provide the requested information [29], precluding the calculation of the SMD for one EMG outcome (i.e. RMS amplitude), but the study was included in the review.

3.2 Study Characteristics

Study and participant characteristics are described in Table 1. All studies had a cross-sectional design, and were published between 2002 and 2019. Five studies (29.4%) investigated lateral hip pain (hip abductor tendon tear [33], gluteal tendinopathy [27], symptomatic hip external snapping [34], greater trochanteric pain syndrome [35], and lateral hip pain [36]). Seven studies (41.2%) included hip joint pathology (hip osteoarthritis (OA) [26, 28, 29], hip labral pathology [32], hip OA or degenerative labral pathology [6], chronic hip joint pain [38], and femoroacetabular impingement (FAI) [30]). Two studies (11.8%) included anterior cruciate ligament (ACL) injury [31, 38]; two (11.8%) ITB syndrome [2, 39]; and one (5.9%) patellofemoral joint OA [40]. The total cohort of participants included 299 with musculoskeletal conditions and 257 pain-free controls. Most studies

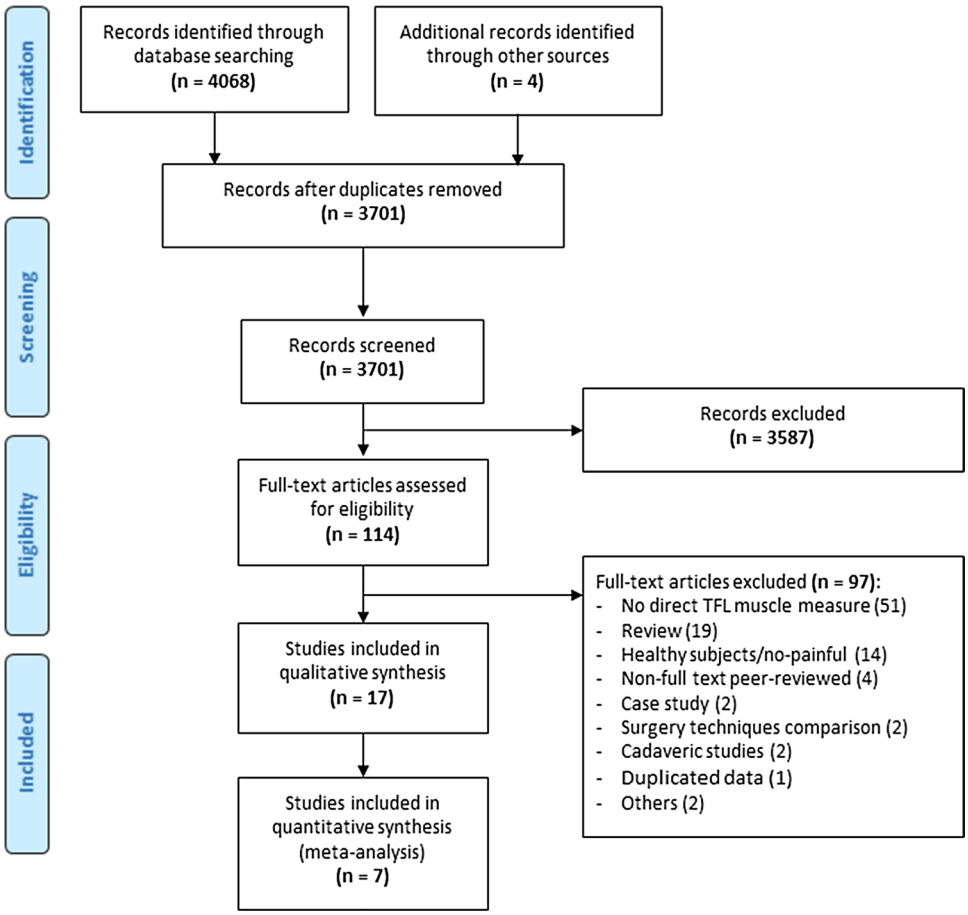

Fig. 1 Preferred reporting items for systematic reviews and meta-analyses flow chart for search and study selection

(82.4%) included matched controls by sex and/or age. The mean age of participants ranged from 24 to 72 years. One study included male participants only, four included females only, and of the remainder ($n = 12$) the proportion of females ranged from 46.7% to 81.3%. Seven studies (41.2%) reported the mean duration of symptoms, which ranged from 1.4 to 85.1 months.

3.3 Quality Assessment

Agreement (89.6%) between the assessors was almost perfect (kappa = 0.847, $p < 0.01$). Table 2 presents the methodological quality assessment scores from the ROBINS-I tool. The mean score was 0.69 (range 0.46–0.91) across all studies; studies of muscle activation rated lower (0.58) than those of muscle structure (0.80). Seven studies did not adequately describe participant characteristics [2, 27, 29, 31, 33, 39, 40], especially physical activity/sport levels and symptom duration. Among muscle activation studies the method used to assess the presence or absence of the musculoskeletal condition differed between the condition and control groups. In contrast, all muscle structure studies used identical clinical examination followed by Magnetic Resonance Imaging (MRI) to confirm diagnosis for both groups. Missing data were not stated in two studies [38, 40], and one study reported a loss of data for TFL muscle greater than 20% [2]. Five studies [29, 32, 36, 38, 39] did not adequately describe statistical analysis.

From studies that measured muscle structure ($n = 8$), three (20%) [26, 36, 40] included muscle volume calculation incorporating multiple slices and accounted for fat content. Two studies [28, 33] did not report reliability of muscle structure measures, and three did not report blinded assessors [6, 26, 30].

Five of nine muscle activation studies clearly described electrode placement [2, 27, 30, 31, 39], and five employed an appropriate amplitude normalisation technique [2, 27, 31, 38, 39] according to the study aim or discussed adequately the limitations of the selected method. Two studies analysed temporal data, providing an adequate description of the method to select EMG onset/offset times [31, 39]. Only one study described the reliability of muscle activation measures [38], and one referenced the reliability results of a previous study [35]. Only one muscle activation study reported blinded assessors [35]. A summary of outcome measures and tasks assessed is presented in Online Resource 3.

Table 1 Characteristics of included articles ($n=17$)

Author (year)	Experimental group characteristics						Control group characteristics			
	Musculoskeletal condition (n)	Diagnostic criteria	DOS (months)	Gender (F:M)	Age (years)	BMI (kg/m^2)	Controls (n)	Gender (F:M)	Age (years)	BMI (kg/m^2)
Zacharias et al. (2016) [26]	Unilateral hip OA ($n=20$); grade 2 ($n=7$), grade 3 ($n=13$)	Imaging	NR	11:9	63.4 ± 5.4	30.0 ± 5.2	Control group matched by age and gender ($n=20$)	11:9	62.1 ± 5.6	24.8 ± 2.8
Arokoski et al. (2002) [28]	Hip OA ($n=27$; unilateral = 15, bilateral $n=12$)	Clinical	Mean 76.8 ± 62.4	0:27	56.2 ± 4.9	Height: 176.7 ± 4.8 cm Weight: 83.9 ± 11.3 kg	Healthy controls matched by age and randomly selected ($n=30$)	0:30	56.3 ± 4.5	Height: 173.8 ± 4.8 cm Weight: 81.4 ± 9.6 kg
Sims et al. (2002) [29]	Unilateral hip OA ($n=19$)	Clinical	Mean 85.2 ± 54	12:7	70.2 (range 52–79)	Height: 165.6 cm (151–177) Weight: 72.9 kg (43–92.5)	Healthy older unmatched adults ($n=19$)	15:4	71.7 (range 60–88)	Height: 162.4 cm (151–185) Weight: 64.8 kg (54–84.5)
Grimaldi et al. (2009) [6]	Unilateral hip OA or degenerative labral pathology ($n=12$; mild $n=6$, advanced $n=6$)	Medical and imaging (X-Ray or MRI)	NR	6:6	*Mild* 46.5 ± 9.5 *Advanced* 57.7 ± 6.7	*Mild* 27.3 ± 3.5 *Advanced* 26.6 ± 4.4	Control group matched by age and sex ($n=12$)	6:6	51.8 ± 9.7	25.9 ± 3.5
Mendis et al. (2014) [32]	Unilateral hip labral pathology ($n=12$)	Clinical and imaging (MRI)	Mean 21 ± 26	8:4	35.0 ± 13.0	22.5 ± 2.1	Healthy age and sex matched control group ($n=12$)	8:4	35.0 ± 2.0	22.9 ± 2.5
Mastenbrook et al. (2017) [37]	Unilateral or the most symptomatic chronic hip joint pain ($n=15$)	Clinical	Mean 42 (range 4.8–156)	15:0	28.3 ± 4.1	24.0 ± 3.3	Healthy asymptomatic matched controls ($n=15$)	15:0	28.3 ± 4.4	24.5 ± 3.2
Casartelli et al. (2011) [30]	Unilateral or the most symptomatic FAI ($n=22$); cam ($n=6$), pincer ($n=4$), combined ($n=12$)	Clinical and imaging (X-ray and MRI)	NR	14:8	32.0 ± 9.0	23.5 ± 3.9	Controls matched for gender, age, and body mass ($n=22$)	14:8	32.0 ± 9.0	23.5 ± 3.2
Sutter et al. (2013) [33]	Unilateral abductor tendon tear ($n=16$)	Imaging (MRI)	NR	13:3	71.8 ± 10.7	23.9 ± 2.1	Patients without abductor tear (unmatched) ($n=19$); contralateral side ($n=18$)	11:8	58.5 ± 18.2	27.5 ± 4.9
Flack et al. (2012) [36]	Unilateral lateral hip pain ($n=10$)	Clinical	NR	10:0	55 (range 43–68)	24.8 ± 3.6	Controls matched by age ($n=10$)	10:0	55 (range 43–68)	23.9 ± 4.1

Table 1 (continued)

Author (year)	Experimental group characteristics						Control group characteristics			
	Musculoskeletal condition (*n*)	Diagnostic criteria	DOS (months)	Gender (F:M)	Age (years)	BMI (kg/m^2)	Controls (*n*)	Gender (F:M)	Age (years)	BMI (kg/m^2)
Jacobsen et al. (2013) [34]	Symptomatic external snapping hip (*n* = 13; 7 bilateral, 6 unilateral)	Clinical	Median 30 (18–60)	7:6	25.5 ± 3.4	23.1 ± 4.2	Control group matched by age and sex (*n* = 13)	7:6	25.6 ± 2.6	23.0 ± 3.0
Allison et al. (2013) [27]	Unilateral GT (*n* = 8); 'affected' hip.	Clinical and imaging (MRI)	NR	5:3	54.0 ± 10.0	24.0 ± 4.5	Healthy controls matched by age (*n* = 8); 'test' hip selected by coin-toss	5:3	51.0 ± 10	25.0 ± 2.4
Ganderton et al. (2017) [35]	Post-menopausal women with unilateral GTPS (*n* = 8)	Clinical	NR	8:0	58.9 ± 3.3	31.4 ± 9.5	Healthy controls post-menopausal women (*n* = 10)	10:0	60.2 ± 2.6	25.3 ± 3.5
Ackland et al. (2018) [40]	Unilateral or the most symptomatic patellofemoral joint OA (*n* = 51)	Clinical and imaging (MRI)	NR	32:19	55 ± 10	27 ± 4	Age-matched healthy controls (*n* = 13)	8:5	52.0 ± 4.0	25.0 ± 3.0
Dingenen et al. (2015) [31]	Unilateral ACL injury (*n* = 15)	Imaging (MRI)	Mean 1.4 ± 0.7	8:7	24.7 ± 5.7	23.1 ± 1.7	Control group (*n* = 15), matched by dominant/ non-dominant leg	10:5	24.2 ± 2.1	22.5 ± 2.0
Flaxman et al. (2018) [38]	Unilateral ACL injury (*n* = 24)	Imaging (MRI) and clinical	14.6 ± 12.5	11:13	F 25.3 ± 8.2 M 31.7 ± 9.0	F/M height: 170.1 ± 5.0 181.9 ± 4.0 F/M weight: 66.7 ± 9.0 84.5 ± 8.3	Control group (*n* = 24) matched by sex, age, BMI and leg dominance	11:13	F 26.2 ± 7.2 M 29.1 ± 6.7	F/M height: 170.2 ± 4.8 182.9 ± 4.9 F/M weight: 64.1 ± 8.7 82.3 ± 10.1
Baker et al. (2018) [2]	Iliotibial band syndrome (*n* = 15)	Clinical	NR	7:8	F 33.4 ± 5.9 M 32.8 ± 6.1	F 22.6 ± 2.3 M 24.0 ± 2.6	Controls matched for gender, age, and body mass (*n* = 15)	7:8	F 31.4 ± 7.5 M 31.1 ± 6.0	F 23.2 ± 2.3 M 23.0 ± 2.0
Brown et al. (2019) [39]	Iliotibial band syndrome (*n* = 12)	Clinical	NR	12:0	32.4 ± 7.9	Height: 1.7 ± 0.1 Weight: 60.6 ± 5.0	Healthy control group (*n* = 20)	20:0	28.9 ± 6.1	Height: 1.6 ± 0.1 Weight: 56.8 ± 5.2

DOS duration of symptoms, *NR* not reported, *F* females, M males, *BMI* body mass index, *CSA* cross-sectional area, *VAS* visual analogue pain scale, *OA* osteoarthritis, *AMI* activity metabolic index, *MRI* magnetic resonance imaging, *ROI* region of interest, *LTPA* leisure time physical activity, *MVIC* maximal voluntary isometric contraction, GT gluteal tendinopathy, *CV* coefficient of variability, *NRS* numerical rating scale, *VISA* Victorian Institute of Sport Assessment, *ML* mediolateral, *AP* anteroposterior, *GTPS* greater trochanteric pain syndrome, *FAI* femoroacetabular impingement

3.4 Muscle Structure

Eight studies compared structural measures of the TFL in lateral hip pain [33, 36], hip joint pathology [6, 26, 28, 32, 37], and patellofemoral joint OA [40], and all used MRI for assessment. Outcome measures included muscle cross-sectional area (CSA, cm^2), muscle volume (cm^3), and fat area/content. From those reporting muscle volume [6, 26, 36, 40], three studies excluded fat area from measurements [26, 36, 40] and one indirectly estimated it using Cavalieri's method [36]. All measurements were made in a supine position. One study included comparison of images made before and after a submaximal hip flexion task [32]. Five studies compared affected with unaffected sides in symptomatic participants and with matched sides in healthy controls [6, 26, 32, 33, 36], and one study compared muscle size between mild and severe OA [28]. A summary of the task, protocol, outcome measures, and effect estimates (SMDs and CIs) of each muscle structure study is presented in Table 3. Muscle structure outcomes are elaborated in the following subsections for each musculoskeletal condition.

3.4.1 Hip Joint Pathology

There was no significant difference in TFL muscle CSA between groups (Fig. 2) [28, 32] or between sides in those with a unilateral condition [32]. Although authors reported a significantly smaller CSA of the TFL in the more severely affected hip within the hip OA group [28], our analysis found this not to be statistically significant (SMD 0.53, 95% CI [− 0.01, 1.07], p value = 0.33). From the three studies [6, 26, 37] pooled for muscle volume measurements (Figs. 3 and 4), a tendency towards hypertrophy was found in participants with hip joint diseases, with small effect sizes and low confidence (p value = 0.07) of the affected side (SMD 0.37, 95% CI [− 0.02, 0.77]), and non-significant of the unaffected side (SMD 0.37, 95% CI [− 0.10, 0.84], p value = 0.12). Only one of those studies excluded fat area when measuring muscle size [26]. When condition severity was considered [6, 26], no differences were found between the affected sides and the control group, even when normalised to BMI [26].

3.4.2 Lateral Hip Pain

There were no statistically significant differences in diameter [33], CSA [34] or volume [36] when participants with lateral hip pain were compared to controls. Analysis of the ratio between the CSA of the TFL and sartorius muscles (as a size reference) revealed a moderate effect size (SMD 0.74, 95% CI [0.05, 1.43], p value = 0.04) for a higher ratio in participants with an abductor tendon tear than controls [33]. When affected side was compared to unaffected side, there were no significant differences for TFL CSA [33], TFL/Sartorius ratio [33] or TFL volume [36] between sides for either group. There were no significant differences for fat content between groups [33].

3.4.3 Patellofemoral Joint OA

Although authors reported a statistically lower TFL muscle volume in participants with the condition compared to controls [40], our analysis found no effect (SMD -0.07, 95% CI [− 0.68, 0.54], p value = 0.81). There was a tendency for atrophy, with a moderate effect size (SMD − 0.61, CI 95% [− 1.23, 0.00], p value = 0.05), when normalised muscle volume (cm^3/kg) was analysed [40].

3.5 Muscle Activation

Nine studies compared amplitude and temporal variables of TFL EMG; three in lateral hip pain [27, 35], two in hip joint pathology [29, 30], two in ACL injury [31, 38], and two in ITB syndrome [2, 39]. Only one study used muscle functional magnetic resonance imaging (mfMRI) to estimate the pattern of muscle recruitment through the calculation of signal intensity changes on the mfMRI in individuals with acetabular labral tear [32]. Different EMG methods were used across studies. The EMG amplitudes were normalised to peak muscle activity during gait [34, 35], to the average of peak amplitudes during gait [27], to the middle five seconds of EMG during a submaximal contraction [29], and to the maximal voluntary isometric contraction (MVIC) [2, 38]. One study reported non-normalised root mean square amplitude during a maximal contraction [30], and two measured non-normalised EMG onset timing during a postural transition task [31] and during an overground running trial. Outcome measures included peak amplitude, mean amplitude, root mean square amplitude, linear envelopes, synergies, muscle activation variability, time to peak, EMG onset time (seconds) and EMG activation timing (% of the gait cycle). Table 4 summarises EMG and mfMRI protocols, assessed tasks, outcome measures and effect estimates (SMDs and CIs) for each muscle activation study. A walking task was performed in three studies [27, 34, 35]; one study used the transition from double stance to single stance (eyes open and closed) [31]; one active hip flexion [30]; one a stepping task [29]; one a standing force matching task to elicit combinations of sagittal, frontal and transverse plane moments [38]; and two a running trial [2, 39]. All studies used surface EMG electrodes for TFL with different electrode placements. Due to differences in outcome measures, methods and tasks assessed, meta-analysis was not possible. Muscle activation outcomes are elaborated in the following subsections for each musculoskeletal condition.

Table 2 Methodological quality assessment using the ROBINS-I tool [16] for all studies

Study	External validity				Internal validity									Total score
	1	2	3	4	5	6	7	8	9	10	11	12	13	(_/1)
Muscle structure studies														
Mastenbrook et al. (2017) [37]	1	1	1	0.5	1	N/A	N/A	0.5	1	1	1	1	1	0.91
Mendis et al. (2014) [32]	1	1	1	0	1	N/A	N/A	0.5	1	1	1	1	0.5	0.86
Zacharias et al. (2016) [26]	1	1	1	0.5	1	N/A	N/A	1	1	0	0.5	1	1	0.82
Grimaldi et al. (2009) [6]	1	1	1	0.5	1	N/A	N/A	0.5	1	0	1	1	1	0.82
Flack et al. (2012) [36]	0.5	1	1	0	1	N/A	N/A	1	1	1	1	1	0.5	0.82
Arokoski et al. (2002) [28]	1	1	1	0.5	1	N/A	N/A	0.5	0	1	1	1	1	0.82
Sutter et al. (2013) [33]	1	1	0.5	0.5	1	N/A	N/A	0.5	0	1	0.5	1	1	0.73
Ackland et al. (2018) [40]	0.5	1	0.5	0	1	NA	NA	1	1	0	1	0	1	0.64
Muscle activation studies														
Casartelli et al. (2011) [30]	1	1	1	0.5	0.5	1	0.5	N/A	0	0	0.5	1	1	0.67
Ganderton et al. (2017) [35]	0.5	1	1	0	0.5	0.5	0	N/A	0.5	1	0.5	1	1	0.63
Dingenen et al. (2015) [31]	0.5	1	0.5	0.5	0.5	1	1	N/A	0	0	0.5	1	1	0.63
Flaxman et al. (2018) [38]	1	0.5	1	0	0.5	0.5	1	NA	1	0	0.5	0	0.5	0.63
Baker et al. (2018) [2]	0.5	1	0.5	0.5	0.5	1	1	NA	0	0	0.5	0.5	1	0.58
Jacobsen et al. (2013) [34]	1	1	1	0.5	0.5	0.5	0	N/A	0	0	0.5	1	1	0.58
Allison et al. (2017) [27]	0	1	0.5	0	0.5	1	1	N/A	0	0	0.5	1	1	0.54
Sims et al. (2002) [29]	1	1	0.5	0.5	0.5	0.5	0.5	N/A	0	0	0	1	0.5	0.50
Brown et al (2019) [39]	0	0.5	0.5	0	0.5	1	1	NA	0	0	0.5	1	0.5	0.46

Black shading—'yes' (score = 1), grey shading—'partial' (score = 0.5), white shading—'no' (score = 0), N/A—not applicable (item removed from scoring)

External validity: (1) source of population; (2) eligibility criteria; (3) participant characteristics; (4) sampling. Internal validity: (5) identifying condition/controls; (6) electrodes placement; (7) EMG normalisation; (8) muscle size calculation; (9) reliability of outcomes measures; (10) blinded assessor; (11): confounders; (12) missing data; (13) statistical tests

Adapted from Semciw et al. 2016 [16]

3.5.1 Lateral Hip Pain

Two studies included comparison of EMG amplitude measures between groups [34, 35]. One study [34] found that the TFL EMG during specific time points of the gait cycle relative to the peak activity of that muscle across the gait cycle, was similar between individuals with and without external snapping. The other study [35] presented peak and average amplitudes, normalised to the respective peak muscle activation during the gait cycle and time normalised to 100-points (% of gait cycle). No significant differences were found for average amplitude between groups, but a greater proportion of peak muscle activity during swing phase was found in the condition group with a small effect size (SMD 2.23, 95% CI [0.95, 3.51], p value < 0.01) [35]. However, the normalisation method employed in these two studies precludes direct comparison of amplitude between groups. There were no significant differences between symptomatic and control groups on other comparable variables, such as, time to peak and muscle activation variability [36]. Although the ensemble curves of the TFL EMG amplitude were not discussed by Ganderton *et al* [35], the shape of the ensemble average illustrated a different pattern of TFL activation across the stance phase during gait (larger and longer bursts of activation) in the gluteal tendinopathy group compared to controls. This observation is similar to the findings reported using synergy analysis by Allison et al. [27]. Authors found that TFL contributes more to the synergy that is activated across stance phase than the synergy that was primarily active in early stance in the GT group. This differs from controls where TFL was similarly represented in both synergies. In addition, both synergies showed a longer burst of muscle activity after heel strike for the gluteal tendinopathy group than healthy controls [27].

3.5.2 Hip Joint Pathology

Calculation of SMDs was not possible as the value of the outcome measure (i.e., RMS of the EMG signal) was not reported nor provided by authors when requested [29]. The authors reported no significant differences for the magnitude of TFL EMG normalised to a submaximal task (hip abduction in side-lying) during a weight shift phase between groups or sides [29]. Results suggest no variations of TFL pattern through the stepping task. In another study [30], authors reported lower non-normalised RMS amplitude (μV) of TFL EMG activity in individuals with FAI compared to controls ($p = 0.048$) during a maximal voluntary contraction of hip flexion, but our analysis found a non-significant effect (SMD − 0.61, 95% CI [− 1.32, 0.10], p value = 0.09). Another study [32] conducted a signal intensity analysis after a hip flexion exercise, and found a similar pattern of increased signal intensity (interpreted to infer similar levels of activation during the exercise) between groups and between sides.

Table 3 Tensor fascia latae muscle structure results ($n = 8$)

Author/condition	Imaging protocol		Outcome	Musculoskeletal condition			Control group			SMD	95% CI
	Task/position	Image analysis		Mean/AF	SD/%	*N*	Mean/AF	SD/%	*N*		
Grimaldi et al. [6] Hip OA or degenerative labral pathology	Resting Supine with legs extended to a neutral position	A T2 True Fast Imaging with Steady State Progression (FISP) sequence using 2 series of 28 × 6 mm contiguous slices from the iliac crest to the most distal extent of the GM muscle was employed. Osiris software package was used to measure ACSA (cm^2) on each image in which the muscle appeared. Muscle volume (cm^3) was calculated by multiplying CSA by slice width and then adding the volumes from each slice	Muscle volume (cm^3)								
			Affected side	Mild 82.5	0	6	74.9	24	12	0.32	[− 0.67, 1.30]
				Adv 86.2	38	6	74.9	24	12	0.37	[− 0.62, 1.36]
			Unaffected side	Mild 73.8	19	6	75.4	26	12	− 0.06	[− 1.04, 0.92]
				Adv 89.5	27	6	75.4	26	12	0.51	[− 0.49, 1.51]
Arokoski et al. [28] Hip OA	Resting Supine	The length of the femur was divided into 4 sections (T1-weighted axial scans of the pelvis and thigh and the ACSA (cm^2) of muscles was determined from the following regions: (1) upper and (2) lower border of acetabulum (TFL measure), (3) upper and (4) middle thigh	Muscle CSA (cm^2)	8.9	1.8	27	9.4	2.0	30	− 0.26	[− 0.78, 0.26]
			Better hip	10.2	2.7	27	–	–		0.53	[− 0.01, 1.07]
			Worse hip	8.9	2.1	27	–	–			
Zacharias et al. [26] Hip OA	Resting Supine with both feet secured to avoid any hip rotation	A multi-planar localiser scan was performed from above the iliac crest to mid femur to identify the muscles of interest. A coronal T1 fast spin echo was then performed to include the region of interest. This was followed by an axial T1 fast spin echo that was acquired as a single stack. Two NSA (number of sample averages) were used for both sequences. The area of TFL was traced using Sante DICOM editor software, excluding any fatty infiltration. Final volume was calculated by the summation of ACSA and multiplying it by slice thickness	Muscle volume (cm^3)								
			Affected limb	65.2	27.1	20	52.4	20.0	20	0.53	[− 0.10, 1.16]
			Unaffected limb	66.0	34.0	20	52.0	19.1	20	0.50	[− 0.13, 1.13]
			Normalised muscle volume (cm^3/kg)								
			Affected limb	0.7	0.2	20	0.7	0.2	20	0.00	[− 0.62, 0.62]
			Unaffected limb	0.7	0.3	20	0.7	0.2	20	0.00	[− 0.62, 0.62]
			Fat area/content								
			1 or less (score of 0 or 1)	19	95%	20	20	100%	20	− 0.05	[− 0.18, 0.08]
			2 or more (score of 2–4)	1	5%		0	0%	20	0.05	[− 0.08, 0.18]

Table 3 (continued)

Author/condition	Imaging protocol		Outcome	Musculoskeletal condition			Control group			SMD	95% CI
	Task/position	Image analysis		Mean/AF	SD/%	*N*	Mean/AF	SD/%	*N*		
Mastenbrook et al. [37] CHJP	Resting Supine with their lower extremities in neutral	TFL ROI was measured in the transverse plane from the slice showing the first appearance of the ischial tuberosity distally to the slice showing the centre of the femoral head. Muscle boundaries were semi-automatically outlined using the auto trace feature within the ROI for consecutive slices	Muscle volume (cm^3)	33	11.0	15	31	9.0	14	0.19	[− 0.54, 0.92]
Mendis et al. [32] Acetabular labral tear	Resting; supine with a pillow under their knees	Transverse scans were performed through the pelvis from the iliac crest to just below the lesser trochanter of the femur using a T2 weighted sequence. Approximately 26 slices were scanned with a slice thickness of 8 mm and an inter-slice gap of 8.8 mm in 5 min. TFL muscle was measured from consecutive slices spanning the femoral head of the hip joint, calculating its average ACSA	Muscle CSA (cm^2)								
			Injured side/dominant	6.2	2.5	12	5.8	2.2	12	0.16	[− 0.64, 0.97]
			Non-injured/non-dominant	5.9	2.2	12	5.7	1.9	12	0.09	[− 0.71, 0.89]
Flack et al. [36] Lateral hip pain	Resting Supine	The circumference was traced using Osirix. T1-weighted scans were used for assessing hip abductor muscle volume, and T2-weighted scans for diagnoses of pathology. The anatomical area covered for each scan was the highest point of the iliac crest superiorly, to the base of the lesser trochanter inferiorly and from the anterior to posterior skin surfaces of the participant. Volume was calculated according Cavalieri's method	Muscle volume (cm^3)								
			Symptomatic hip	51.3	13.8	10	50.5	18.8	10	0.05	[− 0.83, 0.92]
			Asymptomatic hip	51.1	9.1	10	49.0	20.0	10	0.13	[− 0.75, 1.01]
Sutter et al. [33] Lateral hip pain (abductor tendon tear)	Resting Supine	The image was selected at the level of the centre of the femoral head for quantitative measurements of ACSA on a transverse T1-weighted sequence	Muscle CSA (cm^2)	5.21	1.41	16	5.67	2.15	19	− 0.24	[− 0.91, 0.42]
			Ratio TFL/sartorius (cm^2)	2.5	1.01	16	1.89	0.59	19	0.74	[0.05, 1.43]
			Fat area/content								
			1 or less (score of 0 or 1)	9	56.3%		6	31.6%		0.25	[− 0.07, 0.57]
			2 or more (score of 2–4)	7	43.8%	16	13	68.4%	19	− 0.25	[− 0.57, 0.07]

Table 3 (continued)

Author/condition	Imaging protocol		Outcome	Musculoskeletal condition			Control group			SMD	95% CI
	Task/position	Image analysis		Mean/AF	SD/%	*N*	Mean/AF	SD/%	*N*		
Ackland et al. [40] PFJ OA	Resting Supine with both knees fully extended and legs strapped together	MR images were obtained from a Siemens (Erlangen Germany) 3T Trio MR scanner using a T2-weighted fat-suppressed (water excitation). Slice thickness was 1 mm, with no gaps between slices. The entire pelvic region was imaged, from the sacral promontory to the inferior aspect of the pubic arch. The volume of the TFL was measured from axial MR images and the muscle was segmented by semi-automatically digitising muscle CSA on each axial MR image, from its proximal origin to distal insertion, and excluded fat. To estimate total muscle volume, CSAs of each axial slice were summed and multiplied by slice thickness	Muscle volume (cm^3)	62.9	22.9	51	64.6	22.2	13	− 0.07	[− 0.68, 0.54]
			Normalised muscle volume (cm^3/kg)	0.8	0.3	51	1.0	0.4	13	− 0.61	[− 1.23, 0.00]

CSA cross sectional area, *TFL* tensor fascia latae, *SD* standard deviation, *AF* absolute frequency, *SMD* standardised mean differences, *CI* confidence interval, *ACSA* anatomical cross-sectional area, *GM* gluteus maximus, *CHJP* chronic hip joint pain, *MVC* maximal voluntary contraction, *AP* anteroposterior, *PFJ OA* patellofemoral joint osteoarthritis

3.5.3 ACL Injury

One study investigated the TFL EMG onset time during transition from double to single leg stance [31]. With eyes closed, a delayed EMG onset was found for individuals with ACL injury compared to controls, with a significant moderate effect size (SMD 0.83, 95% CI [0.08, 1.58], p value = 0.03). The eyes open condition was not different between groups. No significant differences were found between dominant and non-dominant sides. Another study [38] measured normalised peak EMG during standing on a force platform while participants isometrically increased ground reaction forces towards different directions. No effect was found for the peak of TFL EMG between groups (SMD = 0.00, 95% CI [− 0.57, 0.57]).

3.5.4 ITB Syndrome

One study measured TFL EMG amplitude in runners with and without ITB syndrome during a running trial at three and thirty minutes of a run [2]. Although authors reported a greater EMG amplitude ($p = 0.02$) at three minutes in the ITB syndrome group (11%) than controls (7%), our analysis showed a non-significant small effect (SMD 0.59, 95% CI [− 0.19, 1.37], p value = 0.14). No significant differences were found between groups at thirty minutes of the run test. The other study [39] investigated the onset activation timing of TFL (expressed as the % of the gait cycle) during the terminal swing of an overground running, pre- and post- a treadmill run to fatigue. No effects were found for the TFL terminal swing activation timing (either pre- or post-fatigue) between groups (SMD_{pre} 0.28, 95% CI [− 0.47, 1.04], p value = 0.46; SMD_{post} 0.06, 95% CI [− 0.69, 0.80], p value = 0.88).

4 Discussion

The primary aim of this study was to systematically review the evidence for differences in structure or activation of the TFL muscle between individuals with lower limb musculoskeletal conditions and condition-free controls. We identified 17 eligible studies, encompassing numerous lower limb conditions, with varied outcome measures, and different methodological and assessment techniques. Inconsistent findings were found regarding relative muscle size differences for specific conditions, such as, lateral hip pain and patellofemoral joint OA, and no significant differences were found in individuals with hip joint pathology. Based on the reported EMG data, it is not possible to make robust conclusions about TFL muscle activation (for either the level of activation or detection of temporal events), but some differences in patterns of muscle activation were observed in individuals with lateral hip pain.

4.1 Structure of the TFL Muscle in Musculoskeletal Conditions

The meta-analysis does not provide convincing evidence for or against a contribution of TFL dysfunction in hip joint diseases. The non-significant and imprecise small effect size needs to be considered in light of the low number of studies and participant numbers, particularly the study by Grimaldi et al. [6] in which the sample was separated into two groups (mild and advanced), decreasing the statistical power due to small sample sizes ($n = 6$ for each group). When the severity of a condition is considered, the TFL muscle size does not seem to be different from controls [6]. Muscle volumes of gluteus maximus, minimus and medius muscles [25, 26] have been associated with the severity of the condition, suggesting greater atrophy, especially for deeper muscles, in more significant diseases. Grimaldi et al. [6] also measured upper and lower portions of the gluteus maximus and found no differences in muscle volume for the upper portion, as was reported for the TFL muscle. Grimaldi et al. [6] suggest that these two muscles that function as superficial hip abductors and are synergists in that action, tend to maintain their size around the affected hip. Thus, they are affected in a similar manner by hip joint pathology.

A moderate effect size was found for a higher CSA ratio of the TFL to sartorius muscle in participants with an abductor tendon tear than controls [33]. This could imply either hypertrophy of the TFL or atrophy of the sartorius muscle, but care should be taken with the interpretation of this effect estimate as the confidence intervals were wide making the estimate imprecise. Sutter et al's [33] retrospective analysis should be interpreted with care, as they recruited controls that differed in age and BMI; both factors known to affect muscle size [41]. Conversely, participants with patellofemoral joint OA showed a tendency towards atrophy of the TFL muscle when a normalised muscle volume was calculated [40], but again wide ranges of confidence intervals do not allow a robust interpretation of the estimated effect. This implies that regardless of sample size there is likely no difference between groups. The capacity of the TFL muscle to generate force might be lower in this population [41]. Whether this lower capacity is isolated to TFL or part of generalised lower extremity force reduction is unknown. Additional studies of the TFL muscle structure or activation in people with patellofemoral joint OA are required to generate reobust conclusions for this condition.

Of interest, the mean reported muscle volumes varied from 33 cm^3 to 86.2 cm^3 within individuals with musculoskeletal conditions. Although this wide range might be partially explained by differences in participant sex, age and

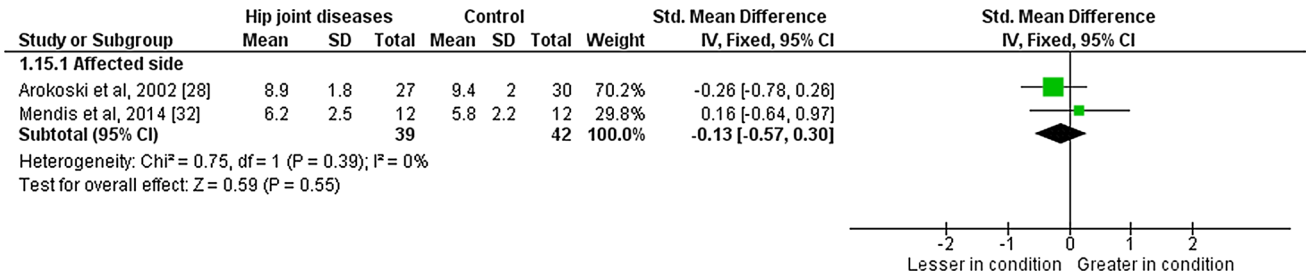

Fig. 2 Muscle cross-sectional (CSA) area (affected side). Forrest plot detailing standardised mean differences for TFL CSA of the affected side between individuals with hip joint pathology and controls. *TFL*, tensor fascia latae

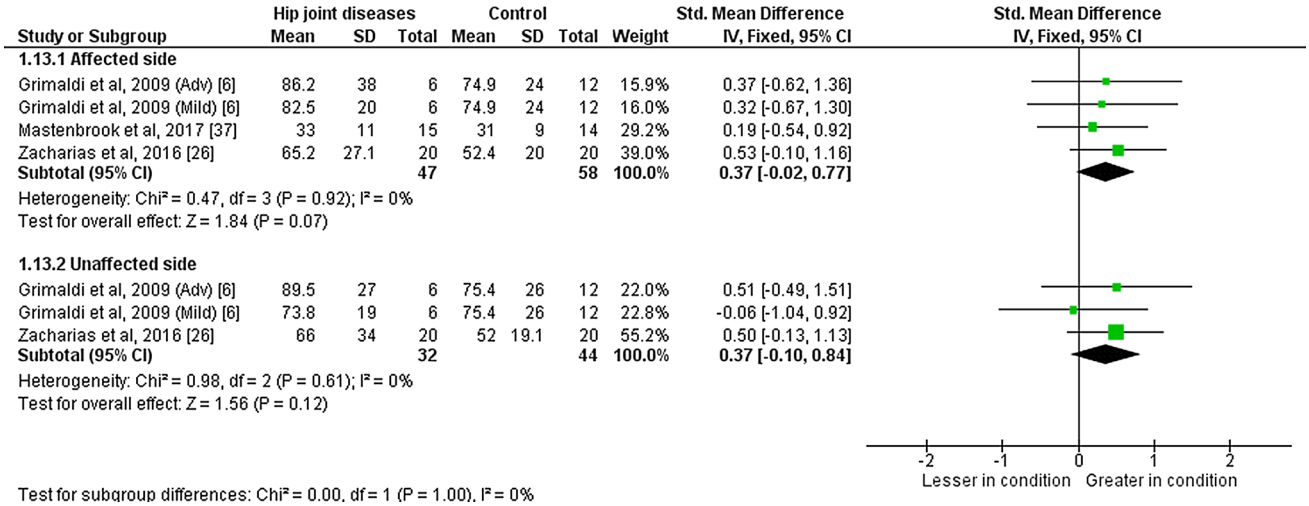

Fig. 3 Muscle volume (affected side). Forrest plot detailing standardised mean differences for TFL volume of the affected side between individuals with hip joint pathology, lateral hip pain and patellofemoral joint OA, and controls. *OA*, osteoarthritis; *TFL*, tensor fascia latae

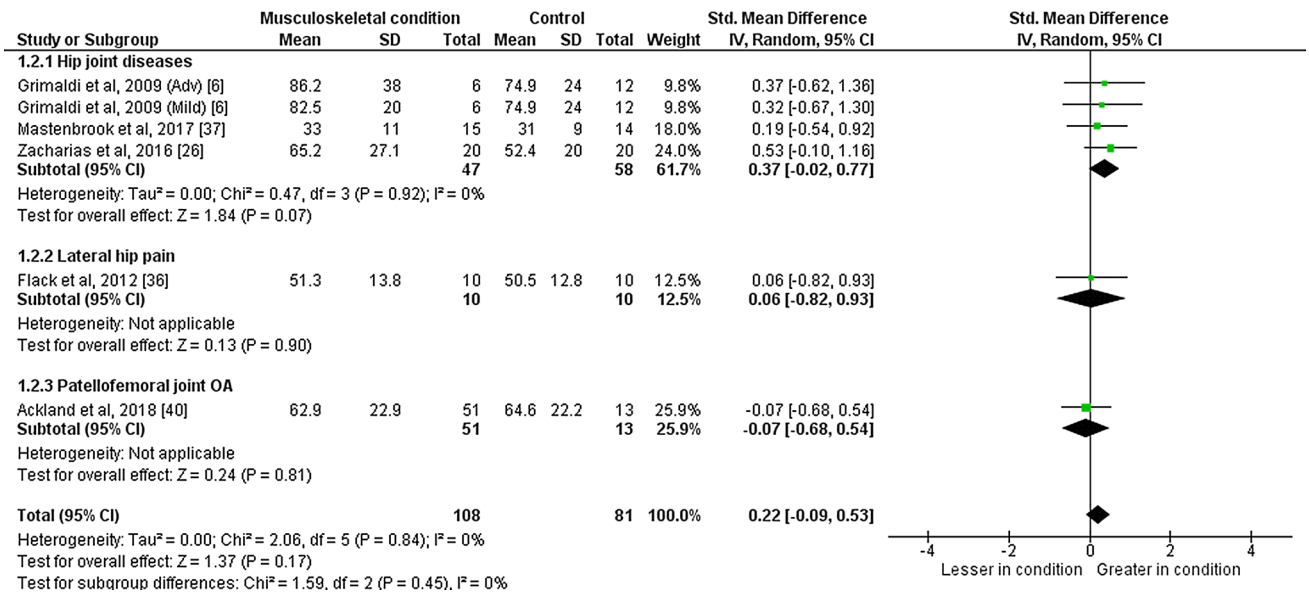

Fig. 4 Muscle volume (unaffected side). Forrest plot detailing standardised mean differences for TFL volume of the unaffected side between individuals with hip joint pathology, and lateral hip pain, and controls. *TFL*, tensor fascia latae

Table 4 Tensor fascia latae muscle activity results ($n=9$)

Author/condition	EMG/mfMRI protocol		Outcome	Musculoskeletal condition			Control group			SMD	95% CI
	Task/EMG electrode type and placement	Data acquisition and processing		Mean	SD	*N*	Mean	SD	*N*		
Dingenen et al. [31] ACL injury	Transition from DLS to SLS (60 degrees of hip flexion) Surface electrodes, based on the instructions of Basmajian and De Luca (1985). The silver–silver chloride, pre-gelled bipolar surface EMG electrodes were placed over the muscle belly and aligned with the longitudinal axis of the muscle, with a centre-to-centre distance of 10 mm. The minimum distance between electrode pairs was set at 3 cm	The raw EMG data were first rectified and then filtered by 6th order Butterworth low-pass filter with a cutoff frequency of 45 Hz to generate the envelope of the EMG. A fixed window of 100 ms before the stance transition (during the DLS phase) was compared with a moving window of the same length along the measurement. An increase of more than 2 SD on top of the average baseline activity was identified as the onset of muscle activity in reaction to the transition	EMG onset time (sec)								
			Eyes open	− 0.12	0.09	15	− 0.16	0.06	15	0.45	[− 0.28, 1,18]
			Eyes closed	− 0.09	0.13	15	− 0.17	0.07	15	0.83	[0.08, 1.58]
Flaxman et al. [38] ACL injury	Isometrically modulated ground reaction forces during a standing task to elicit combinations of sagittal, frontal and transverse plane moments (12 directions) while matching 60% of maximal effort. Participants controlled a projected image of a cursor on a screen by pushing against the force platform with their foot while maintaining equal body weight on each leg Surface EMG were recorded with bipolar electrodes placed over the muscle bellies according to SENIAM guidelines	EMG was sampled at 1000 Hz with a 20 ± 00 Hz bandwidth and a 6 dB/octave filter slope using a 10-channel wireless EMG system. EMG was high-pass filtered at 20 Hz with a second order dual-pass Butterworth filter, full-wave rectified, low-pass filtered at 10 Hz with a 2nd order dual-pass Butterworth filter. EMG was normalised to EMG max of the MVIC trials (EMGexp/EMGmax), and computed as a 50 ms mean about the maximum value in the conditioned EMG signal across all MVIC exercises. EMG, kinematic and kinetic data were time averaged over the half second of successful force target match and ensemble averaged across repetitions	Peak muscle amplitude (EMG/EMG_{max})	0.21	0.10	24	0.21	0.11	24	0.00	[− 0.57, 0.57]
Allison et al. [27] Lateral hip pain (GT)	Walking Bipolar surface electrodes, in parallel with the muscle fibres (inter-electrode distance-20 mm)	EMG signals were amplified 500 times (CMRR > 100 dB) and band-pass filtered (19–1000 Hz). Digitally filtered using a zero-lag fourth order Butterworth between 50–500 Hz, full-wave rectified, and low-pass filtered at 8 Hz. EMG envelopes were normalised to the average of the peak amplitudes of each muscle across the five complete strides cycles. Stride was time-normalised to 100 samples	Muscle synergies	TFL contribute more to the Synergy-1 (across stance phase) than Synergy-2 (early stance).		8	TFL was similarly represented in both synergies		8	NA	NA

Table 4 (continued)

Author/condition	EMG/mfMRI protocol		Outcome	Musculoskeletal condition			Control group			SMD	95% CI
	Task/EMG electrode type and placement	Data acquisition and processing		Mean	SD	*N*	Mean	SD	*N*		
Ganderton et al. [35] Lateral hip pain (GTPS)	Walking TFL surface electrode application and placement was completed using the recommendations of Basmajian and De Luca (1985)	Raw signals were sampled at 2000 Hz (CMRR > 80 dB at 60 Hz; gain of 1000; band pass filtered 20–900 Hz). The surface electrode was high pass filtered at 10 Hz. All data were then full-wave rectified and further processed with a low pass filter (fourth order Butterworth) at a cutoff frequency of 6 Hz to generate linear envelopes. EMG signals were amplitude normalised to the respective peak muscle activation recorded during the gait cycle and time normalised to 100-points (% of gait cycle)	Peak amplitude (%)								
			Stance: 0–30	75.18	25.71	8	65.95	34.80	9	0.28	[− 0.67, 1.24]
			30-TO	88.73	14.96	8	82.96	23.33	9	0.28	[− 0.68, 1.23]
			0-TO	90.73	17.01	8	100.0	0	9	− 0.76	[− 1.75, 0.24]
			Swing	80.57	14.61	8	37.97	20.71	9	2.23	[0.95, 3.51]
			Average amplitude (%)								
			Stance: 0–30	55.87	20.10	8	43.03	25.30	9	0.53	[− 0.44, 1.50]
			30-TO	57.03	8.16	8	48.39	14.93	9	0.67	[− 0.32, 1.66]
			0-TO	57.55	9.95	8	48.16	13.58	9	0.74	[− 0.25, 1.74]
			Swing	45.44	11.79	8	32.77	16.11	9	0.84	[− 0.16, 1.85]
			Stance: 0–30	17.65	2.91	8	16.85	3.50	9	0.23	[− 0.72, 1.19]
			30-TO	17.33	7.55	8	15.07	3.57	9	0.37	[− 0.59, 1.33]
			0-TO	32.99	16.03	8	32.30	14.08	9	0.04	[− 0.91, 1.00]
			Swing	–	–		–	–		–	–
			Variability (CV)								
			Total GC	0.30	0.09	8	0.35	0.13	9	− 0.42	[− 1.39, 0.55]
			0-TO	0.27	0.12	8	0.32	0.11	9	− 0.41	[− 1.38, 0.55]
			0–30	0.26	0.11	8	0.30	0.12	9	− 0.33	[− 1.29, 0.63]
			30–60	0.28	0.14	8	0.35	0.14	9	− 0.47	[− 1.44, 0.50]
Jacobsen et al. [34] Lateral hip pain	Walking Surface electrodes, according to the SENIAM recommendations in a bipolar derivation with Ag/AgCl electrodes with 22 mm of centre-to-centre spacing	EMG signals were band-pass filtered at 20–500 Hz. The raw EMG signals were low-pass filtered with a cutoff limit of 10 Hz to create linear envelopes. The linear envelopes were normalised in amplitude to the maximum EMG signal during walking	% Maximum amplitude								
			Pre activation (50 ms before heel strike)	0.32	0.16	13	0.34	0.12	13	− 0.14	[− 0.91, 0.63]
			Acceptance	0.61	0.15	13	0.68	0.72	13	− 0.13	[− 0.90, 0.64]
			Mid stance	0.34	0.17	13	0.35	0.12	13	− 0.07	[− 0.83, 0.70]
			Late stance	0.23	0.16	13	0.21	0.09	13	0.15	[− 0.62, 0.92]
			Swing phase	0.33	0.16	13	0.26	0.09	13	0.52	[− 0.26, 1.31]

Table 4 (continued)

Author/condition	EMG/mfMRI protocol		Outcome	Musculoskeletal condition			Control group			SMD	95% CI
	Task/EMG electrode type and placement	Data acquisition and processing		Mean	SD	*N*	Mean	SD	*N*		
Sims et al. [29] Hip OA	Reaction time stepping task (controlling balance in the ML direction) Bipolar surface electrodes were placed on the skin over the muscle, parallel to the muscle fibres	The data were band-pass filtered between 20 and 500 Hz and sampled at 1000 Hz. To capture the amplitude of the EMG signal, it was rectified, low pass filtered (50 Hz sixth order Butterworth filter), and the root mean square (RMS) of the EMG signal was calculated from the onset of the ML weight shift towards the stance leg, until foot off. The RMS activity was normalised to the middle 5 sec of EMG activity recorded during a standard submaximal contraction (hip abduction against gravity for 8 sec in side-lying)	RMS amplitude								
			Affected limb	NP	NP		NP	NP		–	–
			Unaffected limb	NP	NP		NP	NP		–	–
Mendis et al. [32] Acetabular labral tear	Standing from a neutral position to 90º hip flexion against resistance (50% MVC) mfMRI was used to investigate the recruitment pattern, by discriminating between activated and non-activated muscle after performance of an exercise	Signal intensity of the TFL muscle was recorded using a region of interest (ROI) of 1 cm^2 on the same slices of interest used for muscle CSA measurement (see Table 3). The ROI was placed in the middle of the muscle and avoided visible blood vessels and fascia. For each subject, average signal intensity of each muscle was measured from baseline and post-exercise scans for each side and the percent change in signal intensity was calculated	% Of change in signal intensity								
			Injured side/dominant	51.0	6.5	12	52.6	5.7	12	− 0.25	[− 1.06, 0.55]
			Non-injured/ Non-dominant	51.1	8.6	12	55.9	7.6	12	− 0.57	[− 1.39, 0.25]
Casartelli et al. [30] FAI	Active flexion of the hip (hip flexion MVC torque) Two pairs of silver-chloride surface electrodes (inter-electrode distance of 25 mm) were placed proximally at 17% on the line from the anterior superior iliac spine to the lateral femoral condyle	EMG signals were amplified with a bandwidth frequency ranging from 10 Hz to 500 Hz (gain 1000) and digitized online at a sampling frequency of 2 kHz. EMG RMS amplitude was calculated during 500 ms around hip flexion MVC torque, using a window length of 125 ms. Only the MVC trial associated to the highest EMG RMS amplitude was retained for analyses	RMS amplitude (μV)	401	251	16	582	323	16	− 0.61	[− 1.32, 0.10]
Baker et al. [2] ITBS	Running trial (from heel strike to peak knee flexion) Bilateral surface EMG was performed with the DelSys Trigno wireless EMG system with a 10 mm intra-electrode spacing, and were placed in the middle portion of the muscle belly	The direct current offset was corrected by subtracting the mean from the average values. A high pass 20 Hz filter was used on the EMG signal. The EMG wave was rectified. A low pass filter was then used on the EMG signal. A RMS analysis was used for one-second moving average. The EMG signal was normalised to MVIC for the running trials	% MVIC amplitude								
			At 3 min run	11	8	15	7	4	12	0.59	[− 0.19, 1.37]
			At 30 min run	8	4	12	6	4	9	0.48	[− 0.40, 1.36]

Table 4 (continued)

Author/condition	EMG/mfMRI protocol		Outcome	Musculoskeletal condition			Control group			SMD	95% CI
	Task/EMG electrode type and placement	Data acquisition and processing		Mean	SD	*N*	Mean	SD	*N*		
Brown et al. [39] ITBS	Overground running trial (speed of 3.35 m/s ± 10%) pre- and post- a treadmill run to fatigue. Bipolar surface EMG (MA-300 EMG System) with a 20 mm inter-electrode distance were applied in parallel with the direction of the muscle fibres and in locations as described by Perotto, Delagi, Iazzetti, and Morrison (1994)	The preamplified EMG signals were low pass filtered with a cutoff frequency of 2000 Hz using the internal low pass filter in the MA-300 system. The EMG data were then sampled at 4800 Hz. The sampled data was then bandpass filtered from 10 to 2000 Hz. EMG data were then full-wave rectified and a linear envelope was created using a low-pass second-order Butterworth filter. To determine a threshold for the onset of muscle activity, the peak activity from all trials (MVIC and dynamic running trials) and the mean EMG value of the resting trial were calculated, and ten percent of the difference between the maximum and the mean resting value was added to the mean resting value and considered the threshold value	Terminal swing activation timing as a percent of the gait cycle								
			Pre-fatigue	95.3	6.0	12	93.5	6.3	16	0.28	[− 0.47, 1.04]
			Post-fatigue	95.8	4.5	12	95.6	2.4	16	0.06	[− 0.69, 0.80]

EMG electromyography, *SD* standard deviation, *SMD* standardised mean differences, *CI* confidence interval, *MVIC* maximal voluntary isometric contraction, *CMRR* common-mode rejection ratio, *CV* coefficient of variation, *ACL* anterior cruciate ligament, *sec* seconds, *ms* milliseconds, *GTPS* gluteal trochanteric pain syndrome, *GC* gait cycle, *0–30* early stance (0–30% gait cycle), *30-TO* late stance (30% to toe off), *O-TO* total stance; swing: toe off to end of the gait cycle, *GT* gluteal tendinopathy, *NP* not provided, *mfMRI* muscle functional magnetic resonance imaging, *FAI* femoroacetabular impingement, *RMS* root mean square, *MVC* maximal voluntary contraction, *ITBS* iliotibial band syndrome

BMI [12, 42] between conditions, other factors require consideration. For instance, reported muscle size can be affected by the selected region of interest from the MRI, and variation in inclusion or exclusion of fat areas within the muscle compartment and outside the epimyseal border [43]. From the three pooled studies for muscle volume on the affected side, only Zacharias et al. [26] excluded the epimuscular fat area when manually tracing the muscle borders. Inclusion of this fat would potentially underestimate any loss of muscle contractile tissue. The volume normalised to body mass in that study was not significant between groups. This highlights the importance of including BMI measures when analysing muscle size. However, two studies [26, 33] estimated the fat content showing similar values between the condition and control groups, which could be explained by the subjective nature of the measurement. Other potential confounders, such as sex and age were mostly accounted for in all studies when matching the control group by these variables. Therefore, (lack of) differences between groups are unlikely to be related to these factors. Other variables that were not consistently measured across studies were physical activity or fitness level and leg dominance, which can also affect the size of a muscle, especially when comparing between individuals with and without pain.

4.2 Activation of the TFL Muscle in Musculoskeletal Conditions

Based on the EMG amplitude measures, the level of TFL muscle activation does not appear to be different for any hip condition. However, care should be taken when comparing studies with different normalisation methods. Normalisation to maximum EMG signals recorded during a particular task enables sensitive comparison of the pattern of change in muscle activation across the task, but precludes comparison of the amplitude of activation between symptomatic and non-symptomatic groups, as it provides no reference of EMG amplitude to a standard value that is comparable between groups (e.g. maximum voluntary contraction) [44]. Even though a small effect for a greater proportion of peak activity during swing phase was found in one study [35], this cannot be interpreted as a greater activity of the muscle during swing phase. However, it does reflect that the muscle activity reduces less, relative to peak activity, during swing than for the controls. Likewise, comparisons of activity levels between muscles, tasks and individuals are not possible to interpret when normalising to a submaximal task [29], because participants might have used a different pattern of muscle activation between the task under investigation and the submaximal task. Further, interpretation of non-normalised EMG amplitude between groups requires caution [30] as the non-normalised amplitude does not control for potential differences of physiological or anatomical characteristics between participants that may affect the raw EMG amplitude (e.g., subcutaneous fat thickness, inter-electrode distance) [48]. On the other hand, Allison et al. [27] analysed the peak normalised amplitude as synergies (group of muscles acting together), showing potential differences in the pattern of muscle activation of the TFL in individuals with lateral hip pain. Whether these findings suggest that the TFL is 'overactive' or not, cannot be determined. The difference in contribution of TLF to the synergy might be related to different movement patterns such as modified hip adduction and hip internal rotation. These movements have been reported to differ between individuals with and without gluteal tendinopathy during walking and single leg stance [45, 46].

Amplitude and timing outcomes have been reported for conditions at the knee (i.e., ACL injury and ITB syndrome). Again, the level of TFL muscle activation (normalised to MVIC) was not different between the condition and controls groups [2, 38]. Although normalisation to MVIC can provide a comparator that enables interpretation of amplitude, the EMG signal recorded in the standardised MVIC may differ from other tasks that involve different contraction types, body positions, and muscle lengths. The capacity of a participant to elicit maximal contraction might also be limited by pain or a specific feature of the condition [50]. Using surface electrodes in dynamic contractions such as in running requires specific considerations, as the electrode might move relative to the muscle and change the orientation of muscle fibres [51]. From the two studies investigating the timing of TFL activation, a greater delay of EMG onset was found in the injured group [34]. Possible causes are argued to be "neuromuscular dysfunction" [47] and/or arthrogenic muscle inhibition post ACL injury [48]. A recently published study [49] suggests that a combination of contractile and non-contractile structures play an important role in dynamic rotatory stabilization of the tibia through attachments to the ITB. These include the complex formed by the TFL/gluteus maximus muscles along with the biceps femoris and the anterolateral ligament complex. This could underpin a role for retraining and prevention of ACL injuries [49], but requires further evaluation. For the second study [49], the onset of the TFL muscle was not different (e.g., as early or delayed onset of activity) in runners with ITB syndrome, and it does not change with a fatigue run. The author's rationale for investigation of onset during swing was that this might explain the increased hip adduction and internal rotation often reported in runners with this condition [17]. Although plausible, other temporal features of the EMG signal (e.g., pattern of muscle activity across the gait cycle) could also explain some of these kinematic differences, but were not measured. EMG recordings from the TFL muscle are likely to be influenced by crosstalk of adjacent muscles (e.g., gluteus muscles). This is problematic if surface electrodes are used because it limits the ability to discriminate the source

of the signal from which the temporal event is identified [50]. Other issues, such as poor signal-to-noise ratio and movement artefact are also common when recording muscle activity from conventional surface electrodes during dynamic tasks [51, 52].

4.3 Considerations

Although this review has an appropriate methodology, interpretation of the findings of this review requires consideration of several methodological issues of the included studies. There are a limited number of studies concerning the potential role of dysfunction of the TFL muscle in any specific condition. This is compounded by with small sample sizes and observations made in different musculoskeletal conditions, which together limit the ability to draw robust conclusions for a particular condition. All studies had a cross-sectional design, limiting the establishment of a causal relationship between any TFL impairment and the musculoskeletal condition. The methodological quality of the included studies varied considerably, and was lower for studies of EMG than muscle structure. Reliability of outcome measures and blinded assessors were poorly described among EMG studies. It is important to consider that the critical appraisal tool employed was adapted from its original version, and the validity of the version used was not independently established. Only measures that provided specific assessment of the TFL muscle were included in this review; therefore, hip strength or ITB tightness were not included.

At a study level, two main issues were identified in EMG studies: the type of electrode used and the amplitude normalisation method employed. All studies used surface electrodes to measure TFL EMG. Crosstalk from adjacent muscles is likely with thin, small muscles such as TFL, which would affect the specificity of the EMG signal [51, 52]. Other electrode types, such as intramuscular electrodes, can be used in these situations but require additional considerations (e.g. safety, placement). Methods to normalise EMG amplitude are necessary when the purpose of the study is to compare the EMG amplitude between groups. Normalisation to peak/mean during task does not allow comparison of amplitudes between groups as the value used for normalisation may differ between groups [44]. Thus, comparison of EMG amplitude is not possible, as these methods do not indicate the degree of muscle activation relative to a standard measure such as MVC [44, 53]. However, normalisation to peak or mean EMG is appropriate for comparison of patterns (e.g. times of peak activation) of muscle activity between individuals [44] and could be interpreted from some studies [27, 35].

Although the methodological quality of muscle structure studies was generally good, the lack of standardised protocols available for the calculation and assessment of muscle size and fat content can potentially lead to misinterpretation of the results (e.g., tracing the fat content, the number of slices included for the estimation of muscle volume) and it may be important to consider when reporting pooling data from systematic reviews. We recommend that future EMG studies should consider the methodological issues raised in this review. First, research should carefully consider the task to assess, and draw on observations from clinical practice to inform their selection. Second, the EMG normalization method should be carefully considered with respect to the comparisons that are to be made. Third, as the TFL muscle is small and thin it is important to consider potential for crosstalk from underlying and adjacent muscles. Intramuscular electrodes may be preferred to overcome crosstalk problems, but other technical and application issues need to be considered [54].

The available literature of direct measures of the TFL muscle structure and activation either refutes or does not allow a clear interpretation of whether this muscle differs between individuals with and without lower limb musculoskeletal conditions. The TFL muscle has often been considered problematic in conditions of the knee (e.g. ITB syndrome, patellofemoral pain) and hip, but this review highlights that there is little evidence from studies of structure and activation that support the clinical notion. A limitation of our systematic review is that the scope was purposefully limited to direct measures of TFL muscle involvement in lower limb conditions. There may be other measures that include the contribution of the TFL muscle that might provide a different perspective of its role in lower limb conditions, but for many (e.g., muscle strength which includes synergist muscles) it is difficult to disentangle the specific contribution of the TFL. Appropriately designed studies that consider clinical observations and precise technical methods to measure muscle function are required. Unfortunately, at present, there is not yet sufficient laboratory evidence to support or to refute consideration of this muscle in clinical practice [9].

5 Conclusion

Based on available evidence of direct measures of the TFL muscle it is difficult to make conclusive statements about its role in the management (assessment and treatment) of lower limb musculoskeletal conditions. Further studies are required with larger sample sizes and appropriate methodologies to better understand the contribution of the TFL in these conditions.

Compliance with Ethical Standards

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Conflict of Interests Manuela Besomi, Liam Maclachlan, Rebecca Mellor, Bill T. Vicenzino, and Paul W. Hodges declare that they have no conflict interest.

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