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Review

Muscle size and composition in people with articular hip pathology: a systematic review with meta-analysis

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SUMMARY

Objective: To synthesise and evaluate the current evidence investigating muscle size and composition in non-inflammatory articular hip pathology.
Methods: A systematic review of five electronic databases, using three concepts; articular hip pathology (e.g., osteoarthritis (OA)); hip muscles; and outcomes (e.g., muscle size and adiposity) was undertaken. Studies addressing non-inflammatory or non-traumatic articular hip pain, using measures of muscle size and adiposity were included and appraised for risk of bias. Data was extracted to calculate standardised mean differences (SMD) and pooled where possible for meta-analysis.
Results: Thirteen cross-sectional studies were included; all studies measured muscle size and 5/13 measured adiposity. In OA, there was low to very low quality evidence of no difference in hip muscle size, compared with matched controls. In unilateral OA, there was low to very low quality evidence of smaller size in gluteus minimus (SMD −0.38; 95% confidence interval (CI) −0.74, −0.01), gluteus medius (−0.44; 95% CI: −0.83, −0.05) and gluteus maximus (−0.39; 95% CI: −0.75, −0.02) muscles in the symptomatic limb. Individual studies demonstrated non-uniform changes in muscle size in OA. No significant difference was observed in muscle size in other pathologies or in adiposity for any group.
Conclusion: There is some low quality evidence that specific hip muscles are smaller in unilateral hip OA. Variation in the magnitude of differences indicate changes in size are not uniform across all muscles or stage of pathology. Studies in larger cohorts investigating muscle size and composition across the spectrum of articular pathologies are required to clarify these findings.
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Introduction

Muscles surrounding the hip are essential for optimal joint loading and stability[1]. Deficits in hip muscle strength are described across the spectrum of articular hip pathologies, including hip osteoarthritis (OA)[2,3], femoroacetabular impingement syndrome (FAIS)[4], labral pathology[5] and dysplasia[6]. Clinical trials to address these deficits have resulted in variable outcomes within OA and other pathologies[7–11], indicating there is still much to learn regarding the relevance of strength deficits in these conditions.

Muscle size is associated with strength[12,13], yet previous systematic reviews of hip pathology have found little or no differences in hip muscle size compared with matched peers[2,3]. The absence of differences in hip muscle size despite differences in strength could have several explanations. First, it may be because strength measures reflect the net force production of a group of muscles, but size is typically measured for individual muscles. Second, individual studies have typically been under-powered to detect differences between groups. Meta-analysis of data from multiple studies may help to resolve these issues. Third, fatty infiltration within muscles can affect force generating capacity, and confound measures of muscle size and function[3,14]. In joint pathology, fatty infiltration can occupy space within healthy muscle tissue, decreasing its potential contractile force in relation to its overall size[15–17]. One previous

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review of muscle composition in hip OA suggested greater adiposity, but this conclusion was drawn from data of only two cohorts, which involved small samples and pooled data from different muscle groups[3]. Additional studies investigating muscle size and composition in OA have been published recently[18,19], providing data with the potential to clarify these findings.

Muscle size and composition have previously been evaluated in advanced OA[2,3]. No review of the literature has considered muscle changes with respect to different grades of pathology or across the spectrum of articular hip pathology that are purported to precede and contribute to the pathogenesis of OA. Identification of changes in muscle structure earlier in pre-arthritic conditions could justify the development of earlier targeted interventions. This review aimed to synthesise and evaluate current evidence regarding muscle size and adiposity in non-inflammatory articular hip pathology in comparison to asymptomatic control populations and limbs.

Methods

The protocol for this systematic review was registered with PROSPERO (CRD42016041771). The search strategy was part of a larger review investigating size, composition and inflammatory markers in articular pathology.

Search strategy

A systematic search was conducted using five electronic databases (MEDLINE, CINAHL, Embase, SPORTDiscus and AMED), up to December 2017. Keyword search terms related to three main concepts; (i) articular hip pathology; (ii) hip muscles, and (iii) outcomes: muscle size, adiposity, fibre types and inflammatory markers. Synonyms within concepts were mapped to subject headings or searched under title and abstract, with subject headings and truncations modified according to database specifications (Supp. Table 1). Database results were combined and exported to Endnote reference management software (Version X7.4) for eligibility screening.

Eligibility

Participants of any age with articular hip pain, of a non-inflammatory or non-traumatic nature were included. Participants of surgical groups were excluded, unless pre-surgical history of non-inflammatory hip pathology was present (Table I). Observational, cohort studies, case–control and pre-intervention groups of clinical trials were included. Reviews, case studies, conference abstracts and protocol papers, as well as non-English language publications were excluded.

Screening

Titles and abstracts of studies were screened by two reviewers (*PRL* and *KJC*). Disagreement in study eligibility was discussed and a consensus reached with the aid of a third reviewer (*AIS*). Full text of the retained studies were obtained for further review of eligibility. Reference lists of included articles were screened for additional eligible articles that had not been identified in the initial search.

Risk of bias

PRL and *KJC* independently used the Epidemiology Appraisal Instrument (EAI)[20], to assess risk of bias, level of blinding, balance of groups, consistency of assessment outcomes and statistical reporting of the included studies. The EAI was modified from forty-three criteria to thirty criteria considered most relevant for the included studies (Supp. Table 2). Criteria were graded according to the objectives of the review rather than the quality and purpose of individual studies. A score from 0 (not satisfied) to 2 (fully satisfied) was applied to each item, with a summary score tallied for individual studies (%) (Table I). Discrepancies in scoring were managed by consensus (*PRL* and *KJC*) and a third reviewer (*AIS*) consulted if no consensus was reached. Studies were then ranked by their overall percentage score, and delineated into quartiles [Q1–4], where the fourth quartile represents studies with the highest score.[21]

Data extraction

Data on study purpose, design, population, and outcome measures were collected by two reviewers (*PRL* and *MGK*) using a standardised data extraction form. Details of muscles assessed, method of outcomes, nature of articular pathology, and type of investigation were recorded. Mean and standard deviation (SD) of the main outcomes, muscle size, adiposity, fibre type and inflammatory markers were extracted (or imputed)[22] to calculate standard mean differences (SMDs) between groups. Where data were presented in graphical form only, it was estimated from figures.

Data analysis

Inter-rater reliability of the EAI was determined with StataIC 13.1 (StataCorp LP, College Station, TX, USA) statistical software. Level of agreement was quantified by Cohen's kappa statistic[23], where >0.90 was considered almost perfect, 0.80–0.90 strong, 0.60–0.79 moderate, 0.40–0.59 weak, 0.21–0.39 minimal and 0.00–0.20 was considered to have no agreement.[24]

Studies were grouped according to pathology (OA, FAIS, labral pathology, dysplasia, Perthes disease). Data from included studies were reported in tables. Qualitative synthesis was conducted on pathological groups where two or more studies were reported. Outcome measures of muscle size and adiposity were grouped for analysis. Where multiple size measures were reported for the same outcome, three-dimensional measures (e.g., muscle volume) were prioritised over CSA/thickness for qualitative synthesis (Table I). If multiple included studies of a similar population and outcome had sufficient homogeneity, quantitative analysis was performed using Review Manager (RevMan) (Version 5.3.5 Copenhagen, Denmark: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014). A SMD with 95% CI was calculated from continuous data, and dichotomous data were presented as odds ratios (OR) with 95% CI. For analysis of outcomes that reported within group, between side comparisons (e.g., symptomatic vs asymptomatic limb), a standardised paired difference (SPD) was calculated using R statistical software (version 3.3.2; https://cran.r-project.org/)[25], metafor package[26]. The SPD and 95% CI were calculated from the sample size, mean and SD of the difference between limbs. An additional requirement for SPD calculation was the between-limb correlation (r)[27,28]. If between limb correlation was not reported, a conservative estimate of r = 0.5 was used[29]. Multiple studies within each outcome (between pathological and control conditions, or between sides), were pooled in a meta-analysis using a random effects model.

SMDs and SPDs of 0.2, 0.5 and 0.8 were interpreted as small, moderate and large effect sizes, respectively[30]. To highlight relative differences in muscle size, the percentage difference between participants with articular pathology and control participants was calculated (percentage difference $= ((A - C)/((A + C)/2)) \times 100$, where A = articular pathology and C = controls). Subgroup analyses were performed for different stages or grades of pathology (e.g., mild and advanced OA). Statistical heterogeneity across the pooled

Table I
Study eligibility criteria

Population			Outcome	
Articular Pathology	Muscle		Size*	Composition†
	Hip	Thigh		
Osteoarthritis	Gluteus	Sartorius	1. Volume	Adiposity
Femoroacetabular impingement	- maximus	Rectus femoris	2. Cross-Sectional Area (CSA)	Qualitative
- Cam	- medius	Vastus	3. Thickness	- Goutallier
- Pincer	- minimus	- lateralis	4. Width	Quantitative
Dysplasia	Obturator	- medialis	5. Circumference	- Radiological density
Legg-Calve-Perthes Disease	- internus	- intermedius		- Echo intensity
Acetabular Labral Pathology	- externus	Adductor		
Slipped capital femoral epiphysis	Piriformis	- longus		
Hip pain	Gemellus	- brevis		
Groin pain	- Inferior	- magnus		
	- Superior	Pectineus		
	Quadratus Femoris	Gracilis		
	Psoas	Biceps femoris		
	- major	Semimembranosus		
	- minor	Semitendinosus		
	Iliacus			
	Iliocapsularis			
	Tensor fascia latae			

Qualitative

- Goutallier system (rating 0–4), (0) normal muscle, (1) the muscle contains some fatty streaks, (2) fatty infiltration is important but more muscle than fat, (3) equal amounts of fat and muscle and, (4) more fat than muscle[18].

Quantitative

- Radiological density, measured in Hounsfield units (HU). A 1% increase in adiposity corresponds to a 0.75–1 HU reduction in density[3].
- Echo intensity, computer assisted grey-scale analysis of the ultrasound image. Expressed as a value between 0 (black) and 255 (white)[40].

* Size measured using methods such as magnetic resonance imaging (MRI), computerised tomography (CT), and ultrasound. Listed in descending order of priority for analysis.

† Adiposity (fatty infiltrate), using qualitative and quantitative grading systems.

data was assessed using an I^2 statistic, with 25% considered low, 50% moderate and 75% as high levels of heterogeneity.[31]

The Grades of Recommendation, Assessment, Development and Evaluation (GRADE)[32,33] approach was modified for observational data[34] and applied to assess the quality of evidence for each meta-analysis. Meta-analysis was graded accounting for indirectedness (downgraded if clinically heterogeneous), imprecision (downgraded if upper or lower CI spanned an SMD or SPD of 0.5 in either direction), risk of bias (downgraded if mean modified EAI scored less than 60%), and inconsistency (downgraded if I^2 was 25% or greater). An overall rating of quality was defined as either high, moderate, low or very low quality evidence[35]. Where individual studies were not sufficiently homogenous to be included in a meta-analysis, a best evidence synthesis was used to provide an overall rating for the body of evidence[36,37]. Grading of the best evidence synthesis was completed using previously published criteria[37,38]. They were graded as strong (≥2 studies with low risk of bias and ≥75% agreement), moderate (≥2 studies including at least one low risk of bias and ≥75% agreement), limited (≥1 moderate/high risk of bias studies, with ≥75% agreement, or one low risk of bias study), conflicting (inconsistent findings <75% agreement), and no evidence.

Results

Study selection

The search strategy identified 4,108 articles as part of a larger review investigating size, composition and inflammatory markers in articular pathology. Following removal of duplicates and screening, 13 articles with a focus on muscle size and adiposity met the eligibility criteria and were included in this review (Fig. 1). Additional studies, identified as part of the search strategy, relating to inflammatory markers will be analysed in a separate systematic review.

Characteristics of included studies

The populations studied included hip OA (nine studies, $n = 202$)[14,18,19,39–44]; dysplasia (two studies, $n = 64$)[45,46]; FAIS (one study, $n = 37$)[46]; acetabular labral pathology (one study, $n = 12$)[47], and Perthes disease (one study, $n = 35$)[48]. Thirteen studies examined muscle size[14,18,19,39–48], and five examined adiposity[18,19,40,41,45] (Table II). Seven studies compared symptomatic groups ($n = 212$) to an asymptomatic control group ($n = 175$) with 39% of participants diagnosed with OA. Six studies compared symptomatic and asymptomatic limbs within individuals ($n = 150$) of which 87% were participants diagnosed with unilateral OA. Approximately two thirds of total participants (63%) were drawn from clinical groups with the remaining participants recruited from community (27%) or pre-surgical groups (10%).

Risk of bias

Overall agreement between assessors was 90%. The median (range) quality score was 53% (34–70%) (Table II). Of the included studies, only 1 (8%) clearly described the participation rate for recruitment [Item 7][46]; 4 (31%) described environmental covariates and confounders [item 12][14,18,42,47], or adjusted for them in the analysis; 3 (27%) reported outcomes for the level of exposure [Item 40][14,18,42]; and 3 (27%) reported the outcomes by subgroups of the study population (Supp. Table 3).[14,18,42]

Deviation from prospero

Thigh muscles were originally included in the planned protocol submitted to Prospero. On completion of the literature search, two studies were identified that investigated size of a thigh muscle

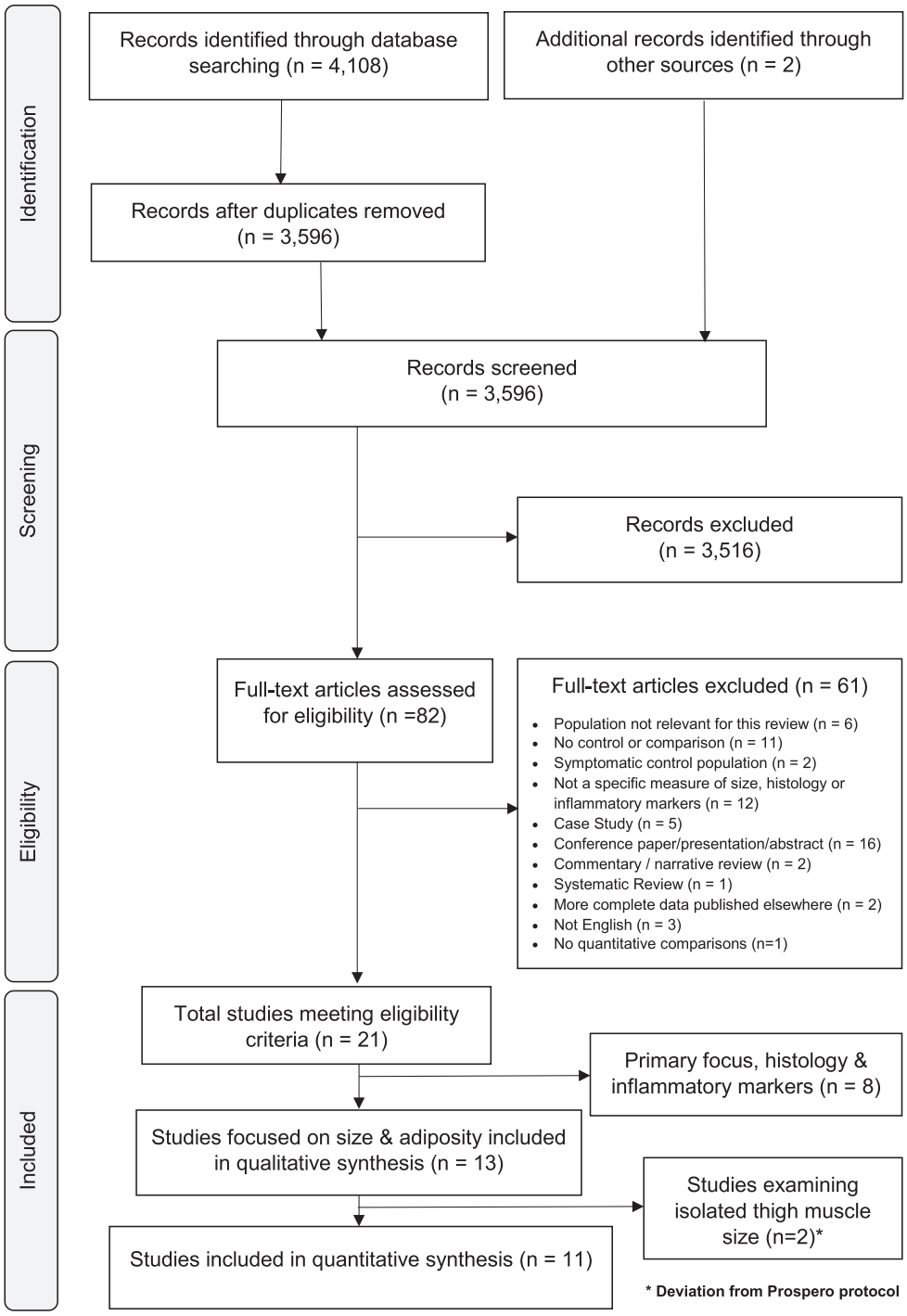

Fig. 1. Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA)[68] flow diagram.

(vastus lateralis)[43,44]. Because of limited data, thigh muscle size was excluded from this review.

Muscle size

Eleven studies investigated muscle size in hip OA[14,18,19,39–42], acetabular labral tears[47], Perthes disease[48], dysplasia[45,46], and pincer FAIS[46] (Table III). The limited evidence from pathologies that were described in single studies are reported in tables (Table V) (Supp. Fig. 2).

Hip osteoarthritis

Seven studies examined symptomatic hip OA (n = 155, 58M); 86% were diagnosed radiographically, with 65% of these participants classified with advanced OA, using the Kellgren and Lawrence (K&L) grading ≥3.

Symptomatic vs asymptomatic participants. Muscle volume (cm^3), measured from magnetic resonance imaging (MRI) in two cohorts, was published in three studies (hip OA: n = 26, 12M; Control: n = 32, 15M) (Fig. 2)[14,18,42]. Pooled data provided low, to very low quality evidence of no significant difference in volume of gluteus

Table II
Summary of included studies

Author (Year)	Population		Whole muscles investigated	Outcome investigated		EAI Quality Score	
	Pathology group	Control group		Muscle size	Muscle composition	Avg. (%)	Quartile
Arokoski (2002)	Hip Osteoarthritis (OA) $N = 27$ [27 males] Age (Mean ± SD) 56.2 ± 4.9 years Diagnosis: Based on American College of Rheumatology criteria (Radiographically), Grade 1 (29.6%), Grade 2 (29.6%), Grade 3 (40.8%)	$N = 30$ [30 males] Age (Mean ± SD) 56.3 ± 4.5 years Diagnosis: Based on American College of Rheumatology criteria, Grade 0 (Nil pain or functional limitation) Control (Contralateral leg)	Lower border of acetabulum • Gluteus Maximus • TFL	Muscle cross-sectional area (CSA)		1.40 (70)	Fourth
Fukumoto (2012)	Hip OA (advanced) $N = 24$ (14 bilateral & 10 unilateral) [0 males] Age (Mean ± SD) 56.8 ± 6.4 years Diagnosis: Radiographic (K&L) - Grade 3/4 ($n = 24$)	Healthy (without hip OA) $N = 16$ [0 males] Age (Mean ± SD) 57.7 ± 6.4 years Diagnosis: Not radiologically confirmed	Gluteus maximus Gluteus medius	Muscle thickness	Echo intensity	0.93 (47)	Second
Grimaldi (2009a)	Hip OA (unilateral) $N = 12$ [6 males] Age (Mean ± SD) 46.5 ± 9.5 years (Gr 1–2), 57.7 ± 6.7 years (Gr 3–4) Diagnosis: Radiographic (K&L), Grade 1–2 ($n = 6$, 3M), Grade 3–4 ($n = 6$, 3M)	Healthy (without hip pain) $N = 12$ [6 males] Age (Mean ± SD) 51.8 ± 9.7 years Diagnosis: Clinical presentation nil pain. Not radiographically confirmed Additional within group comparison with contralateral leg.	Gluteus maximus • Upper • Lower TFL	Muscle volume		1.10 (55)	Third
Grimaldi (2009b)	Hip OA (unilateral) $N = 12$ [6 males] Age (Mean ± SD) 46.5 ± 9.5 years (Gr 1–2), 57.7 ± 6.7 years (Gr 3–4) Diagnosis: Radiographically (K&L) - Grade 1–2 ($n = 6$, 3M), Grade 3–4 ($n = 6$, 3M)	Healthy (without hip pain) $N = 12$ [6 males] Age (Mean ± SD) 51.8 ± 9.7 years Diagnosis: Clinical presentation nil pain. Not radiographically confirmed Additional within group comparison with c ontralateral leg.	Gluteus medius Gluteus minimus Piriformis	Muscle volume		1.10 (55)	Third
Haefeli (2015)	Dysplastic Hips $N = 45$ (45 Hips) [45% (≈20) male] Age (Mean ± SD) 34 ± 10 years Diagnosis: Radiographically (LCE 14–25°) Crowe Classification - Grade 1: ($n = 43$), Grade 2: ($n = 2$) Pincer morphology $N = 37$ (40 hips) [gender not estimable] Age (Mean ± SD) 33 ± 11 years Diagnosis: Radiographically (LCE) >39°)	Healthy (without hip pain) $N = 26$ (30 hips) [gender not estimable] Age (Mean ± SD) 54 ± 12 years Diagnosis: Confirmed with MRI (non-orthopaedic reasons)	Iliocapsularis Rectus Femoris	Muscle CSA, Muscle thickness, Muscle width Muscle circumference		1.07 (53)	Second
Liu (2012)	Developmental Dysplasia of the Hip (DDH) (unilateral) $N = 19$ [7 males] Age (Mean (range)): 47 (35–61) years Diagnosis: Radiographically Crowe Classification - Grade 2 ($n = 8$), Grade 3 ($n = 11$)	Control: Contralateral leg $N = 19$ [7 males] Age (Mean (range)): 47 (35–61) years Diagnosis: Patient reported, examined radiographically but not reported	Gluteus Medius	Muscle CSA	Radiological density (HU)	0.69 (34)	First
Mendis (2014)	Acetabular labral pathology $N = 12$ [4 males] Age (Mean ± SD) 35 ± 12 years Diagnosis: Clinical examination and MRI investigation	Healthy controls $N = 12$ [4 males] Age (Mean ± SD) 35 ± 13 years Diagnosis: Patient reported asymptomatic and MRI investigation	Iliacus Psoas Iliopsoas Sartorius Rectus femoris TFL	Muscle CSA		1.17 (58)	Fourth
Momose (2017)	Hip OA $N = 50$ (12 males) Age (Mean (Range)) 62 (30–80) years Diagnosis: Radiographically confirmed K&L: Grade 2 ($n = 4$), Grade 3 ($n = 8$), Grade 4 ($n = 38$)	Control: Contralateral leg $N = 50$ (12 males) Age (Mean (Range)) 62 (30–80) years Diagnosis: Patient reported unilateral symptoms, nil investigation	Gluteus Medius	Muscle Volume Muscle CSA	Radiological Density (HU)	1.27 (63)	Fourth
Rasch (2007)	Hip OA (unilateral) $N = 22$ [4 males] Age (Mean ± SD) 67 ± 7 years	Control: Contralateral leg $N = 22$ [4 males] Age (Mean ± SD) 67 ± 7 years	Gluteus maximus Psoas Rectus femoris	Muscle CSA	Radiological density (HU)	0.80 (40)	First

(*continued on next page*)

Table II (*continued*)

Author (Year)	Population		Whole muscles investigated	Outcome investigated		EAI Quality Score	
	Pathology group	Control group		Muscle size	Muscle composition	Avg. (%)	Quartile
	Diagnosis: Clinical diagnosis of pre-operative THA. No reports of radiological diagnosis	Diagnosis: Patient reported symptom free. No reported radiological confirmation					
Robben (1999)	Perthes Disease (unilateral) $N = 35$ [gender not estimable] Age (Mean): 5.4 years Diagnosis: Prospective sonographic and radiographically, nil criteria reported	Healthy control $N = 59$ (37 males) Age (Mean (range)): 7.4 (1.7–18.1) years Diagnosis: Patient reported, nil symptoms or previous orthopaedic disease	Rectus femoris	Muscle thickness		0.77 (38)	First
Zacharias (2016)	Hip OA (unilateral) $N = 20$ (9 males) Age (Mean ± SD) 63.4 ± 5.4 years Diagnosis: Confirmed radiographically • Grade 2 ($n = 7$) • Grade 3 ($n = 13$)	Healthy Control (matched) $N = 20$ (9 males) Age (Mean ± SD) 62.1 ± 5.6 years Diagnosis: No radiological evidence of OA Additional within group comparison with contralateral leg.	Gluteus maximus Gluteus medius Gluteus minimus TFL	Muscle volume	Fatty infiltration (Goutallier Classification)	1.13 (57)	Third

Abbreviations: Avg. Average score epidemiological appraisal score out of two; EAI, epidemiological appraisal instrument; CSA, Cross-sectional area; HU, Hounsfield Units; K&L - Kellgren and Lawrence; LCE, lateral centre edge; OA, osteoarthritis; TFL, tensor fascia latae; THA total hip arthroplasty; SD, standard deviation; SMD, standard mean difference.

maximus (GMax), medius (GMed), minimus (GMin) or tensor fascia latae (TFL) muscles between individuals with hip OA and asymptomatic peers (Fig. 2). These non-significant findings remained when participants were sub-grouped according to stage of OA pathology (mild and advanced) (Fig. 3) (low to very low quality evidence, Supp Table 4).

Data from two further studies were unable to be pooled because different methods of measurement were used. In single studies no difference was found for CSA (MRI) of GMax and TFL between individuals with hip OA ($n = 27$; 27M) and controls ($n = 30$, 30M)[39], and no significant difference in GMax or GMed muscle thickness (US) was found between individuals with hip OA ($n = 24$; 0M) and controls ($n = 16$; 0M) (Table III).[40]

Symptomatic vs asymptomatic limb. Four studies compared muscle volume between symptomatic and asymptomatic limbs[14,18,19,42]. There was very low to low quality evidence of significantly smaller GMax (−0.39; 95% CI: −0.75, −0.02), GMin (−0.38; 95% CI: −0.74, −0.01)[14,18,42], and GMed (−0.44; 95% CI: −0.83, −0.05)[18,19,42] muscle volume on the symptomatic side (Supp. Fig. 1). There was no significant difference in muscle volume for TFL (Table IV). When sub-grouped according to stage of pathology, muscle volume was not different between limbs in people with mild OA, whereas GMax was significantly smaller in the symptomatic limb of individuals with advanced OA (moderate effect: 0.55; 95% CI: −0.75, −0.02 (Table IV) (Supp. Fig. 1); low quality evidence (Supp. Table 4)).

Two studies compared CSA between symptomatic and asymptomatic limbs using MRI and CT[39,41]. When combined in a meta-analysis, there was low quality evidence of moderately smaller GMax size (−0.53; 95% CI: −0.83, −0.23), in the symptomatic than asymptomatic limb (Table IV). Data for other muscle groups could not be pooled. Data from single studies showed significantly smaller CSA of TFL using MRI[39], and CSA of psoas and rectus femoris (RF) using CT (Table III).[41]

Dysplasia

Symptomatic vs asymptomatic participants. CSA of RF and iliocapsularis was examined by MRI in dysplasia ($n = 45$) asymptomatic controls ($n = 26$)[46]. There was no significant difference in CSA observed in Iliocapsularis or RF between individuals with dysplasia and controls.

Symptomatic vs asymptomatic limb. One study assessed GMed CSA using CT[45]. The muscle was separated along its length into three planes from proximal to distal. GMed was significantly smaller in all planes; distal (−0.76; 95% CI: −1.27, −0.25), middle (−0.74; 95% CI:−1.25, −0.23) and proximal (−0.59; 95% CI: −1.09, −0.11) (Table V).

Other articular pathology

Single studies compared pincer FAIS ($n = 37$)[46], acetabular labral pathology ($n = 12$)[47] and Perthes disease ($n = 35$)[49], with control populations. There was no difference in CSA in pincer FAIS and acetabular labral pathology using MRI. Muscle thickness measured using ultrasound showed limited evidence of being significantly smaller in a single study ($P < 0.05$) (Table V), based on reported results with SMD's unable to be calculated.

Adiposity

Adiposity was measured in five studies using a range of methods. Qualitative assessment was made using the Goutallier classification in MRI[18], while quantitative measures of adiposity were determined through the use of echo intensity (US)[40], and radiological density (CT)[19,41,45] (Table III).

Table III
Muscle size and composition (Osteoarthritis)

Author, Year	Comparison	Muscles investigated (location within muscle)	Method of Measurement	Outcomes Investigated				
				Muscle Size			Muscle Composition	
					OA vs Control group* SMD[95%CI] (% difference)	Comparison between limbs (OA)		
Arokoski (2002)	OA vs Control	Tensor Fascia Latae (TFL) Gluteus Maximus (Lower border acetabulum)	MRI 1.5T	Cross-sectional area	TFL −0.26 [−0.78, 0.26] (−5.46%) GMax −0.20 [−0.72, 0.32] (−3.49%)	TFL **−0.51 [−0.91, −0.11]** (−13.61%) GMax **−0.49 [−0.89, −0.09]** (−9.12%) (negative, decreased muscle size in OA)		
Fukumoto (2012)	OA vs Control	Gluteus maximus Gluteus medius	Ultrasound	Thickness	GMax −0.29 [−0.93, 0.34] (−5.31%) GMed −0.39 [−1.03, 0.25] (−6.96%)		Echo Intensity	*OA vs Control group* GMax 0.41 [−0.23, 1.05] (5.01%) GMed **0.83 [0.17, 1.49] (13.33%)**
Grimaldi (2009a)†,‡	OA vs Control	Gluteus maximus • Upper (U) • Lower (L) TFL (Origin to Insertion)	Magnetic Resonance Imaging (MRI) 1.5T	Volume	*Mild OA (Gr 1–2)* GMax (U) 0.40 [−0.60, 1.39] (12.02%) GMax (L) 0.08 [−0.90, 1.06] (2.59%) TFL 0.07 [−0.91, 1.05] (2.33%) *Adv. OA (Gr 3–4)* GMax (U) 0.18 [−0.81, 1.16] (5.16%) GMax (L) 0.03 [−0.95, 1.01] (0.88%) TFL 0.17 [−0.82, 1.15] (6.71%) (negative, decreased muscle size in OA)	*Mild OA (Gr 1–2)* GMax (U) −0.21 [-1.01, 0.60] (−3.87%) GMax (L) −0.22 [−0.37, 1.03] (−5.92%) TFL 0.37 [−1.20, 0.45] (11.13%) *Adv. OA (Gr 3–4)* GMax (U) −0.78 [−1.70, 0.13] (−23.57%) GMax (L) −0.62 [−0.25, 1.50] (−21.83%) TFL −0.08 [−0.72, 0.88] (−3.76%)		
Grimaldi (2009b)†	OA vs Control	Gluteus medius Gluteus minimus Piriformis (Origin to Insertion)	Magnetic Resonance Imaging (MRI) 1.5T	Volume	*Mild OA (Gr 1–2)* GMed 0.69 [−0.32, 1.71] (15.16%) GMin 0.04 [−0.94, 1.02] (1.16%) Piriformis 0.00 [−0.98, 0.98] (0.00%) *Adv. OA (Gr 3–4)* GMed 0.00 [−0.98, 0.98] (0.00%) GMin −0.07 [−1.05, 0.91] (−2.35%) Piriformis 0.00 [−0.98, 0.98] (0.00%)	*Mild OA (Gr 1–2)* GMed 0.03 [−0.77, 0.83] (0.54%) GMin −0.24 [−0.58, 1.05] (−8.79%) Piriformis −0.07 [−0.87, 0.73] (−3.51%) *Adv. OA (Gr 3–4)* GMed −0.44 [−1.27, 0.40] (−12.98%) GMin −0.18 [−0.63, 0.98] (−8.00%) Piriformis −0.53 [−1.38, 0.33] (−16.39%)		
Momose (2017)	OA vs Control leg	Gluteus medius	Computed tomography (CT) Trans-axial scans	Volume Cross-sectional area		GMed Vol: **−0.90 [−1.23, −0.57] (−23.17%)** CSA: **−0.83 [−1.15, −0.51] (−20.60%)**	Radiological density (HU)	*Between sides (OA)* GMed **−0.84 [−1.17, −0.52] (−24.41%)**
Rasch (2007)	OA vs Control leg	Gluteus maximus (top of greater sciatic foramen) Psoas (Third lumbar vertebrae) Rectus femoris (20 cm proximal to knee)	Computed tomography (CT)	Cross-sectional area		GMax **−0.57 [−1.02, −0.12]** (−13.91%) Psoas **−0.83 [−1.31, −0.35]** (−21.45%) RF **−0.61 [−1.06, −0.15]**	Radiological density (HU):	*Between sides (OA)* GMax **−0.88 [−1.31, −0.34]** (−57.78%) Psoas

(*continued on next page*)

Table III (*continued*)

Author, Year	Comparison	Muscles investigated (location within muscle)	Method of Measurement	Outcomes Investigated				
				Muscle Size			Muscle Composition	
					OA vs Control group* SMD[95%CI] (% difference)	Comparison between limbs (OA)		
						(−15.59%) (Negative, decrease in size OA)		−0.21 [−0.64, 0.21] (−5.56%) RF −0.14 [−0.56, 0.28] (−1.90%) (Negative, decreased density)
Zacharias (2016)	OA vs control	Gluteus maximus Gluteus medius Gluteus minimus Tensor fascia latae (Origin to Insertion)	Magnetic resonance imaging 3.0T	Volume	*Weight normalised* GMax −**0.73 [−1.38, −0.09]** (−15.38%) GMed −**0.69 [−1.33, −0.05]** (−12.66%) GMin −**1.15 [−1.83, −0.48]** (−31.58%) TFL 0.00 [−0.62, 0.62] (0.00%) *OA (Gr 2)* GMax 0.39 [−0.48, 1.25] (11.12%) GMed 0.40 [−0.47, 1.27] (11.49%) GMin 0.02 [−0.85, 0.88] (0.50%) TFL 0.07 [−0.79, 0.93] (2.64%) *OA (Gr 3)* GMax −0.09 [−0.79, 0.61] (−2.91%) GMed −0.08 [−0.80, 0.64] (−2.15%) GMin −0.53 [−1.24, 0.18] (−17.68%) TFL **0.78 [0.06, 1.51]** (31.44%) *OA (Combined)* GMax 0.06 [−0.56, 0.68] (1.90%) GMed 0.14 [−0.48, 0.76] (3.89%) GMin −0.37 [−1.00, 0.26] (−11.72%) TFL 0.53 [−0.10, 1.16] (21.77%) (negative indicates decreased size in OA)	*Weight normalised* GMax −**0.61 [−1.09, −0.14]** (−12.50%) GMed −**0.48 [−0.94, −0.02]** (−7.79%) GMin −**0.96 [−1.49, −0.43]** (−22.22%) TFL 0.00 [−0.44, 0.44] (0.00%) *OA (Gr 2)* GMax −0.14 [−0.61, 0.88] (−4.61%) Gmed −0.08 [−0.66, 0.82] (−2.84%) Gmin −0.13 [−0.61, 0.87] (−3.65%) TFL 0.02 [−0.76, 0.73] (0.56%) *OA (Gr 3)* GMax −0.52 [−0.06, 1.10] (−16.75%) GMed −0.35 [−0.21, 0.91] (−9.86%) GMin −0.75 [−0.14, 1.37] (−21.36%) TFL −0.02 [−0.53, 0.56] (−0.84%) *OA (Combined)* GMax −0.40 [−0.86, 0.05] (−12.27%) GMed −0.26 [-0.70, 0.19] (−7.34%) GMin −0.55 [−1.02, 0.08] (−14.89%) TFL −0.02 [−0.46, 0.41] (−1.22%)	Fatty Infiltration (Goutallier Classification) Odds Ratio	<u>*OA vs Control group*</u> Increased Fatty infiltration GMax **OR, 15.55 [1.73, 139.65]** GMed OR, 5.54 [0.25, 123.08] GMin **OR, 10.52 [2.27, 48.76]** TFL OR, 3.15 [0.12, 82.16]

Abbreviations: CSA, cross-sectional area; GMax, gluteus maximus; GMed, gluteus medius; GMin, gluteus minimus; HU, Hounsfield Units; K&L – Kellgren and Lawrence; LCE, lateral centre edge; OA, osteoarthritis; OI, obturator internus; ON, Osteonecrosis; OR, Odds ration; RF, rectus femoris; TFL, tensor fascia latae; THA total hip arthroplasty; SD, standard deviation; SMD, standard mean difference. Bold text signifies statistical significance.

* When control data for both the left and right legs is reported, with no statistically significant difference between sides, then the leg with the most conservative difference to the pathological data was used[34].

† When studies reported outcomes for different stages of pathology (e.g.,. mild, advance OA) without reporting on the combined sample, data from the most advanced stage of pathology was included for meta-analysis[34].

‡ If muscles were delineated into separate functional components without providing the whole muscle data, the component most representative of whole muscle size was used.

Table IV
Summary of findings table of osteoarthritis muscle size (within group comparison)

Outcome	Muscle	Stage of Pathology	Participants	I^2 (%)	*P*-value	SMD
Volume	Gluteus Maximus[14,18]	Mild[14,18]	13	0	0.53	−0.18 [−0.72, 0.37]*
		Advanced[14,18]	19	0	0.03	**−0.55 [−1.03, −0.07]***
		Overall[14,18]	32	0	0.04	**−0.39 [−0.75, −0.02]***
	Gluteus Medius[18,19,42]	Mild[18,42]	13	0	0.11	−0.03 [−0.57, 0.51]*
		Advanced[18,42]	19	0	0.91	−0.38 [−0.84, 0.09]*
		Combined[19]	50	–	–	**−0.90 [−1.23, −0.57]***
		Overall[18,19,42]	82	50.6	0.08	**−0.44 [−0.83, −0.05]***
	Gluteus Minimus[18,42]	Mild[18,42]	13	0	0.53	−0.18 [−0.73, 0.37]*
		Advanced[18,42]	19	19.7	0.06	−0.53 [−1.08, 0.03]*
		Overall[18,42]	32	0	0.04	**−0.38 [−0.74, −0.01]***
	Tensor fascia latae[14,18]	Mild[14,18]	13	0	0.87	0.18 [−0.38, 0.73]
		Advanced[14,18]	19	0	0.53	−0.04 [−0.49, 0.41]*
		Overall[14,18]	32	0	0.79	0.05 [−0.30, 0.40]
Cross-sectional area	Gluteus Maximus[39,41]	Overall[39,41]	49	0	0.79	**−0.53 [−0.83, −0.23]***

Abbreviations, SMD, Standard mean difference.
* Negative favours a smaller size in osteoarthritis. Bold font indicates significant finding with the *P*-value of the I^2 (%).

Hip osteoarthritis

Adiposity was examined in hip OA in four studies, but due to methodological heterogeneity, data could not be pooled.[18,19,40,41]

Symptomatic vs asymptomatic participants. Two studies compared hip OA with asymptomatic controls using Goutallier classification[18] and Echo intensity[40] to ascertain adiposity. There was limited evidence that people with OA, had higher GMed adiposity when measured with US echo intensity (0.83; 95% CI: 0.17, 1.49)[40]; but were no different when rated qualitatively (OR 5.54; 95% CI: 0.25, 123.08)[18]. In GMax, qualitative assessment revealed greater adiposity in hip OA than controls (OR 15.55; 95% CI: 1.73, 139.65)[18], but there was no significant difference when measured with echo intensity (0.41; 95% CI: −0.23, 1.05)[40]. Qualitatively, greater GMin (OR 10.52; 95% CI: 2.27, 48.76) adiposity was observed in hip OA than controls in a single study[18] (Table III).

Symptomatic vs asymptomatic limb. Two studies used radiological density to assess adiposity[19,41]. Adiposity of GMax, Psoas and RF was compared between symptomatic hip OA and the contralateral (asymptomatic) limb using CT ($n = 22$, 4M)[41]. Greater adiposity of GMax was found in the symptomatic limb (−0.88; 95% CI: −1.31, −0.34), but there was no difference in either psoas or RF muscles (Table III). When using CT ($n = 50$, 12M) to assess radiological density between unilateral OA and the contralateral asymptomatic limb there was a significantly lesser density in GMed, interpreted as greater adiposity in OA (−0.84; 95% CI: −1.17, −0.52).[19]

Dysplasia

Between limb comparisons in a single study of unilateral dysplasia using CT showed significantly greater adiposity of GMed in the symptomatic limb at three locations; proximal (−1.82; 95% CI: −2.56, −1.09), middle (−2.38; 95% CI: −3.27, −1.50) and distal (−1.86; 95% CI: −2.61, −1.12)[45]. (Table V).

Discussion

This review aimed to synthesise the literature regarding muscle size and adiposity for non-inflammatory articular hip pathologies. The main findings were low to very low quality evidence of no difference in volume of hip muscles between individuals with hip OA and matched asymptomatic peers, regardless of stage of pathology. When compared to the asymptomatic limb, there was low to very low quality evidence that GMin, GMed and GMax muscles were smaller on the side with hip OA. GMax was smaller on the symptomatic side in advanced unilateral pathology. Investigation of adiposity in OA demonstrated conflicting evidence regarding the magnitude of difference, with limited studies in articular hip pathology other than hip OA.

Muscle size

Previous systematic reviews with meta-analysis of muscle size in OA have had inconsistent results[2,3]. This may be explained by the methods in which data from multiple muscle groups, and data derived from different measurement methods was pooled. In the present review, variation in magnitude of size differences were observed in individual studies using both between group (31.44% to −31.58%) and between limb comparisons (11.13% to −23.57%). Some muscles were larger and some smaller in the presence of pathology supporting the hypothesis that muscles may be impacted differently in articular pathology. Muscles have previously been reported to atrophy at different rates[50], so by evaluating separate muscles rather than pooling multiple muscles, we were able to identify subtle variations seen within individual muscles. Furthermore, discrete functional and structural compartments within muscles[51,52] indicate measures of CSA and thickness, particularly when not performed in the same anatomical location, may not be appropriate to pool with muscle volume[2]. This review attempted to address these limitations by only pooling data related to the same muscles and measurement method.

Hip OA vs control

Individuals with hip OA had no significant difference in muscle size when compared with matched asymptomatic peers. The meta-analyses in this review pooled raw muscle volume (cm^3) from two cohorts[14,18,42]. Muscle size is associated with body-weight[53,54], so it is possible that the findings of our meta-analyses were confounded by using un-normalised muscle volume (e.g., normalised to body-weight). Interestingly, only one of the included studies provided additional data that had been normalised to body-weight[18]; with GMin, GMed and GMax being significantly smaller in people with hip OA compared with controls. Normalising raw muscle size measures to body size, as performed by Zacharias *et al.*, may help address some of the limitations associated in comparing symptomatic and control populations and should be considered for analysis in future studies of muscle size.

Hip OA vs asymptomatic limb

In individuals with advanced hip OA, meta-analysis showed moderate to very low quality evidence that the GMax, GMed, and

Table V
Muscle size in other articular pathology

Author, Year	Comparison	Muscles investigated	Method of Measurement	Outcomes Investigated				
				Muscle Size			Muscle Composition	
				Muscle	Method	SMD[95%CI]	Method	SMD[95%CI]
Haefeli (2015)	Dysplasia vs Control	Iliocapsularis Rectus Femoris Location: Height of the femoral head (centre)	Magnetic Resonance Arthrogram (MRA)	Rectus Femoris	CSA	−0.24 [−0.70, 0.22]		
					Thickness	−0.14 [−0.61, 0.32]		
					Width	−0.06 [−0.52, 0.40]		
					Circumference	−0.01 [−0.47, 0.45]		
				Iliocapsularis	CSA	0.16 [−0.30, 0.63]		
					Thickness	**0.51 [0.04, 0.98]**		
					Width	**0.54 [0.07, 1.01]**		
					Circumference	**0.57 [0.10, 1.04]**		
	Pincer (FAI) vs Control	Iliocapsularis Rectus Femoris Location: Height of the femoral head (centre)	Magnetic Resonance Arthrogram (MRA)	Rectus Femoris	CSA	0.44 [−0.04, 0.92]		
					Thickness	0.29 [−0.19, 0.76]		
					Width	**0.71 [0.22, 1.19]**		
					Circumference	**0.71 [0.22, 1.20]**		
				Iliocapsularis	CSA	−0.01 [−0.48, 0.46]		
					Thickness	−0.43 [−0.91, 0.05]		
					Width	**−0.49 [−0.97, −0.01]**		
					Circumference	**−0.61 [−1.09, −0.13]**		
Liu (2012)	DDH vs Control	Gluteus Medius Location: 3 planes (A,B &C) evenly distributed along line from the greater trochanter to height of L5.	Computed tomography (CT) Slices 2.0 mm thick at 2.0 mm intervals	Gluteus Medius	CSA	A: **−0.59 [−1.09, −0..11]** B: **−0.74 [−1.25, −0.23]** C: **−0.76 [−1.27, −0.25]**	Muscle Density (HU)	A: **−1.82 [−2.56, −1.09]** B: **−2.38 [−3.27, −1.50]** C: **−1.86 [−2.61, −1.12]**
Mendis (2014)	Acetabular Labral Pathology vs Controls	Iliacus Psoas Iliopsoas Sartorius Rectus femoris TFL Location: Iliacus & Psoas: Measured from the iliac crest to where the muscles fuse. Iliopsoas, sartorius and TFL: measured spanning the femoral head. Rectus femoris: measured at origin on lesser trochanter	Magnetic resonance imaging (MRI) 1.5 T	Iliacus Psoas Iliopsoas Sartorius Rectus Femoris TFL	CSA	*ALP vs Control* −0.03 [−0.83, 0.77] 0.15 [−0.65, 0.96] −0.05 [−0.85, 0.75] 0.11 [−0.69, 0.91] −0.33 [−1.13, 0.48]		
				Iliacus Psoas Iliopsoas Sartorius Rectus Femoris TFL		0.16 [−0.64, 0.97] *Comparison between limbs* 0.13 [−0.43, 0.70] −0.12 [−0.69, 0.45] 0.00 [−0.57, 0.57] 0.11 [−0.46, 0.69] −0.11 [−0.67, 0.46] 0.12 [−0.45, 0.69]		
Robben (1999)	Perthes Disease vs Control	Rectus Femoris Location: Midpoint between the superior border of the patella and the ASIS	Ultrasound 7 MHz linear array.	Rectus Femoris Rectus Femoris	Thickness	*Perthes vs Control (mm)* 12.3 (16.1) ($P < 0.05$) *Comparison between limbs* 12.3 (14) ($P = 0.2$)		

Abbreviations: ALP, Acetabular labral pathology; ASIS, Anterior superior iliac spine; CSA, Cross-sectional area; FAI, Femoroacetabular impingement; HU, Hounsfeld Units; OA, osteoarthritis; TFL, tensor fascia latae; SD, standard deviation; SMD, standard mean difference.

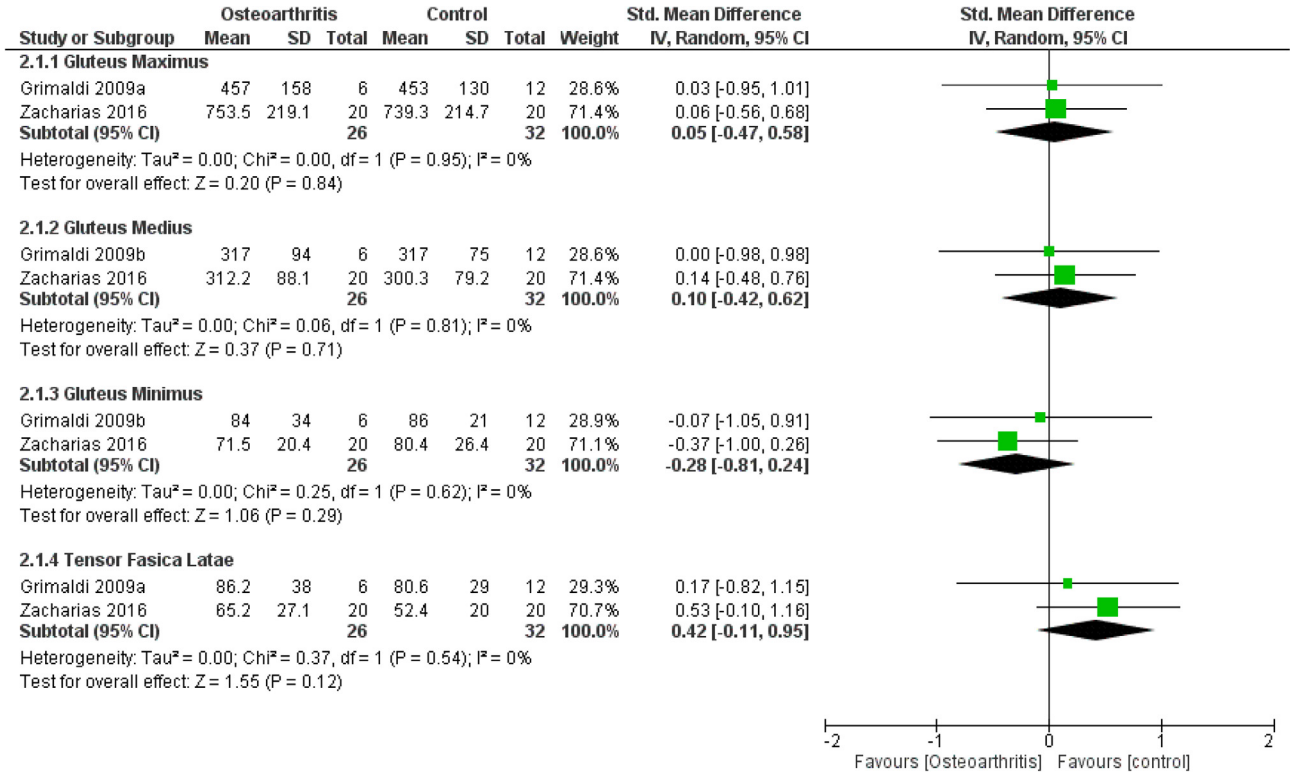

Fig. 2. Forest plot comparing muscle volume in hip osteoarthritis and controls. Abbreviations: CI, confidence interval; IV, Inverse variance; Random, random effects model; SMD, standard mean difference; SD, standard deviation. Individual studies SMD; pooled SMD.

GMin muscles are significantly smaller in the symptomatic limb[14,18,19,42]. Single studies also showed smaller size of TFL[39], RF and psoas[41] muscles. Effects sizes differed between muscles and measurements, supporting the hypothesis that individual muscles are not uniformly affected in hip OA. The generalised asymmetry in muscle size between symptomatic and asymptomatic limbs may also relate to findings with previous reviews, which identified asymmetries in strength and gait patterns in unilateral OA[3,55]. There does appear to be larger differences when observing between limb comparisons, compared to between group comparisons. This may be because between limb comparisons control for person level confounders such as gender and body mass index that could draw effect estimates towards the null in these groups. It is also important to consider that between-limb comparisons may be biased by factors such as modified load sharing between limbs leading to greater size on the unaffected limb than the affected limb.

Stage of pathology

It has been reported that atrophy of abductor muscles (Gmax, GMed and GMin) is related to clinical severity in OA[56]. Sub-group analysis failed to demonstrate any significant difference in muscle size at different stages of OA when compared to a control population (Fig. 3)[14,18,42]. Between limb comparisons in unilateral OA showed a significantly smaller GMax with no difference in GMed and GMin. The potential for atrophy of GMax, in advanced pathology could correspond to observations of lower hip extension angle and increased hip adduction moments in gait in individuals with advanced hip OA[55,57–59]. The small sample size of these studies may contribute to these results highlighting a need for studies with larger sample sizes to identify differences that could be investigated in future longitudinal studies. Should muscular changes be established to be relevant in symptom progression then it provides a target for future interventions to potentially mitigate impairments and impact clinical progression in advanced pathology.

Pathologies other than hip OA

Limited studies considered pathologies other than hip OA, which makes definitive conclusions difficult. Individual studies reported some preliminary indication that hip muscle size might depend on pathology and relate to the specific demands placed on the surrounding musculature by local pathology[45]. Dysplasia and FAIS with pincer morphology, have been linked to compromised and enhanced passive stability of the hip joint, respectively. Data from a single study comparing a control population with pincer FAIS and dysplasia supports the hypothesised role of iliocapsularis in hip joint stabilisation[60,61]. Iliocapsularis showed evidence of significantly smaller size in the presence of FAIS with pincer morphology, which could be interpreted as lower demand on activation of that muscle in groups with enhanced passive stability of the hip[46]. In dysplasia, iliocapsularis[46] was significantly larger, which might be interpreted as greater demand for activation of local muscles in a pathology associated with low passive stability. These findings should be treated with caution and require clarification through additional studies with larger samples sizes and direct measures of muscle activity.[52,60]

Adiposity

All included studies showed significant fatty infiltration of at least one muscle investigated[18,19,40,41]. Greater adiposity of hip muscles in hip OA is consistent with observations for local musculature in other regions affected by articular pathology[16,62]. The variability in adiposity between muscles and studies (2–58%)[41] suggests that not all muscles are impacted in the same way, or that

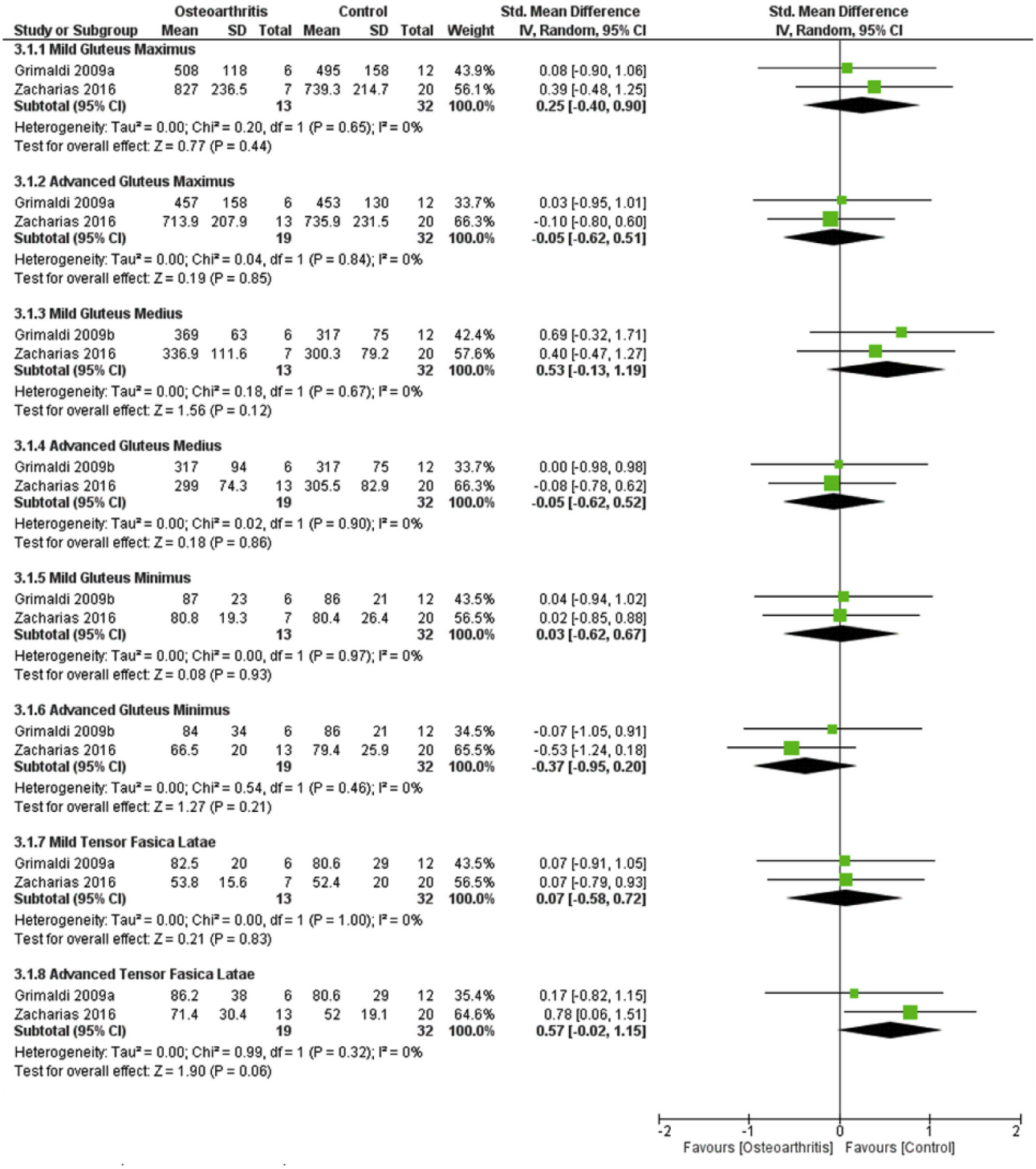

Fig. 3. Forest plot comparing mild and advanced hip osteoarthritis and controls. Abbreviations: CI, confidence interval; IV, Inverse variance; Random, random effects model; SMD, standard mean difference; SD, standard deviation. Individual studies SMD; pooled SMD.

the effect might depend on the stage of pathology[56]. Measurement methods may have also influenced results. Qualitative assessment of adiposity have reported limitations[63], while quantitative measures using ultrasound have also been identified to overestimate adiposity[40]. Recent advancements in image processing enable accurate quantitative assessment of intramuscular fat and should be considered in future research[64]. Evidence that adiposity can vary within the muscle[45,65,66], also highlights the need to clearly specify and standardise the location of measurements used in future studies.

Adiposity in pathologies other than OA, was only undertaken in unilateral dysplasia showing greater levels of GMed adiposity in the symptomatic limb. Findings derived from a single study should be interpreted with caution and highlight the need for additional studies of adiposity in this and other articular hip pathology to elucidate the potential for variations in adiposity.

Limitations and direction for future research

Differences in study design (muscles and measures) and the small number of available studies rendered meta-analysis impossible for conditions other than hip OA. The small sample sizes were indicative of questionable statistical power in the included studies, and this downgraded the level of evidence due to "imprecision". The inclusion of cross-sectional studies means it is impossible to infer causation or that the differences represented a "change" in size or adiposity in association with the pathology. Longitudinal studies are critical to consider possible causality and potential roles in disease progression.

Conclusion

This review identified some low quality evidence of smaller size in specific hip muscles of the symptomatic limb in unilateral OA. It also highlights the variability in the magnitude of difference in hip muscle size between those with hip pathology and those without. Fatty infiltration was identified in multiple muscles and conditions, in limited studies, and consequently it is difficult to draw definitive conclusions. Considering the methodological limitations identified in this review, further work is required in larger cohorts and longitudinal studies to investigate muscle size and composition across various stages of articular pathologies; and to investigate the potential effect of targeted interventions.

Competing interests

None.

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The University of Queensland research scholarship provided funding for Peter Lawrenson in the completion of this systematic review, but was not involved in any aspect of study design, manuscript development or submission.

CRediT authorship contribution statement

P.R. Lawrenson: Conceptualization, Methodology, Data curation, Formal analysis, Writing - original draft, Writing - review & editing. **K.M. Crossley:** Conceptualization, Methodology, Formal analysis, Writing - original draft, Writing - review & editing. **B.T. Vicenzino:** Formal analysis, Writing - review & editing. **P.W. Hodges:** Formal analysis, Writing - review & editing. **G. James:** Formal analysis, Writing - review & editing. **K.J. Croft:** Data curation, Writing - review & editing. **M.G. King:** Data curation, Writing - review & editing. **A.I. Semciw:** Conceptualization, Formal analysis, Writing - original draft, Writing - review & editing.

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Supplementary data

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