Osteoarthritis and Cartilage 24 (2016) 1727–1735

Osteoarthritis and Cartilage

Hip abductor muscle volume in hip osteoarthritis and matched controls

A. Zacharias † § *, T. Pizzari ‡ §, D.J. English † ¶, T. Kapakoulakis ||, R.A. Green † §

† Department of Pharmacy and Applied Science, College of Science, Health and Engineering, La Trobe University, Victoria, Australia
‡ Department of Rehabilitation, Nutrition and Sport, College of Science, Health and Engineering, La Trobe University, Victoria, Australia
§ Sport, Exercise and Rehabilitation Research Focus Area, Australia
|| Bendigo Health Care Group, Bendigo, Victoria, Australia
¶ Fusion Physiotherapy, Bendigo, Victoria, Australia

ARTICLE INFO

Article history:
Received 16 November 2015
Accepted 2 May 2016

Keywords:
Osteoarthritis
Hip
Muscle volume
Stabiliser
MRI
Fatty infiltration

SUMMARY

Objective: Hip abductor muscle strength and function is negatively impacted by the presence of hip osteoarthritis (OA). This study aimed to quantify differences in hip abductor muscle volume, fatty infiltration and strength in a unilateral hip OA population when compared to a control group. Impact of radiographic severity of OA on these variables was also examined.
Methods: Volumes of gluteus maximus (GMax), medius (GMed) minimus (GMin) and tensor fascia lata (TFL) was measured using MRI and muscle volume asymmetry between limbs was calculated. Fatty infiltrate within muscles was graded using the Goutallier classification system. Hip abduction and rotation strength was tested using a dynamometer. Differences between groups or limbs were analysed using *t*-tests and differences in fatty infiltration using non-parametric tests.
Results: A statistically significant decrease in muscle volume was identified in GMax ($P < 0.01$), GMed ($P < 0.02$) and GMin ($P < 0.01$) on the affected side in the OA group compared to both the contralateral side and the control group and differences were related to severity of OA. Hip abduction and internal rotation strength was reduced in the OA group. Increased levels of fatty infiltration were identified in the affected limbs of the OA group for GMax ($P = 0.01$) and GMin ($P = 0.04$).
Conclusion: Gluteal muscle atrophy, increased gluteal fatty infiltration and hip strength deficits were evident in the affected hips of OA participants. Since severity of OA was related to the extent of atrophy and fatty deposits, rehabilitation programs targeting these muscles could reverse or halt the progression of these structural and functional deficits.

Introduction

Osteoarthritis (OA) affects 26% of females and 16% of males aged over 55 years old in Australia[1] and primarily affects weight bearing joints such as the hip and knee. Muscular weakness, joint pain and reduced ambulatory capacity are characteristic of patients with lower limb OA[2]. Atrophy of the muscles around the affected joint has been identified in OA[3] and muscle weakness can be a predictor for the presence of asymptomatic OA[4]. There are suggestions that muscle weakness could be a primary risk factor for OA[5] and atrophy or weakness of the periarticular muscles has been implicated in the development, progression and severity of OA[6]. Considering these findings and perceptions, there has been interest in the activation, size, strength and function of the deep hip stabilising muscles in the presence of hip OA[7].

The gluteus medius (GMed) and minimus (GMin) function as hip abductors and are considered to be the major stabilisers at the hip joint[8]. Hip abductor strength deficits are commonly seen in hip OA patients[9]. Weakness in muscle can be manifested as either a reduction in muscle size or muscle activity[10]. While strength deficits have been identified in hip OA populations when compared to a control, muscle volume (or size) has not been shown to differ between OA patients and controls in a systematic review[9]. Although, there is preliminary evidence to suggest that atrophy of these muscles may only be evident in advanced OA[11].

* Address correspondence and reprint requests to: A. Zacharias, Department of Pharmacy and Applied Science, College of Science, Health and Engineering, La Trobe University, Bendigo, Victoria 3552, Australia. Tel: 61-3-5444-7542.
E-mail address: a.zacharias@latrobe.edu.au (A. Zacharias).

http://dx.doi.org/10.1016/j.joca.2016.05.002
1063-4584/

It is logical that deep hip muscle volume changes exist in OA given the consistent strength deficits identified in this population[9]. The amount of force that can be produced by a muscle is directly proportional to its cross-sectional area (CSA)[12]. However, the total CSA of a given muscle is a measure of both contractile and non-contractile tissue[13] and in muscle atrophy, fatty tissue occupies the space left behind by degenerating muscle fibres[14]. Muscle function can be influenced by the amount of fatty infiltration[15] so it is important to exclude all non-contractile tissue while analysing the total CSA of muscle. This has not been performed to date in studies of hip OA populations.

Therefore the aim of this study was to quantify gluteal muscle atrophy in hip OA patients by comparison of muscle volume with an age- and gender-matched control group. Secondary aims of this study were to relate atrophy of these muscles to severity of OA, and to compare levels of fatty infiltration and hip strength between OA and control groups.

Methods

Participants

Forty participants were included in the study; 20 with unilateral hip OA and 20 age- and gender-matched control participants. Participants were included in the OA cohort if they had radiologically confirmed unilateral hip OA of at least Grade 2[16], were able to walk unaided and scored <40 on the Oxford Hip Score[17] indicating moderate to severe levels of disability. Control group participants were age-matched (±5 yrs) with no radiological evidence of hip OA and scored >40 on the Oxford Hip Score. Participants were excluded from the study if they had contraindications to magnetic resonance imaging (MRI) scanning techniques (e.g., pacemaker, pregnancy or claustrophobia), and other medical, physical or neurological conditions that could lead to changes in the hip abductor muscle.

Unilateral hip OA patients meeting inclusion criteria were identified by a primary practitioner from the Osteoarthritis, Hip and Knee Service at the local hospital or other medical and physiotherapy practices. Control participants were recruited from the local community via advertisements in the local media and were screened for eligibility using inclusion criteria by the primary researcher. The study was approved by the Human Ethics Committee of the University and the local health network (HREC/12/BHCG/40).

Participant characteristics

Demographic data collected included age, weight and height. Hip-related disability was measured using the Oxford Hip Score, a patient-reported outcome measure that has been shown to have good test–retest reliability (ICC (2,1) = 0.89)[18]. Self-reported physical activity was assessed by calculating the activity metabolic index (AMI) using the Minnesota Leisure Time Physical Activity Questionnaire which has been shown to have high test–retest reliability for total activity (r_s = 0.79–0.88) and for subcategories of activity intensity (r_s = 0.69–0.86)[19].

MRI procedure

To control for limb dominance between groups, the muscle volume of the affected limb for each OA participant was compared with the same limb (stance or skill)[20] in the matched control participant. The tested limb in both groups has been referred to as the affected limb. Participants were screened for contraindication to MRI procedures by the MRI technician. Participants were imaged in a supine position with both feet secured to avoid any hip rotation. A Philips Achieve 3.0 Tesla scanner (SENSE XL Torso Coil 16 channel) was used for the MRI procedures. MultiTransmit was used to combat dielectric shading and coil inhomogeneity was corrected by using CLEAR (Philips homogeneity correction algorithm) at the time of image acquisition. A multi-planar localiser scan was performed from above the iliac crest to mid femur to identify the muscles of interest (particularly to be distal to insertion of tensor fascia lata (TFL)). 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 scanning parameters for the axial images were: Field of view = 290 mm × 400 mm, 56 slices of 6 mm slice thickness with 0 mm inter slice gap, repetition time = 715 milliseconds (ms), echo time = 7.1 ms, Voxel size = 0.39 × 0.39 × 6.0 mm.

Muscle volume measurements

Tracings of the gluteus maximus (GMax), GMed, GMin and TFL were performed using Sante DICOM editor software (Santesoft, Athens, Greece). The area of muscle on each slice was calculated by manually tracing individual muscle fascial outlines[11,21]. Any fatty infiltration of the muscle was excluded from the tracings to obtain a measure of only muscle tissue (Fig. 1). The final muscle volume for each muscle was calculated by the summation of the CSA of each muscle and multiplying it by the slice thickness (6 mm). Two assessors independently analysed the muscle volume and fatty infiltrate on both affected and unaffected limbs of five participants to allow the examination of inter-rater reliability.

Rating of fatty infiltration

Rating of the extent of fatty infiltration was conducted for the OA and control group on three consecutive slices using the Goutallier classification system[22]. This system allows classification of infiltration on a rating of 0–4 with; 0 being completely normal muscle, (1) the muscle contains some fatty streaks, (2) fatty infiltration is present but there is more muscle tissue than fat, (3) there are equal amounts of fat and muscle and, (4) being indicative of more fat than muscle[22]. The slices analysed for fatty infiltration for GMax, GMed and GMin were centred at the level of the superior aspect of the greater sciatic foramen and one immediately above and below [23]. The largest part of the TFL muscle belly is situated more inferiorly, and therefore the middle slice for the TFL was at the level of the fovea of head of femur and the two other slices immediately above and below. The average of the Goutallier scores across the three sections indicated the level of fatty infiltration for each muscle[22].

Strength measurement

Hip abduction, internal and external rotation strength was measured using a hand held dynamometer (Lafayette manual muscle test system, Lafayette, IN). The testing protocol was based on techniques used in previous studies[24]. Hip abduction strength was measured in the side lying position and rotation strength was measured with the participant in a sitting position. Resistance was applied just above the malleolus with the participant exerting a 3s isometric maximum voluntary contraction (MVC) against the resistance[24]. Verbal encouragement was given to the participant during each activity. Three trials of each activity was performed and the maximum value from the three trials was used for analysis.

Data analysis

All statistical analysis was completed using IBM SPSS program (Version 22, Chicago, IL USA). Muscle strength was normalized to

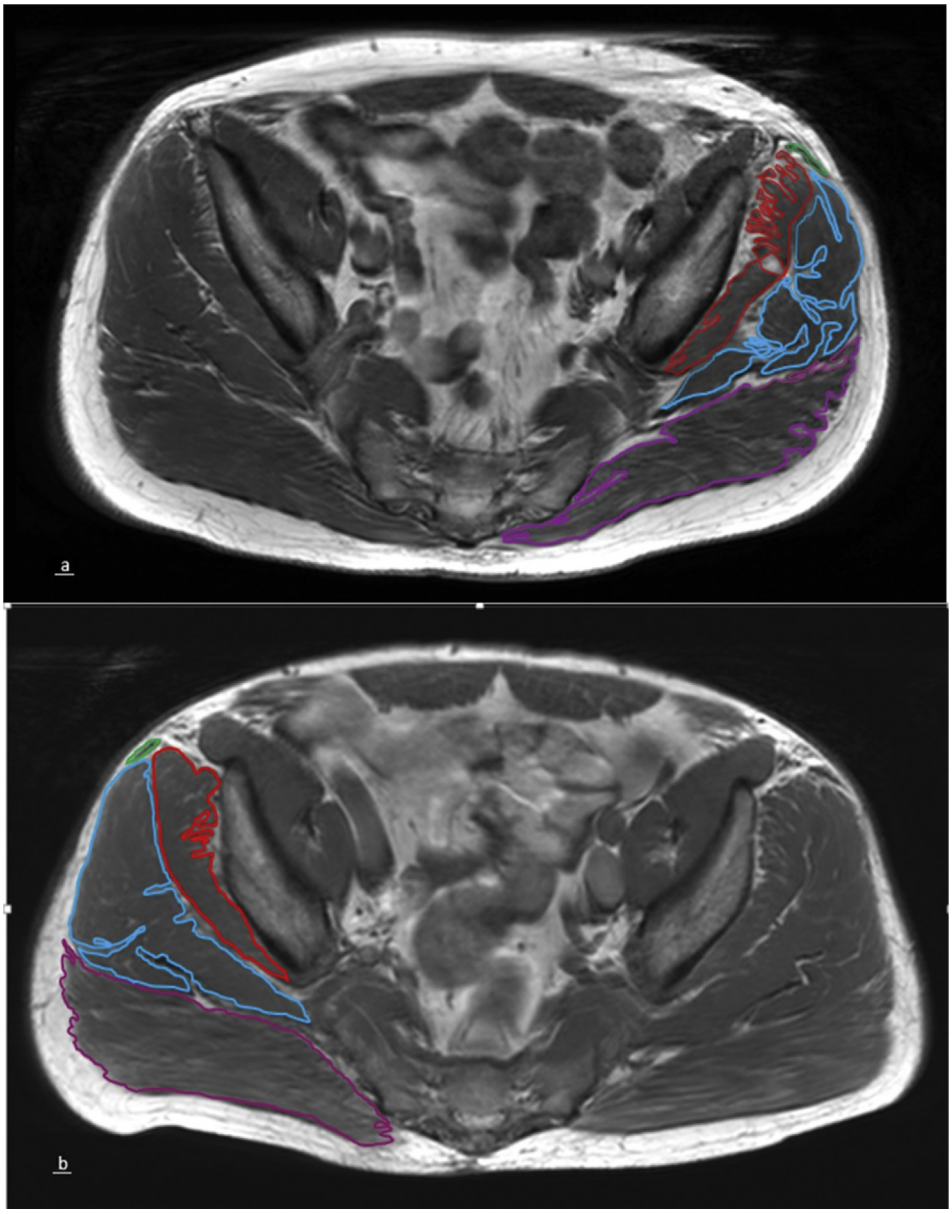

Fig. 1. MRI tracings for muscle volume calculation in GMax, GMed, GMin and TFL; (a) Left hip OA (b) Right hip matched control.

correct for differences in body weight[25]. Since the primary function of the hip abductor muscles is to balance the weight of the body over the stance limb during walking[26] and there was a significant difference in weight between the two groups, muscle volume was also normalized using body weight. Tests of normality (Kolmogorov–Smirnov) were conducted on all continuous data. Levene's test was performed to test for homogeneity of variances between groups and modified degrees of freedom were applied where required. Muscle volume asymmetry between sides in each participant was calculated by subtracting the muscle volume of the affected side from the volume of the unaffected side (unaffected–affected) for each muscle. Effect sizes were calculated for asymmetry between groups by subtracting the mean of the control group from the mean of OA group and dividing it by the pooled standard deviation. Effect sizes were then rated according to Cohen *et al.*[27].

A paired *t*-test was used to compare volume of each muscle between affected and unaffected limb within each group. Student's *t*-test was used to compare side-to-side muscle asymmetry and strength and demographic data between OA and control groups. For further analysis of the influence of OA severity on muscle volumes, the OA group was dichotomised into Grade 2 and Grade 3 OA based on radiological evidence of levels of OA[16]. The muscle volume asymmetries between the two OA groups and the control were then analysed using a one-way analysis of variance (ANOVA). *Post hoc* testing included least significant difference test where the variances were not significantly different ($P \leq 0.05$) and Dunnett's T3 where the variances were significantly different ($P \geq 0.05$).

A chi-square test was used to differentiate the grades of fatty infiltration and cells were collapsed into two groups to minimize cells with an expected total less than 5. Ratings were dichotomised

into ratings of 1 or less (score of 1 or 0 on the Goutallier classification system) and ratings of 2 or more (score of 2–4 on the Goutallier classification system). A rating of 1 could indicate age related changes whereas greater levels of fatty infiltration can be indicative of severe functional impairments[22] and can also result in loss of muscle strength[14]. The Wilcoxon Signed Ranks test was performed to determine difference in fatty infiltrate between affected and unaffected sides within groups and a Kruskal–Wallis test was performed to determine whether there were any differences in levels of fatty infiltration between the two grades of OA and/or the control group.

Inter-rater reliability for muscle volume measurements and rating of fatty infiltrations were calculated using intraclass correlation coefficients (ICC 2, 1) and the root mean square coefficient of variation (CV%). An ICC score of $\geq$0.75 was considered good reliability, a score between 0.50 and 0.75 was considered moderate and a score less than 0.50 was rated as poor inter-rater reliability[28].

Results

Comparison of demographic and strength data

There were no statistical differences in age and height between the two groups (Table I). Participants in the OA group had a higher body mass index (BMI) and a lower Oxford Hip Score and physical activity levels than the control group. The control group had higher strength scores for hip abduction and internal rotation. There were no statistical differences between groups for hip external rotation strength.

Comparison of muscle volumes between affected and unaffected limbs within groups

Decreased muscle volume was identified in the affected limb in the OA group for GMax ($P < 0.01$), GMed ($P < 0.01$) and GMin ($P < 0.01$) but no differences were identified in TFL ($P > 0.05$) (Table II). No asymmetry was identified between limbs in the control group for any muscles ($P > 0.05$).

Comparison of muscle volumes between groups

Greater asymmetry with reduced volume of the affected limb with medium to large effect sizes was present in the OA group when compared to the control group for GMax ($t(26.2) = 3.886$, $P = <0.001$, effect size = 1.37), GMed ($t(38) = 2.325$, $P = 0.02$, effect size = 0.75) and GMin ($t(38) = 3.249$, $P = 0.003$, effect size = 1.08) but no asymmetry was identified for TFL ($t(38) = 0.024$, $P = 0.98$, effect size = <0.01) (Table III). No differences were identified in muscle volumes of the unaffected limbs between groups for GMax ($t(38) = -0.148$, $P = 0.88$), GMed ($t(38) = -1.23$, $P = 0.22$), GMin ($t(38) = -1.36$, $P = 0.18$) and TFL ($t(38) = 0.499$, $P = 0.62$). The raw muscle volumes for the participant groups are presented in Appendix 1.

There was statistically significant muscle atrophy on the affected side in the grade 3 OA group compared to the control groups for GMax ($F(2, 37) = 11.048$, $P < 0.01$), GMed ($F(2, 37) = 4.353$, $P < 0.01$) and GMin ($F(2, 37) = 8.511$, $P < 0.01$) but there were no differences in TFL ($F(2, 37) = 0.000$, $P = 0.80$) (Fig. 2). There was greater muscle atrophy on the affected side in the grade 3 OA group compared to the grade 2 OA group for GMin ($F(2, 37) = 8.511$, $P = 0.02$) but no differences were identified between the two groups in GMax ($F(2, 37) = 11.048$, $P = 0.19$), GMed ($F(2, 37) = 4.353$, $P = 0.09$) and TFL ($F(2, 37) = 0.000$, $P = 0.98$). No muscle atrophy was identified when comparing the grade 2 OA groups and the control group ($P > 0.05$ for all muscles).

Comparison of levels of fatty infiltrate between affected and unaffected limbs within groups

Comparison of limbs in the OA group identified higher levels of fatty infiltration in the affected limb for GMin ($Z = -3.162$, $P = 0.002$). There was also a trend towards higher levels of fatty infiltrate within the affected limb for GMax in the OA group ($Z = -1.732$, $P = 0.083$). No differences were identified when comparing the affected and unaffected limbs in the control group.

When dichotomised into the different grades of OA, increased levels of fatty infiltrate were identified in the affected limb of the grade 3 OA group for GMin ($Z = -3.000$, $P = 0.003$) but no difference in levels of fatty infiltrate was identified within GMax, GMed or TFL. There were no differences identified in levels of fatty infiltrate when comparing the affected and unaffected limbs for the grade 2 OA group.

Comparison of levels of fatty infiltrate between groups

The higher levels of fatty infiltration identified in the affected limbs of the OA group were statistically significant for GMax (χ^2 (1) = 6.53, $P = 0.01$), and GMin (χ^2 (1) = 8.438, $P = 0.04$) when compared to the control group but no differences in fatty infiltration were identified for GMed (χ^2 (1) = 0.526, $P = 0.468$) and TFL (χ^2 (1) = 0.00, $P = 1.0$).

Higher levels of fatty infiltration were also identified in the affected limbs of the grade 2 OA group for GMax ($P < 0.01$) when compared to the control group (Fig. 3). There were also higher levels of fatty infiltration in the affected limbs of the grade 3 OA group for GMin ($P < 0.01$) when compared to the control group but no significant differences in were identified for GMed and TFL. Full Goutallier ratings of level of fatty infiltration for all groups are shown in Appendix 2.

Inter-rater reliability was good for both muscle volume with ICC values ranging from ICC 0.80–0.98 and rating of fatty infiltration (ICC = 0.90). The CV% for all measures were <10% except for TFL (11.0%).

Discussion

The current study has identified significant affected side muscle atrophy with medium to large effect sizes in an OA population when compared to the contralateral side and also when compared to an age and gender-matched control group for GMed, GMin and GMax. Although, when dichotomised into grade of OA, only the

Table I
Participant characteristics

	OA Mean (SD) $n = 20$	Control Mean (SD) $n = 20$	P
Age (years)	63.4 (5.4)	62.1 (5.6)	0.47
Gender (% female)	55%	55%	–
Height (cm)	165.8 (8.3)	167.5 (9.6)	0.57
Weight (kg)	83.0 (18)	69.7 (9.7)	0.006
BMI (kg/m^2)	30.0 (5.2)	24.8 (2.8)	<0.001
Activity metabolic index	39.5 (40.8)	131.2 (75.3)	<0.001
Oxford hip score	24.3 (8.7)	47.0 (2.2)	<0.001
External rotation strength (N/kg)*	0.12 (0.06)	0.16 (0.09)	0.15
Internal rotation strength (N/kg)*	0.12 (0.07)	0.17 (0.07)	0.04
Abduction strength (N/kg)*	0.15 (0.09)	0.25 (0.10)	0.004

* Affected limb.

Table II
Difference in muscle volume (unaffected–affected) within groups

	Total OA ($n = 20$) Mean (SD) % difference	Grade 2 OA ($n = 7$) Mean (SD) % difference	Grade 3 OA ($n = 13$) Mean (SD) % difference	Control ($n = 20$) Mean (SD) % difference
GMax (cm^3)	98.4 (111.4)	38.9 (90.6)	130.5 (111.2)	−4.3 (48.6)
	11.5%*	4.4%*	15.4%*	0.5%
GMed (cm^3)	23.5 (29.2)	9.6 (22.0)	30.9 (30.6)	5.2 (19.2)
	6.9%	2.7%	9.3%*	1.7%
GMin (cm^3)	11.4 (14.7)	−2.3 (23.7)	16.0 (11.0)	−1.0 (8.4)
	13.7%*	2.7%*	19.4%*	1.2%
TFL (cm^3)	0.65 (12.5)	−0.2 (7.4)	1.1 (14.7)	−0.4 (7.0)
	0.9%	0.3%	1.5%*	0.7%

* $P < 0.01$.

Table III
Normalised muscle volumes and muscle asymmetry (unaffected–affected limb) for control and OA group

	Total OA ($n = 20$) mean (SD)			Control ($n = 20$) mean (SD)			Effect size
	Affected hip	Unaffected hip	Asymmetry	Affected hip	Unaffected hip	Asymmetry	Asymmetry between groups
GMax (cm^3/kg)	9.0 (1.5)*	10.2 (2.1)	1.2 (1.4)†	10.5 (2.4)	10.3 (2.4)	−0.1 (0.6)	1.37
GMed (cm^3/kg)	3.7 (0.6)*	4.0 (0.6)	0.2 (0.3)†	4.2 (0.8)	4.3 (0.8)	0.0 (0.2)	0.75
GMin (cm^3/kg)	0.8 (0.2)*	1.0 (0.2)	0.1 (0.1)†	1.1 (0.3)	1.1 (0.3)	−0.0 (0.1)	1.08
TFL (cm^3/kg)	0.7 (0.2)	0.7 (0.3)	−0.0 (0.1)	0.7 (0.2)	0.7 (0.2)	−0.0 (0.1)	<0.01

* Comparison of affected or unaffected limbs between groups, $P < 0.05$.
† Asymmetry comparison between groups, $P < 0.05$.

grade 3 OA group demonstrated significant atrophy of the gluteal muscles when compared with the control group. The influence of the severity of OA was also evident in the GMax and GMin muscles, with greater levels of asymmetry in grade 3 when compared to grade 2 OA patients. There were greater levels of fatty infiltration seen in GMax and GMin in the OA group overall when compared to the control group and decreased abductor and internal rotation strength in the OA group when compared to the control group.

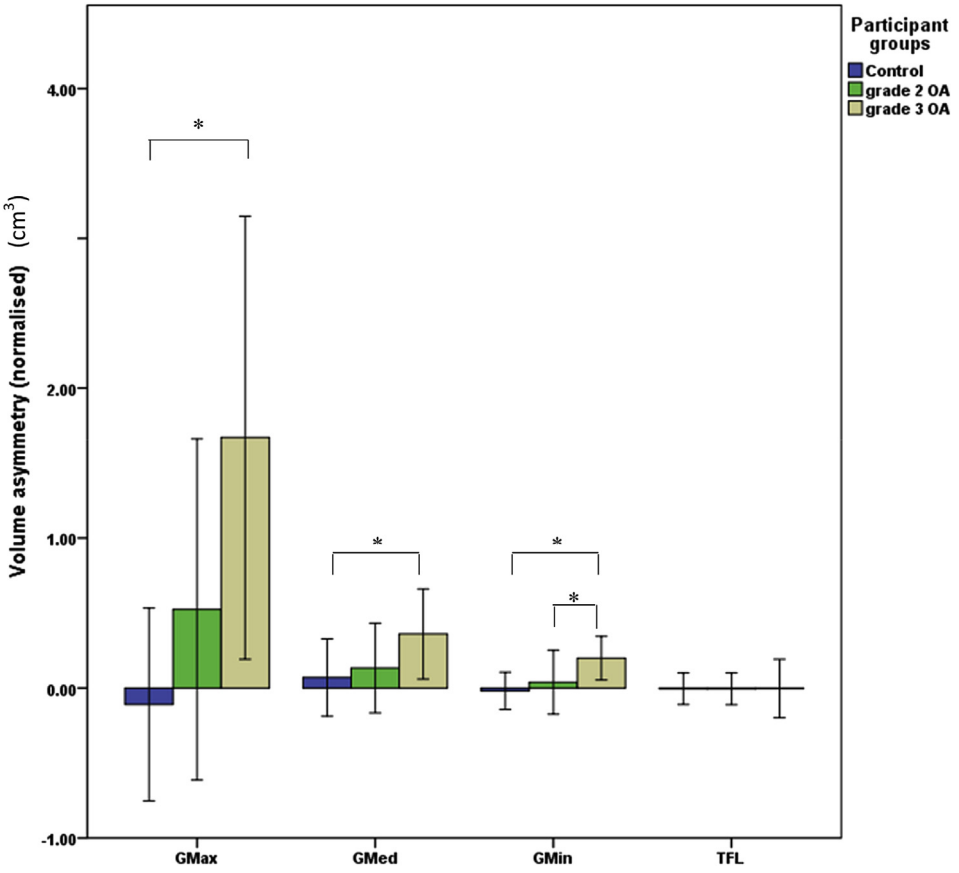

Fig. 2. Volume asymmetry (unaffected–affected) normalised data comparisons based on radiological severity of OA; Mean (SD), GMax, GMed, GMin, TFL, *($P < 0.01$).

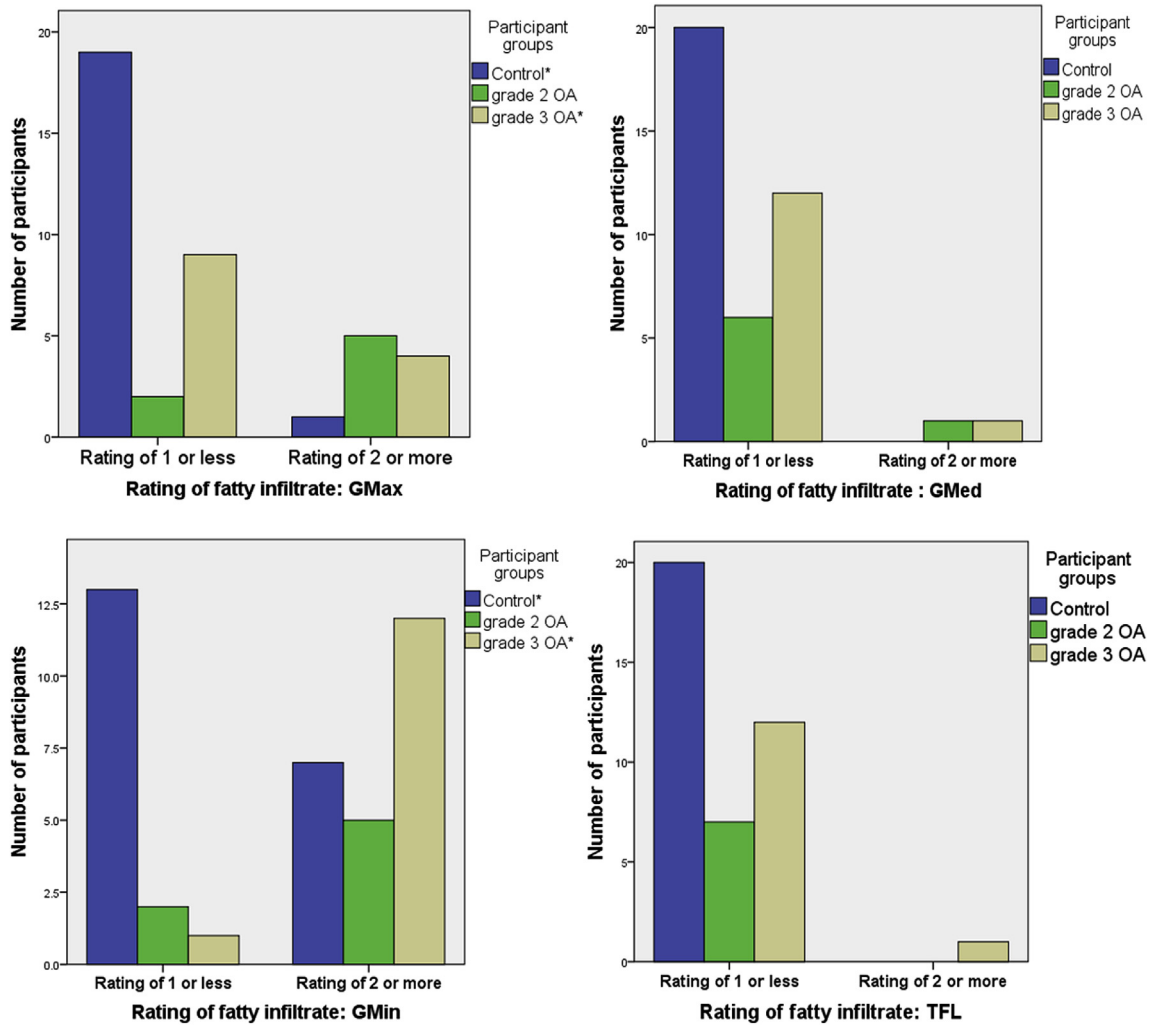

Fig. 3. Rating of fatty infiltration based on severity of OA in GMax, GMed, GMin and TFL; *difference between groups, $P < 0.01$.

There were no changes identified in TFL which is consistent with previous literature[21].

Decreased muscle volume and infiltration of non-contractile tissue around the affected joint could be a result of functional disuse of these muscles and this is consistent with the lower activity levels found in the OA group in this study. Alternatively, the asymmetry identified might be assumed to be a result of hypertrophy of the muscles on the unaffected side as this side becomes the favoured limb for weight bearing function[11]. However, the lack of muscle volume difference in the unaffected limb in the current study confirms that the asymmetry was a result of atrophy in the affected limb, with a significant loss of muscle volume in the OA group when compared with the control group in all gluteal muscles analysed in this study.

GMed and GMin atrophy has been identified in normal ageing, with atrophy present in approximately one third of people aged between 60 and 69 years and one half of the population over 70 years[29]. The progression of atrophy in advanced OA has been identified previously in unilateral OA[11] although no significant differences were identified in a meta-analysis when comparing either an OA and control group or affected and unaffected sides[9]. The inconsistent findings in this review could be because the included studies either failed to account for fatty infiltration in the muscles[11,21], had small participant numbers[10,21], failed to normalise muscle volumes[2,30] or included participants with bilateral OA[30]. All of these methodological problems have been eliminated in the current study since muscle volume was calculated after exclusion of surrounding non-contractile tissue and muscle volumes were normalised to account for body weight differences between groups.

Disability seen in hip OA is directly linked to loss of muscle strength and joint range of motion[31]. The decreased strength of hip abduction and internal rotation in the OA population in the current study is consistent with the decreased muscle volumes in the GMed and GMin. The GMed and GMin are important hip joint stabilisers with GMed being responsible for the stabilisation of the hip and pelvic rotation during gait[8] and GMin stabilising the head of the femur within the acetabulum during the gait cycle[32]. Muscle dysfunction could therefore lead to lack of pelvic stability and antalgic gait, which are typically seen in hip OA.

This study also identified significant atrophy in GMax in the affected limb. GMax plays an essential role in bipedal locomotion and reduced functioning of the GMax can compromise many aspects of the gait cycle[33] and other everyday activities such as sit to stand[34]. The upper fibres of the GMax act along with the hip abductors[21,35] during loading and single limb support[35], while the

lower fibres of GMax are considered as a major hip extensor and external rotator[35] and are also active at heel strike during gait, helping to absorb ground reaction forces[21]. A decrease in hip extension during the late stance phase of gait has been identified as a key feature of gait in a population with hip OA[36] and can be an important determinant of disease progression[37]. Atrophy of the GMax may be a response to the reduced stimulus as a result of the declining hip extension movement. Such disuse atrophy may be proposed to occur predominantly or initially in the lower GMax given its role as a hip extensor. This hypothesis is supported in a previous study[21] that identified atrophy in the lower fibres of the GMax in a group of OA participants with no significant changes in the upper fibres of GMax in the affected limb.

The current study identified significant atrophy in the GMax, but no differences were identified in hip external rotation strength when comparing the two groups This could be due to the fact that there are other agonist external rotator muscles in the seated position (e.g., piriformis)[38] with the GMax being more active in activities involving extension[39]. Comparison of limbs in unilateral OA has identified decreased hip extension strength in the affected limb[2] but no significant differences were identified when comparing an OA and a control group[30] although the strength results were not normalised to body weight and this could explain the null finding.

It has been reported that fatty infiltration into muscle increases with age[29,40]. Fatty infiltrate can be due to disuse in these muscles[41], denervation of muscles[42] and as a result of certain metabolic disorders[43]. Levels of fatty infiltration in the control group in the current study were similar to previous reports[29]. However, the current study also identified significantly increased levels of fatty infiltrate in the OA group compared to controls. Disuse of muscle can lead to structural changes such as an increase in intramuscular fatty tissue that could lead to a decrease in muscle strength and increased levels of disability[44] and reduced mobility[45], all common signs in populations with hip OA. Development of rehabilitation programs that target these muscles in early OA may potentially halt further atrophy.

The fact that potential confounding characteristics were not included in the in the analysis could be considered a limitation of this study, but key confounders of age and gender were accounted for in the study design. Despite the statistical differences identified in this study the clinical significance of the muscle volume atrophy is not known. However, clinical relevance could be implied by the moderate to large effect sizes[46] and the presence of muscle strength deficits in the OA group. Another limitation of this study was that the muscles were not separated into segments for the analysis of volumes and fatty infiltrate. Unique functional segments have been identified in the hip stabiliser muscles[47,48] and therefore analysis of muscle segments could allow better understanding of atrophy in OA populations. Previous studies[29,49] have identified fatty infiltrate in specific segments of the hip abductor muscles but the segmentation taken into consideration is arbitrary and the methodology has not been validated. Further analysis using intramuscular electromyography could help identify loss of function in these individual muscle segments. The failure to measure hip extension strength is also a limitation of this study as it is not possible to link the finding of reduced volume in GMax to potential functional deficits. It is recommended that hip extension strength be measured in future research with hip OA populations. Finally, given that clinical severity (e.g., pain/function) does not always correlate well with radiographic signs[50] future research could examine the relationship between clinical severity and muscle atrophy.

Conclusion

This study identified reduced muscle volume of GMax, GMed and GMin in the affected side of a group of OA participants when compared with matched controls and increased fatty infiltration of GMax and GMin. These changes were more pronounced with increased severity of OA and may be related to decreased hip abduction and internal rotation strength.

Author contributions

Zacharias, A was involved with conception and design of the study, recruitment of participants, data collection, analysis and interpretation of data, drafted and revised the manuscript and also gave final approval for the version to be submitted.

Pizzari, T was involved with conception and design of the study, helped in data analysis, interpretation of data, revised the manuscript for important intellectual content and gave final approval for the version to be submitted.

English, D assisted with the data collection, revised the manuscript for important intellectual content and gave final approval for the version to be submitted.

Kapakoulakis, T was involved with recruitment of participants, revised the manuscript for important intellectual content and gave final approval for the version to be submitted.

Green, RA was involved with conception and design of the study, assisted with data collection, helped in analysis and interpretation of data, revised the manuscript for important intellectual content and gave final approval for the version to be submitted.

Conflicts of interest

None.

Acknowledgments

The authors wish to acknowledge Amanda Hunt (MRI Supervisor Radiographer, High Street Xray, Bendigo) for providing expert opinions on the manuscript. The authors also wish to acknowledge the La Trobe University, Sport,Exercise and Rehabilitation Research Focus Area for funding this project.

Appendix 1. Raw (non-normalized) muscle volumes for all groups

	Total OA ($n = 20$) Mean (SD)		Grade 2 OA ($n = 7$) Mean (SD)		Grade 3 OA ($n = 13$) Mean (SD)		Control ($n = 20$) Mean (SD)	
	Affected	Unaffected	Affected	Unaffected	Affected	Unaffected	Affected	Unaffected
GMax (cm^3)	753.5 (219.1)	852.0 (247.1)	827.0 (236.5)	866.0 (249.9)	713.9 (207.9)	844.4 (255.5)	739.3 (214.7)	735.0 (231.5)
GMed (cm^3)	312.2 (88.1)	336.0 (90.2)	336.9 (111.6)	346.6 (99.7)	299.0 (74.3)	330.0 (88.3)	300.3 (79.2)	305.5 (82.9)
GMin (cm^3)	71.5 (20.4)	83.0 (19.4)	80.8 (19.3)	83.8 (20.9)	66.5 (20.0)	82.4 (19.5)	80.4 (26.4)	79.4 (25.9)
TFL (cm^3)	65.2 (27.1)	66.0 (34.0)	53.8 (15.6)	53.5 (17.5)	71.4 (30.4)	72.0 (38.7)	52.4 (20.0)	52.0 (19.1)

Appendix 2. Rating of fatty infiltration between participant groups

	Grade 2 OA ($n = 7$)					Grade 3 OA ($n = 13$)					Control ($n = 20$)				
Rating of fatty infiltrate	0	1	2	3	4	0	1	2	3	4	0	1	2	3	4
GMax affected limb (no. of participants)	0	2	5	0	0	0	9	4	0	0	0	19	1	0	0
GMed affected limb (no. of participants)	0	6	1	0	0	0	12	1	0	0	1	19	0	0	0
GMin affected limb (no. of participants)	1	1	5	0	0	0	1	11	0	1	0	13	6	1	0
TFL affected limb (no. of participants)	0	7	0	0	0	1	11	1	0	0	1	19	0	0	0

References

1. Australian Institute of Health and Welfare. A Snapshot of Arthritis in Australia. Canberra: AIHW; 2010. 2010 Contract No.: Cat. no. PHE 126.
2. Rasch A, Byström AH, Dalena N, Berg HE. Reduced muscle radiological density, cross-sectional area, and strength of major hip and knee muscles in 22 patients with hip osteoarthritis. Acta Orthop 2007;78(4):505–10, http://dx.doi.org/10.1080/17453670710014158.
3. Sirca A, Susec-Michieli M. Selective type II fibre muscular atrophy in patients with osteoarthritis of the hip. J Neurol Sci 1980;44:149–59, http://dx.doi.org/10.1016/0022-510X(80)90123-9.
4. Slemenda C, Brandt KD, Heilman DK, Mazzuca S, Braunstein EM, Katz BP, *et al*. Quadriceps weakness and osteoarthritis of the knee. Ann Intern Med 1997;127:97–104, http://dx.doi.org/10.7326/0003-4819-127-2-199707150-00001.
5. Shakoor N, Furmanov S, Nelson DE, Li Y, Block JA. Pain and its relationship with muscle strength and proprioception in knee OA: results of an 8-week home exercise pilot study. J Musculoskelet Neuronal Interact 2008;8(1):35–42.
6. Hurley MV. The role of muscle weakness in the pathogenesis of osteoarthritis. Rheum Dis Clin North Am 1999;25(2):283–98, http://dx.doi.org/10.1016/S0889-857X(05)70068-5.
7. Grimaldi A. Assessing lateral stability of the hip and pelvis. Man Ther 2011;16(1):26–32, http://dx.doi.org/10.1016/j.math.2010.08.005.
8. Gottschalk F, Kourosh S, Leveau B. The functional anatomy of tensor fasciae latae and gluteus medius and minimus. J Anat 1989;166:179–89.
9. Marshall AR, deNoronha M, Zacharias A, Kapakoulakis T, Green RA. Structure and function of the abductors in patients with hip osteoarthritis: systematic review and meta-analysis. J Back Musculoskelet 2016, http://dx.doi.org/10.3233/BMR-150614.
10. French HP, Gilsenan C, Cusack T. Gluteal muscle dysfunction and the role of specific strengthening in hip osteoarthritis: a review. Phys Ther Rev 2008;13(5):333–44, http://dx.doi.org/10.1179/174328808X356401.
11. Grimaldi A, Richardson C, Stanton W, Durbridge G, Donelly W. The association between degenerative hip joint pathology and size of the gluteus medius, gluteus minimus and piriformis muscles. Man Ther 2009b;14:605–10, http://dx.doi.org/10.1016/j.math.2009.07.004.
12. Maughan RJ, Nimmo MA. The influence of variations in muscle fibre composition on muscle strength and cross-sectional area in untrained males. J Physiol 1984;351:299–311, http://dx.doi.org/10.1113/jphysiol.1984.sp015246.
13. Johansen KL, Shubert T, Doyle J, Soher B, Sakkas GK, Kent-Braun JA. Muscle atrophy in patients receiving hemodialysis: effects on muscle strength, muscle quality, and physical function. Kidney Int 2003;63:291–7, http://dx.doi.org/10.1046/j.1523-1755.2003.00704.x.
14. Goutallier D, Pastel J-M, Gleyze P, Leguilloux P, Van Driessche S. Influence of cuff muscle fatty degeneration on anatomic and functional outcomes after simple suture of full-thickness tears. J Shoulder Elbow Surg 2003;12(6):550, http://dx.doi.org/10.1016/S1058-2746(03)00211-8.
15. Rasch A, Byström AH, Dalén N, Martinez-Carranza N, Berg HE. Persisting muscle atrophy two years after replacement of the hip. J Bone Joint Surg Br 2009;91(5):583–8, http://dx.doi.org/10.1302/0301-620X.91B5.21477.
16. Kellgren JH, Lawrence JS. Radiological assessment of osteoarthritis. Ann Rheum Dis 1957;16:494–502.
17. Dawson J, Fitzpatrick R, Carr A, Murray D. Questionnaire on the perceptions of patients about total hip replacements. J Bone Joint Surg Br 1996;78(2):185–90.
18. Martinelli N, Longo UG, Marinozzi A, Franceschetti E, Costa V, Denaro V. Cross-cultural adaptation and validation with reliability, validity, and responsiveness of the Italian version of the Oxford Hip Score in patients with hip osteoarthritis. Qual Life Res 2011;20(6):923–9.
19. Folsom AR, Jacobs Jr DR, Caspersen CJ, Gomez-Marin O, Knudsen J. Test-retest reliability of the Minnesota Leisure Time Physical Activity Questionnaire. J Chronic Dis 1986;39(7):505–11, http://dx.doi.org/10.1016/0021-9681(86)90195-5.
20. Bullock-Saxton JE, Wong WJ, Hogan N. The influence of age on weight-bearing joint reposition sense of the knee. Exp Brain Res 2001;136(3):400–6.
21. Grimaldi A, Richardson C, Durbridge G, Donelly W, Darnell R, Hides J. The association between degenerative hip joint pathology and size of the gluteus maximus. Man Ther 2009;14(6):611–7, http://dx.doi.org/10.1016/j.math.2008.11.002.
22. Goutallier D, Postel J-M, Bernageau J, Lavau L, Voisin M-C. Fatty muscle degeneration in cuff ruptures. Clin Orthop Relat Res 1994;304:78–83.
23. Engelken F, Wassilew GI, Kohlitz T, Brockhaus S, Hamm B, Perka C, *et al*. Assessment of fatty degeneration of the gluteal muscles in patients with THA using MRI: reliability and accuracy of the Goutallier and Quartile classification systems. J Arthroplasty 2014;29:149–53, http://dx.doi.org/10.1016/j.arth.2013.04.045.
24. Thorborg K, Petersen J, Magnusson SP, Holmich P. Clinical assessment of hip strength using a hand-held dynamometer is reliable. Scand J Med Sci Sports 2010;20:493–501, http://dx.doi.org/10.1111/j.1600-0838.2009.00958.x.
25. Rasch A, Dalén N, Berg HE. Test methods to detect hip and knee muscle weakness and gait disturbance in patients with hip osteoarthritis. Arch Phys Med Rehabil 2005;86(12):2371–6, http://dx.doi.org/10.1016/j.apmr.2005.05.019.
26. Moore KL, Dalley AF, Agur AMR. Clinically Oriented Anatomy. 7 edn. Baltimore, MD: Lippincott Williams & Wilkins; 20141134.
27. Cohen J. Statistical Power Analysis for the Behavioral Sciences. 2 edn. Hillsdale, New Jersey: Lawrence Erlbaum; 1988.

28. Portney L, Watkins M. Foundations of Clinical Research: Applications to Practice. 2 edn. London: Appleton & Lange; 2000.
29. Chi AS, Long SS, Zoga AC, Read PJ, Deely DM, Parker L, *et al*. Prevalence and pattern of gluteus medius and minimus tendon pathology and muscle atrophy in older individuals using MRI. Skeletal Radiol 2015;44(2):1727–33, http://dx.doi.org/10.1007/s00256-015-2220-7.
30. Arokoski MH, Arokoski JPA, Haara M, Kankaanpää M, Vesterinen M, Niemitukia LH, *et al*. Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. J Rheumatol 2002;29(10):2185–95.
31. van Baar ME, Dekker J, Lemmens JA, Oostendorp RA, Bijlsma JW. Pain and disability in patients with osteoarthritis of hip or knee: the relationship with articular, kinesiological and psychological characteristics. J Rheumatol 1998;25:125–33.
32. Beck M, Sledge JB, Gautier E, Dora CF, Ganz R. The anatomy and function of the gluteus minimus muscle. J Bone Joint Surg Br 2000;82:358–63.
33. Wilson J, Ferris E, Heckler A, Maitland L, Taylor C. A structure review of the role of gluteus maximus in rehabilitation. N Z J Physiother 2005;33:95–100.
34. Yamada T, Demura S-I. Relationships between ground reaction force parameters during a sit-to-stand movement and physical activity and falling risk of the elderly and a comparison of the movement characteristics between the young and the elderly. Arch Gerontol Geriatr 2009;48(1):73–7, http://dx.doi.org/10.1016/j.archger.2007.10.006.
35. Lyons K, Perry J, Gronley JK, Barnes L, Antonelli D. Timing and relative intensity of hip extensor and abductor muscle action during level and stair ambulation. Phys Ther 1983;63(10):1597–605.
36. Hurwitz DE, Hulet CH, Andriacchi TP, Rosenberg AG, Galante JO. Gait compensations in patients with osteoarthritis of the hip and their relationship to pain and passive hip motion. J Orthop Res 1997;15:629–35, http://dx.doi.org/10.1002/jor.1100150421.
37. Foucher KC, Schlink BR, Shakoor N, Wimmer MA. Sagittal plane hip motion reversals during walking are associated with disease severity and poorer function in subjects with hip osteoarthritis. J Biomech 2012;45(8):1360–5, http://dx.doi.org/10.1016/j.jbiomech.2012.03.008.
38. Masonis JL, Bourne RB. Surgical approach, abductor function, and total hip arthroplasty dislocation. Clin Orthop Relat Res 2002;405:46–53.
39. Fischer FJ, Houtz SJ. Evaluation of the function of the gluteus maximus muscle. An electromyographic study. Am J Phys Med 1968;47(4):182–91.
40. Delmonico MJ, Harris TB, Visser M, Park SW, Conroy MB, Velasquez-Mieyer P, *et al*. Longitudinal study of muscle strength, quality, and adipose tissue infiltration. Am J Clin Nutr 2009;90(6):1579–85.
41. Hides JA, Stokes MJ, Saide M, Jull GA, Cooper DH. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. SPINE 1994;19:165–77.
42. Andary MT, Hallgren RC, Greenman PE. Neurogenic atrophy of suboccipital muscles after a cervical injury. Am J Phys Med Rehabil 1998;77:545–9.
43. Krssak M, Petersen KF, Dresner A, DiPietro L, Vogel SM, Rothman DL, *et al*. Intramyocellular lipid concentrations are correlated with insulin sensitivity in humans: a 1H NMR spectroscopy study. Diabetologia 1999;42:113–6.
44. Manini TM, Clark BC, Nalls MA, Goodpaster BH, Ploutz-Snyder LL, Harris TB. Reduced physical activity increases intermuscular adipose tissue in healthy young adults. Am J Clin Nutr 2007;85:377–84.
45. Visser M, Kritchevsky SB, Goodpaster BH, Newman AB, Nevitt M, Stamm E, *et al*. Leg muscle mass and composition in relation to lower extremity performance in men and women aged 70 to 79: the health, aging and body composition study. J Am Geriatr Soc 2002;50:897–904.
46. Norman GR, Sloan JA, Wyrwich KW. Interpretation of changes in health-related quality of life: the remarkable universality of half a standard deviation. Med Care 2003;41(5):582–92.
47. Semciw AI, Green RA, Murley GS, Pizzari T. Gluteus minimus: an intramuscular EMG investigation of anterior and posterior segments during gait. Gait Posture 2014;39:822–6, http://dx.doi.org/10.1016/j.gaitpost.2013.11.008.
48. Semciw AI, Pizzari T, Murley GS, Green RA. Gluteus medius: an intramuscular EMG investigation of anterior, middle and posterior segments during gait. J Electromyogr Kinesiol 2013;23(4):858–64, http://dx.doi.org/10.1016/j.jelekin.2013.03.007.
49. Pfirrmann CWA, Notzli HP, Dora C, Hodler J, Zanetti M. Abductor tendons and muscles assessed at MR imaging after total hip arthroplasty in asymptomatic and symptomatic patients. Radiology 2005;235(3), http://dx.doi.org/10.1148/radiol.2353040403.
50. Bagge E, Bjelle A, Eden S, Svanborg A. Osteoarthritis in the elderly: clinical and radiological findings in 79 and 85 year olds. Ann Rheum Dis 1991;50:535–59.