Age-related Degeneration of Lumbar Muscle Morphology in Healthy Younger versus Older Men

Dallaway, A. D. F., Hattersley, J., Diokno, M., Tallis, J., Renshaw, D., Wilson, A., Wayte, S., Duncan, M. & Weedall, A.

DOI 10.1080/13685538.2021.1878130
ISSN 1368-5538
ESSN 1473-0790

Publisher: Taylor & Francis

The Aging Male

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/itam20

Age-related degeneration of lumbar muscle morphology in healthy younger versus older men

Alexander Dallaway, John Hattersley, Michael Diokno, Jason Tallis, Derek Renshaw, Adrian Wilson, Sarah Wayte, Andrew Weedall & Michael Duncan

To cite this article: Alexander Dallaway, John Hattersley, Michael Diokno, Jason Tallis, Derek Renshaw, Adrian Wilson, Sarah Wayte, Andrew Weedall & Michael Duncan (2020) Age-related degeneration of lumbar muscle morphology in healthy younger versus older men, The Aging Male, 23:5, 1583-1597, DOI: 10.1080/13685538.2021.1878130

To link to this article: https://doi.org/10.1080/13685538.2021.1878130

Published online: 10 Mar 2021.

THE AGING MALE
2020, VOL. 23, NO. 5, 1583–1597
https://doi.org/10.1080/13685538.2021.1878130

Taylor & Francis
Taylor & Francis Group

ORIGINAL ARTICLE

Age-related degeneration of lumbar muscle morphology in healthy younger versus older men

Alexander Dallaway[a,b], John Hattersley[a,b], Michael Diokno[c], Jason Tallis[a,d], Derek Renshaw[a], Adrian Wilson[b,e], Sarah Wayte[e], Andrew Weedall[e] and Michael Duncan[a,d]

[a]Centre for Sport, Exercise and Life Sciences, Faculty of Health and Life Sciences, Coventry University, Coventry, UK; [b]Coventry NIHR CRF Human Metabolism Research Unit, University Hospitals Coventry and Warwickshire, Coventry, UK; [c]Department of Radiology, University Hospitals Coventry and Warwickshire NHS Trust, Coventry, UK; [d]School of Life Sciences, Faculty of Health and Life Sciences, Coventry University, Coventry, UK; [e]Radiology Physics, Department of Clinical Physics and Bioengineering, University Hospitals Coventry and Warwickshire NHS Trust, Coventry, UK

ABSTRACT

Aim: The aim of this study was to evaluate age-related changes in lumbar paravertebral muscle (LPM) morphology in healthy younger and older adult men.

Methods: T2-weighted axial MRI of the lumbar spine were obtained for 12 healthy older (67.3 ± 6.0 years) and younger (24.7 ± 3.1 years) men. Normalised muscle volume (NMV) and muscle fat infiltrate (MFI) were determined bilaterally for the psoas (PS), quadratus lumborum (QL), erector spinae (ES) and multifidus (MF). MANOVA was used to compare NMV and MFI between age groups. Follow-up ANOVA compared NMV and MFI for each muscle between age groups, with physical activity (PA) as a covariate. Stepwise regression was used to explore the association between muscle morphology.

Results: NMV of the ES and QL were significantly lower in the older group (OG) ($p = 0.040$ and $p < 0.001$, respectively). MFI across all muscles was significantly greater in the OG ($p < 0.001$). PA did not moderate the relationship between aging and muscle degeneration. Non-dominant handgrip strength was associated with NMV ($p = 0.003$).

Conclusions: Age-related atrophy is muscle specific in the lumbar spine; changes in lumbar musculature is independent of PA, handgrip strength may reflect morphological changes in the postural muscles with age. This study supports establishing effective targeted exercise interventions in the lumbar musculature.

ARTICLE HISTORY
Received 29 September 2020
Revised 7 January 2021
Accepted 15 January 2021
Published online 10 March 2021

KEYWORDS
Paravertebral muscles; MRI; fat infiltration; atrophy; spinal sarcopenia; aging

1. Introduction

Sarcopenia is a major health concern [1–3] and socioeconomic burden, responsible for considerable healthcare expenditure in the United Kingdom [4] and United States [5]. With worldwide increases in the number of elderly, the challenges posed by sarcopenia are increasingly great at patient, societal and clinical levels [6,7].

Diagnostic criteria for sarcopenia typically include measurement of appendicular muscle mass [8–13], however, there is increasing evidence highlighting the value of measuring paravertebral muscle degeneration (atrophy and fat infiltration) [14–19]. Narici and Maffulli [20] suggest that the postural muscles may be more susceptible to the effects of age-related sarcopenia than the appendicular muscles. This suggestion is supported as the lumbar musculature is more susceptible to progressive fat infiltration with aging than the lower limbs [21]. Degeneration of the lumbar musculature has attracted interest in recent years, even stimulating ideas of spinal sarcopenia [22,23]. This focus is likely due to the importance of the paravertebral muscles in the maintenance of spinal health [18,24,25], postural support, falls prevention, and assisting with trunk movements during activities of daily living [15,26–30].

Studies have shown that lower back pain and pathology modifies the size and composition of the LPMs [31–36]. However, the extent of muscle atrophy and fat infiltration is confounded by physiological declines associated with normal aging [15,34,37–40]. Few studies have directly investigated the effects of healthy aging on muscle size and fat infiltration in the lumbar spine [39,41,42]. Furthermore, the range of approaches

CONTACT Michael Duncan michael.duncan@coventry.ac.uk Faculty of Health and Life Sciences, Centre for Sport, Exercise and Life Sciences, Coventry University, Alison Gingel Building, Priory Street, Coventry CV1 5FB, UK

used to evaluate age-related changes in LPM morphology makes comparing findings difficult. Disparate methodologies and their influence on age-related degeneration of the LPMs have been explored in detail elsewhere [43]. Lifestyle factors, such as PA, may also modify the relationship between aging and LPM degeneration [44,45]. In our view, PA level has however been generally overlooked as a potential covariate in the literature. Anatomical variations in lumbar sagittal curvature [29] may also influence age-related changes in LPM morphology and their moderating effect should be considered.

Attenuating atrophy and fat infiltration in the LPMs is important to maintain quality of life and offset adverse health outcomes in old age [19,46–50]. The need for further investigation in this area has been widely acknowledged [21,38,39,49,51], particularly in healthy volunteers as undetermined phenotypes are likely hidden in the demographics of general populations [16,52]. To the authors' knowledge, no study to date has included volumetric and fat infiltration measures for the PS, QL, ES and MF in relation to healthy aging. Others have provided cross-sectional areas [53,54], although single representative slices lack the functional relevance of volumetric muscle measures [55]. Given the different functions of the LPMs and their propensity for localised degeneration [51,56–58], atrophic and fat infiltration changes are of interest for each individual muscle surrounding the lumbar spine. Therefore, the main aim of this study was to investigate age-related changes in LPM morphology (i.e. muscle volume and fat infiltration) in healthy younger and older men. Secondary aims were to evaluate the age-response on different muscles in the lumbar spine, the moderating effect of PA and associations with exploratory factors.

2. Materials and methods

Reporting of this prospective observational case matched study is based on the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement [59]. Coventry University Ethics Committee approved the study (P70399) on 13th September 2018.

2.1. Participants

Participants were recruited from the community and from University staff and students in Coventry, UK between November 2018 and June 2019. Inclusion criteria were healthy males aged 18–30 years or above 60 years. Exclusion criteria were BMI outside of 18.5–29.9 $kg \cdot m^{-2}$, smokers, consumption of alcohol on a daily basis and an existing or past medical history of metabolic diseases, neuromuscular disorders or musculoskeletal impairments that may affect muscular strength. Pain-related disability was assessed using the Modified Oswestry Low Back Pain Disability Questionnaire (ODQ-m) [60].

Participants in the younger group (YG) ($n = 12$) were matched to participants in the OG ($n = 12$) based on PA category (IPAQ-SF) [61] and ethnicity. Informed written consent was obtained from all participants and suitability to undergo MRI was assessed through a safety questionnaire immediately prior to the imaging study. Participants' height and mass were measured as whole-body fat and lean mass using bioelectrical impedance analysis (Tanita MC-780MA S, Tanita, Tokyo, Japan). Handgrip strength was also assessed using a handgrip dynamometer (Takei 5401, Takei Scientific Instruments Co Ltd, Japan) and adhering to the Southampton protocol [62].

2.2. Muscle morphology

2.2.1. Imaging acquisition

Scans of the lumbosacral spine were performed in a 3T MR imaging scanner (Discovery MR750w, GE Medical Systems, Milwaukee, WI). Participants were positioned supine in the magnetic bore with a pillow placed under their legs resulting in slight flexion of the hips and knees. A flexible 16-element body-matrix coil (GEM Anterior Array, GE Healthcare, Waukesha, WI) was used in combination with an in-table GEM Posterior Array (GE Healthcare, Waukesha, WI) consisting of a 5×8 array to improve signal reception. Axial T2-weighted fast recovery fast spin-echo (FRFSE) images were acquired from the L2 inferior endplate to the L5 inferior endplate, using a slice thickness of 4 mm, no interslice gap, repetition time (TR) 6643 ms, echo time (TE) 107 ms, acquisition matrix 240×240, flip angle 150°, field of view (FOV) 240 mm, voxel size $0.938 \times 0.938 \times 4$ mm, 30 slices provided sufficient coverage, acquisition time 03:04 min. Images were stored as DICOM format for processing.

2.2.2. Image analysis

Image analysis was performed using ITK-SNAP (ITK-SNAP, version 3.8.0, www.itk-snap.org) [63]. Right and left sides of the psoas, quadratus lumborum, erector spinae and multifidus were manually segmented for each axial slice between the superior endplate of L3 to the superior endplate of L4 (Figure 1). The superior

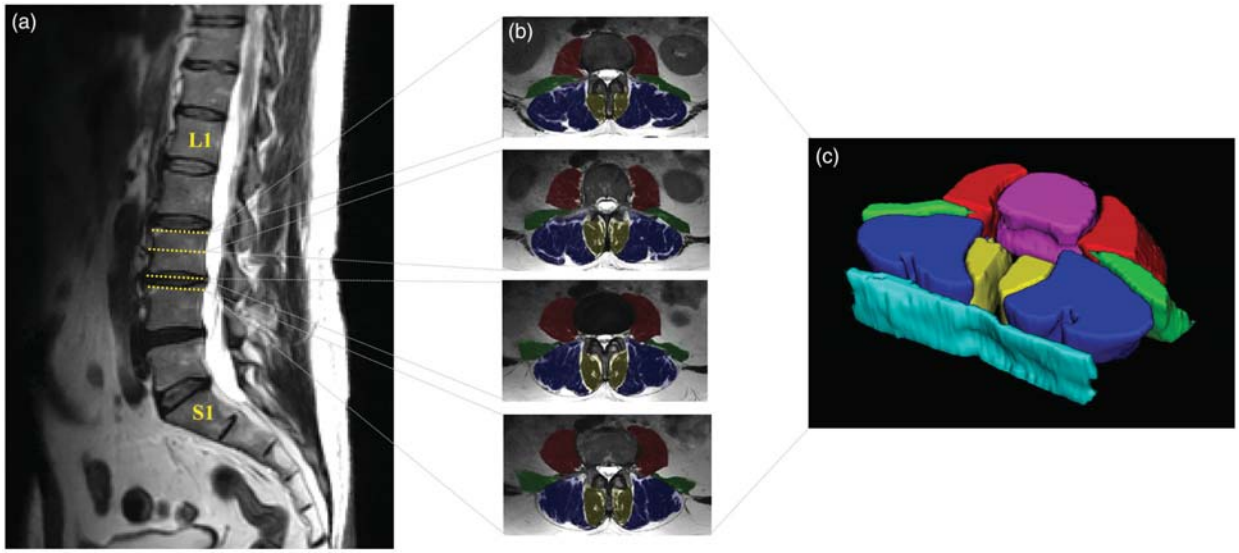


Figure 1. Sagittal (a) and selective axial MRI images (b) of the spine showing the ROI for the psoas, quadratus lumborum, erector spinae and multifidus muscles at the superior endplate L3, mid-vertebral slice L3, intervertebral disc L3/L4 and superior endplate L4. 3-D rendering of the axial image segmentations (c) is show with additional region of subcutaneous back fat and vertebral column for visualisation.

endplate of L4 was chosen as the inferior-most level to avoid obliquity at lower levels. The chosen levels provided identifiable anatomical planes that were approximately parallel to the axial image slices in all participants; minimising inter-subject measurement error.

The summation of axial regions of interest (ROI) provided volumetric measurements for each muscle. Volumetric measures are preferable to cross-sectional areas as they are more meaningful functionally [55,64,65] and minimise errors associated with postural variations during scanning [29]. Due to variations in participants anatomy, the number of analysed slices ranged from 10 to 11. To account for differences in muscle volume as a result of stature, muscle volumes were normalized to the straightline distance between the anterior superior border of the L1 vertebra and the anterior superior border of the S1 vertebra (Figure 2) [66], giving normalized muscle volume (NMV) in arbitrary units (a.u.).

Muscle fat infiltrate (MFI) was used to estimate intramuscular fat infiltration. For MFI, mean signal intensity (MSI) of each muscle across all included slices was reported as a percentage relative to MSI of a homogenous region of subcutaneous back fat across all included slices, given by the equation:

$$\text{MFI}\ (\%) = \text{Muscle MSI}/\text{Subcutaneous Fat MSI} * 100 \quad (1)$$

Similar approaches have been used previously [39,67–69] to account for inter-subject and temporal

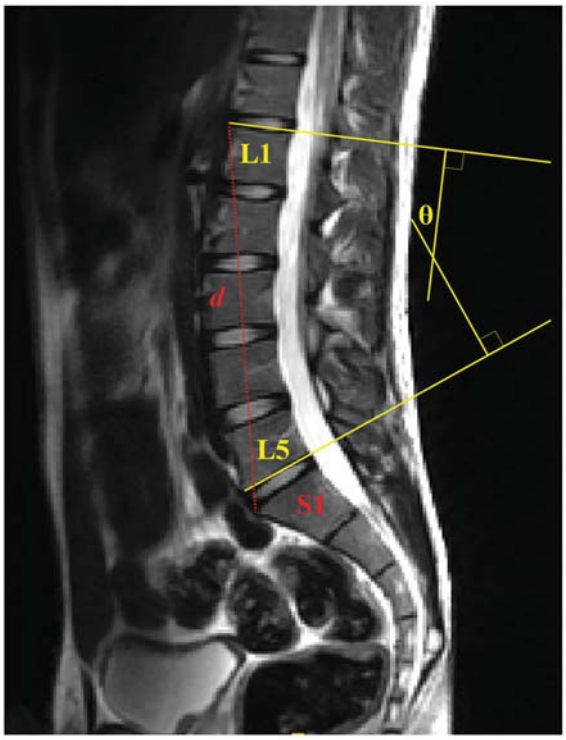


Figure 2. LLA measured using Cobb's method. T2-weighted mid-sagittal MRI image with lines drawn along the superior endplate of L1 and inferior endplate of L5, extending past the vertebral body. Orthogonal lines were added, on the side of convergence of the two lines, and the angle of the intersection (θ) was measured. The straight line distance (d) was measured between the anterior superior border of the L1 vertebra and the anterior superior border of the S1 vertebra.

variations in measured signal intensity due to field strength variations and distance of voxels from the detector coils. The fat ROI was selected from an area of subcutaneous back fat as defining a region of intermuscular fat may be difficult in every individual [67]. Hyperintense regions within the LPMs observed on T2 axial images were considered fatty tissue [46,70].

2.2.3. Vertebral height and lumbar lordotic angle

Sagittal plane images were acquired to measure lumbar lordotic angle (LLA). The images were acquired using a T2-weighted FRFSE sequence with a magnetic field strength of 3 T, slice thickness 6 mm, interslice gap 8 mm, TR 2877 ms, TE 109 ms, flip angle 142°, voxel size $1.41 \times 1.41 \times 8$ mm, images in acquisition 12, acquisition time 01:58 min. The slice representing the mid-vertebral line was identified by the presence of the conus medullaris and spinous processes and used for analysis. The Cobb L1–L5 method was used to measure LAA [71]; the angle between the superior endplate of L1 and the inferior endplate of L5 ($\theta_{L1\text{-}L5}$) (Figure 2).

2.2.4. Reliability

Segmentation of the LPMs was performed independently and sequentially by one observer (AD) for every participant. Segmentation was repeated (AD) on a random sub-sample ($n = 4$) after six months to assess long-term intra-observer reliability and measurement error. To avoid bias, the observer was blinded to the first measurement before the second measurement was completed. The observer was also blinded to the participant's information. Intra-observer reliability of the NMV and MFI measurements were assessed by calculating the average measures intra-class correlation coefficient (ICC) using a two-way mixed absolute agreement model.

2.3. Physical activity measurement

Participants wore an Actigraph GT9X accelerometer, sampling at 90 Hz, on their dominant wrist for 10 consecutive days. Accelerometers were worn on wrists to improve wear-time compliance and detect less traditional modes of PA commonly performed by older adults [72]. Data were processed using Actilife software (version 6.13). To be eligible for processing, data must have been obtained for a minimum of 4 days including one weekend day and at least 10 h of awake time during these days [73]. Valid data were divided into 1 s epochs to increase accurate identification of high-intensity bursts of activity. Average time (hours) spent per day in moderate-to-vigorous PA (MVPA) and vigorous PA (VPA) were calculated using cut-off values of 1031 counts/minute for moderate and 3589 counts/minute for vigorous intensities [74].

2.4. Statistical analysis

Statistical analyses were performed using SPSS software (Version 24.0, IBM, Armonk, NY) and graphical presentation performed using GraphPad Prism (Version 8.3.1, San Diego, CA). For each muscle group, right and left sides were combined and NMV and MFI were presented as mean ± SD unless otherwise stated. All variables were normally distributed (Shapiro Wilk's test: $p > 0.05$) and equal variances between groups were assumed (Levene's test: $p > 0.05$). Independent *t*-tests compared MVPA and VPA between groups. Following a significant result ($p < 0.05$), the moderating effect of the corresponding variable was explored by including it as a potential covariate. Multivariate analysis of variance (MANOVA) was conducted for NMV and MFI. Group differences were investigated based on the linear composite of outcome variables for each muscle. Significant between-subject results were followed up with univariate analysis of variance (ANOVA) to compare NMV and MFI for each muscle between age groups.

An independent *t*-test was used to compare LLA between groups. The strength of the relationships between LLA and muscle morphology outcomes were assessed from the Pearson correlation coefficient. Stepwise multiple linear regression was used to explore potential variables that may be related to total NMV (summation of each muscle's NMV) and mean MFI (mean MFI across all muscles). Input variables included: age (age group), MVPA (h/day), VPA (h/day), BMI, whole body fat composition (%), whole body lean mass (kg), dominant and non-dominant handgrip strength (kg), and LLA (°). A stepwise regression model was chosen due to its ability to reduce the number of predictor variables without substantially reducing the explanatory power of the data [75]. An alpha level of 0.05 was required for statistical significance in all tests. Standardized effect size (η_p^2) and observed power (1-β) were also determined where possible.

3. Results

Descriptive data are presented in Table 1. The OG had more whole-body fat mass ($t(22) = 2.62$, $p = 0.016$), engaged in less VPA ($t(22) = -2.37$, $p = 0.027$) and had weaker dominant ($t(22) = 2.22$, $p = 0.037$) and

Table 1. Demographic and descriptive information of the OG and YG.

	Young group ($n = 12$)	Old group ($n = 12$)
Demographics		
Age (years)***	24.7 ± 3.1	67.3 ± 6.0
Ethnicity (% white)	100	100
Anthropometrics and body composition		
Height (m)	1.78 ± 0.1	1.74 ± 0.1
Mass (kg)	76.4 ± 11.2	79.2 ± 10.8
BMI	24.1 ± 2.2	26.0 ± 2.7
Whole-body lean mass (kg)	59.7 ± 6.9	57.4 ± 6.3
Whole-body fat mass (kg)*	13.6 ± 4.9	19.2 ± 5.4
Physical limitation/disability		
ODQ-m (%)	2.2 ± 2.3	2.2 ± 3.5
Physical activity status		
IPAQ-SF (low : moderate : high)	0 : 4 : 8	0 : 4 : 8
MVPA (average hours per day)	6.6 ± 1.4	6.3 ± 1.5
VPA (average hours per day)*	2.6 ± 0.6	2.1 ± 0.6
Muscle function		
Dominant handgrip strength (kg)*	45.0 ± 7.5	37.4 ± 9.1
Non-dominant handgrip strength (kg)*	42.8 ± 5.3	36.3 ± 7.9

Data reported as mean ± SD.
n.b. IPAQ-SF was used in the matching procedure. ODQ-m: Modified Oswestry Low Back Pain Disability Questionnaire; MVPA: Moderate-to-vigorous physical activity; VPA: vigorous physical activity.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Table 2. Means ± SD for muscle volume normalized to vertebral height (NMV) and mean intramuscular fat infiltration (MFI).

	NMV (a.u.)		MFI (%)	
	Younger	Older	Younger	Older
Psoas	6.40 ± 0.85	6.09 ± 0.78	10.18 ± 1.77	13.04 ± 2.53**
Quadratus lumborum	3.03 ± 0.51	2.23 ± 0.50***	9.46 ± 1.56	14.87 ± 3.56***
Erector spinae	10.13 ± 1.00	8.94 ± 1.65*	13.48 ± 2.79	23.77 ± 5.56***
Multifidus	3.05 ± 0.50	3.38 ± 0.84	18.53 ± 4.74	33.48 ± 6.63***

NMV: normalized muscle volume; MFI: muscle fat infiltrate.
Significant difference with younger group *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

non-dominant ($t(22) = 2.38$, $p = 0.027$) handgrip strength than the YG.

MANOVA revealed statistically significant differences in NMV ($F(4,19) = 5.07$, $p = 0.006$; Wilks' $\Lambda = 0.48$, $\eta_p^2 = 0.52$) and MFI ($F(4,19) = 9.64$, $p < 0.001$; Wilks' $\Lambda = 0.33$, $\eta_p^2 = 0.67$) between age groups. Descriptive statistics for each muscle outcome stratified by age group are presented in Table 2.

3.1. Normalized muscle volume

Follow-up ANOVA revealed a significant effect of age on NMV for the QL ($F(1,22) = 15.98$, $p < 0.001$, $\eta_p^2 = 0.421$, $1\text{-}\beta = 0.968$) and ES ($F(1,22) = 4.77$, $p = 0.040$, $\eta_p^2 = 0.178$, $1\text{-}\beta = 0.551$) muscles (Figure 3). Compared to the YG, the OG had significantly lower NMV for the QL (2.2 ± 0.5 vs 3.0 ± 0.5) and ES (8.9 ± 1.7 vs 10.1 ± 1.0). Differences in NMV between groups were not significant for the PS and MF muscles. The greatest difference between groups was observed in the QL, where the YG exhibited a 36.47% greater NMV than the OG.

3.2. Intramuscular fat infiltration

There was a significant effect of age on fat infiltration for all muscle groups: PS ($F(1,22) = 10.30$, $p = 0.004$, $\eta_p^2 = 0.318$, $1\text{-}\beta = 0.864$); QL ($F(1,22) = 23.10$, $p < 0.001$, $\eta_p^2 = 0.512$, $1\text{-}\beta = 0.996$); ES ($F(1,22) = 32.73$, $p < 0.001$, $\eta_p^2 = 0.598$, $1\text{-}\beta = 1.0$); MF ($F(1,22) = 40.43$, $p < 0.001$, $\eta_p^2 = 0.648$, $1\text{-}\beta = 1.0$) (Figure 4). The greatest mean difference in MFI was observed in the MF, where MFI was significantly greater in the OG compared to the YG (33.48 ± 6.63 vs. 18.53 ± 4.74%, $p < 0.001$). The OG also exhibited significantly greater fat infiltration in the PS (13.04 ± 2.53 vs. 10.18 ± 1.77%, $p = 0.004$), QL (14.87 ± 3.56% vs 9.46 ± 1.56%, $p < 0.001$) and ES (23.77 ± 5.56% vs 13.48 ± 2.79%, $p < 0.001$).

3.3. Physical activity

VPA was not significantly related to NMV ($p = 0.44$) or MFI ($p = 0.94$), when included as a covariate in statistical models (MANCOVA). The effect of age on MFI for all muscles remained significant after controlling for VPA. However, whilst a significant main effect of age

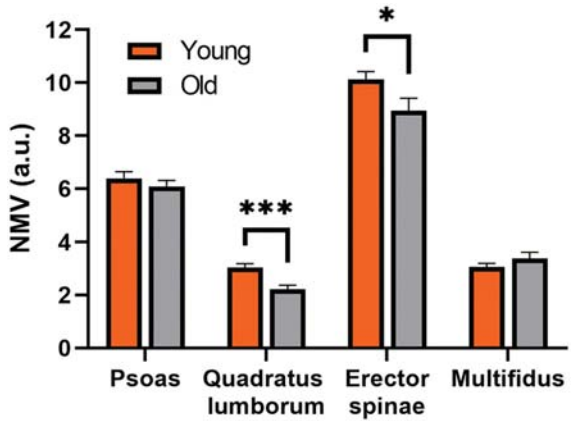


Figure 3. LPM volume (L3 superior endplate–L4 superior endplate) normalized to L1-L5 vertebral height (mean ± SEM). $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

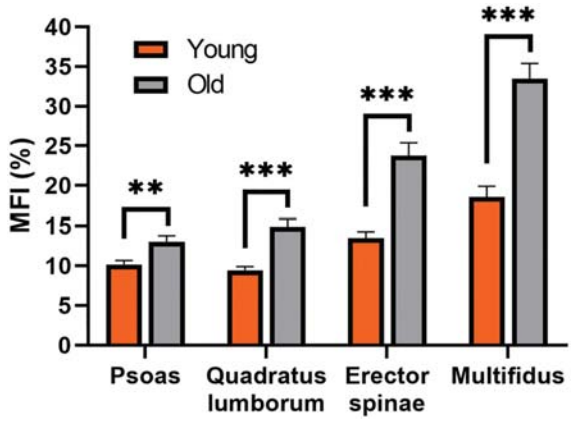


Figure 4. LPM mean fat infiltration (mean ± SEM). $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

on NMV for the QL remained after controlling for VPA, age did not have a significant effect on NMV for the ES after controlling for VPA ($p > 0.05$). Adjusted values indicated that ES NMV remained lower in the OG (9.0 ± 1.5 a.u.) compared to the YG (10.0 ± 1.5 a.u.).

3.4. Lumbar lordotic angle

The OG (36.9 ± 10.6°) had a significantly greater LLA than the YG (26.6 ± 5.2°), $t(22) = 3.02$, $p = 0.006$. Of the morphological features measured across all muscles, only MF NMV was significantly correlated with LLA, $r(22) = 0.434$, $p = 0.034$.

3.5. Exploration of important variables

Of the variables included in the stepwise linear regression, only non-dominant handgrip strength was found to be a significant predictor of total NMV, $F(1,22) = 10.93$, $p = 0.003$, $R^2 = 0.332$. Predicated total NMV was equal to 13.511 + 0.205*(non-dominant handgrip strength). Age was the only significant predictor of mean MFI, $F(1,22) = 38.27$, $p < 0.001$, $R^2 = 0.635$. Predicted mean MFI across all LPMs was equal to 12.922 + 8.370*(age group), where age group was coded as 0 = YG, 1 = OG. Mean MFI in the OG was 8.4% greater than the YG.

3.6. Reliability

Intra-observer reliability was excellent across measures of volume, NMV and MFI. ICC [95% CI], root mean square difference (RMSD) and mean residual difference (%) values are presented in Appendix A. Segmentation agreement maps for visualisation are presented in Appendix B.

4. Discussion

This study aimed to investigate morphological changes in the LPMs with healthy aging. The main finding of this study was that the OG exhibited increased fat infiltration across the lumbar musculature whilst muscle atrophy was only found in the QL and ES muscles. This suggests that morphological changes in muscle fat content, rather than size, are a better indicator of age-related degeneration in the lumbar musculature.

4.1. Age-related fat infiltration in the lumbar musculature

Significantly greater fat infiltration was observed across the LPMs with age, suggesting that myosteatosis has a somewhat global effect on skeletal muscle in the lumbar spine. Type I fibres are more susceptible to fat infiltration with advancing age [76–78], while type II fibres are more vulnerable to atrophic changes [76,79,80]. Since the LPMs are predominantly composed of slow-twitch fibres [81–86], it is unsurprising that changes in muscle composition were more apparent than those in muscle size. Indeed, age accounted for a large proportion (63.5%) of variation in mean MFI in the LPMs.

Despite methodological disparities, other studies have observed similar age effects on muscle composition in the lumbar spine [14,87,88]. However, the effects of aging on certain LPMs are more equivocal. Valentin et al. [39] and Lee et al. [89] reported that the PS is resistant to age-related fat infiltration. Discrepancies with this study may be due to different MRI sequences [39] and study subject characteristics

[89]. The rate of change in muscle fat content in the lumbar differs between Asian and Caucasian populations [90]. Therefore, differences with Lee et al. [89] may be due to ethnicity. The literature does not fully support the current findings for the QL either. While moderate to strong effects of aging on QL muscle density have been reported in large population-based cohorts [91,92] in support of the current results, others have observed insignificant age-related changes in QL fat content [54,69,93,94]. The highly disparate methods used to measure muscle composition precludes conclusions from being drawn, although the large effect sizes in the current study indicate that all LPMs undergo degenerative fat infiltration in older men.

4.2. Muscle-specific atrophy in the lumbar spine

The QL exhibited the greatest age-related atrophy in this study. This is supported by Johannesdottir et al. [91], who also reported that the rate of atrophy in the QL may be as great as 9% per decade in males. Few studies have directly investigated the effect of aging on QL muscle size [54,91,95], however, these studies all observed age-related atrophy. NMV of the ES was also significantly reduced in the OG, however age accounted for only 17% of the variance in ES atrophy, suggesting that factors other than age (e.g. physical inactivity) play an important role in mediating the loss of ES muscle size. Skeletal muscle has been shown to undergo adaptive reductive remodelling in response to both physical inactivity [96,97] and aging [49,98,99]. The effect of muscle disuse atrophy in the elderly is likely exacerbated by their interrelationship [100]. According to Ikezoe et al. [48], mechanical unloading preferentially affects the antigravity muscles. Given that the ES muscles are antigravity muscles and are predominantly composed of type I muscle fibres [81–85] which are susceptible to inactivity atrophy [101], less engagement with PA is a likely mechanism for ES muscle atrophy [102].

Another explanation as to why the ES and QL atrophy considers their fundamental anatomy and biomechanics. The QL and iliocostalis lumborum (IL) of the ES act in the coronal plane due to their lateral positioning [103,104]. Greater trunk excursions in the coronal plane contribute to larger lateral flexion moments [105]. As older adults exhibit reduced coronal plane trunk motion during everyday activities such as walking [106,107], it is likely that the subsequently lower mechanical demands are an insufficient stimulus to attenuate muscle atrophy in the QL and IL. The longissimus muscle of the ES acts primarily in the sagittal plane [108] and with advancing age this extensor muscle is solicited more due to postural changes increasing the thoracolumbar bending moment [109]. It is unknown from the current analysis how much ES atrophy was attributed to a diminished longissimus, however, these findings highlight the potential for a change in planar motion with aging.

Interestingly, MF NMV was larger in the OG. Change in LLA with age may explain why the MF is spared from atrophic decline. The NMV of the MF was significantly and moderately associated with LLA. According to mathematical models, forces applied by the LPMs should be greater in spines with increased lumbar lordosis as larger muscle forces are required to provide biomechanical stability [110]. Therefore, the significantly greater LLA in the OG may provide a training effect for the MF, whereby the MF plays a role in generating follower loads [111] (i.e. resultant forces that travel tangentially to the spine's sagittal curvature to provide lumbar spine stability) [112].

The PS did not appear to atrophy with aging in this study, in agreement others [41,48,88] although not unequivocally so [113]. The relative preservation of PS NMV may be due to changes in motor control strategies. Older adults exhibit a distal-to-proximal redistribution of joint power during gait [114,115], demonstrated by greater reliance on hip flexor activity to propel the leg into swing [114]. As the PS functions as a primary flexor of the hip joint [116–120], increased reliance on the hip flexor muscles in older age may provide sufficient stimulus to attenuate atrophy of the PS.

Fat infiltration appears to have a global effect on the LPMs whereas atrophy appears to be muscle-specific. Mechanisms for this remain undetermined, although the suggestions in the previous paragraph provide a plausible explanation. Increase in intramuscular fat tissue is likely due to slow-twitch fibre distribution in the postural muscles and propensity for these fibres to accumulate fatty deposits with aging. Muscle-specific atrophy likely concerns the specific functions of the lumbar muscles and their exposure to reduced mechanical loading resulting from a shift in the locus of function in motor performance with aging [115]. Furthermore, accretion of intramuscular fat may be an early change in muscle as it ages, which may explain why fat infiltration was the more apparent degenerative feature in the lumbar musculature.

4.3. Influence of physical activity on muscle degeneration

Controlling for VPA did not influence the effect aging had on MFI and on atrophy for the MF, PS and QL.

However, VPA moderated the effect aging had on ES muscle atrophy. This suggests that VPA may have a positive effect on attenuating ES muscle atrophy in older age, although this finding must be interpreted with caution as VPA was not significant as a covariate. Skeletal muscle tissue, like osseous tissue, is mechanoresponsive [121]. Yet PA seemingly has little or no effect on the size or fat content of the LPMs. In support of the current findings, it has been consistently demonstrated that PA does not relate to changes in paravertebral muscle morphology with aging [14,89,122]. This supports the idea that functional changes to the muscle in older age may be responsible for muscle degeneration rather than the mechanical loads exerted on them. The independence of PA on LPM morphology adds to the growing conflicting evidence questioning the effect of exercise interventions for spinal muscles, particularly those involving less volition like the shorter fascicles of MF.

4.4. Handgrip strength as a predictor of paravertebral muscle atrophy

Handgrip strength is well-established as an indicator of muscle status, particularly in elderly populations. In this study, variance in non-dominant handgrip strength explained 33.2% of the variance in the loss of total paravertebral NMV, which suggests that handgrip strength is a good indicator of muscle atrophy in the lumbar spine. However, mechanisms directly linking atrophy of the LPMs and forearm muscle strength are unlikely to exist. A more plausible explanation is that atrophy of the LPMs is simply representative of the systemic decline in muscle status throughout the body.

4.5. Limitations

The current study was limited by its cross-sectional design, although the wide age range and close matching of the groups mitigated this somewhat. Another limitation concerned the measure of fat infiltration. Fat infiltration derived from T2-weighted images [23,56,69,123,124], risks overestimating fatty muscle degeneration because water and other tissues in addition to fat appear hyperintense. The Dixon MRI technique has demonstrated its superiority over and T2-weighted imaging techniques in terms of fat fraction quantification [125]. However, confidence in the current approach is high given the similarity between the results in this study and other studies using Dixon sequences in comparable populations [14,41]. It should be noted that fat composition of the LPMs in the literature has a wide range of 2.1–45% in younger adults [39,126]. Furthermore, fat fraction quantification using a Dixon MRI sequence was not possible in all muscle groups from L2 to L5 due to the poor quality of the resulting images (Appendix C). Finally, the complex geometry of the LPMs may have introduced systematic errors in the image analysis. These muscles, the QL in particular, are not linear and axial slices (which were aligned parallel to the vertebral endplates) may not have produced true cross-sections of every muscle imaged.

5. Conclusion

There is a limited literature describing the natural history of spinal muscle change with age. This study adds to the literature by providing volumetric and fat infiltration data for all the main LPMs in relation to healthy aging. These findings will further understanding of age-related degeneration in the lumbar musculature and extend the concept of spinal sarcopenia. Furthermore, important relationships were revealed with exploratory variables (e.g. handgrip strength) that may enable early diagnosis of spinal sarcopenia.

In this study, aging had a detrimental effect on LPM morphology, although engaging in PA did not appear to attenuate muscle degeneration in old age. The current findings indicate that age-related fat infiltration has a global effect across the lumbar musculature, whereas atrophic changes appear to be muscle-specific. The MF was most susceptible to compositional changes with age, whilst the QL exhibited the greatest reductions in muscle volume. Future studies should look to assess age-related changes in LPM morphology using a longitudinal design over long time periods (>10 years). While it was implied that muscle function is impaired by the accumulation of fatty deposits, the loss of contractile tissue within the lumbar musculature should be investigated with respect to muscle function in older age.

Acknowledgements

The authors thank Alison Campbell, Poonam Patel and David Dixon of the Human Metabolism Research Unit, University Hospitals Coventry and Warwickshire NHS Trust for their assistance in collecting data.

Author contributions

Alexander Dallaway: Conceptualization, Methodology, Software, Formal analysis, Investigation, Data Curation,

Writing – Original Draft, Writing – Review & Editing, Visualization, Project administration; John Hattersley: Investigation, Methodology, Writing – Review & Editing, Supervision, Funding acquisition, Project administration; Michael Duncan: Writing – Review & Editing, Supervision, Funding acquisition, Project administration; Jason Tallis: Writing – Review & Editing; Derek Renshaw: Writing – Review & Editing, Funding acquisition; Michael Diokno: Methodology, Investigation, Software; Adrian Wilson: Methodology, Investigation, Writing – Review & Editing, Andrew Weedall: Software; Sarah Wayte: Methodology, Investigation.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This publication presents independent research funded by Coventry University/University Hospitals Coventry and Warwickshire NHS Trust and carried out with the support of the National Institute of Health Research (NIHR) Coventry and Warwickshire Clinical Research Facility. The views expressed are those of the author(s) and not necessarily those of Coventry University/University Hospitals Coventry and Warwickshire NHS Trust, the NIHR or the Department of Health.

ORCID

Alexander Dallaway http://orcid.org/0000-0002-2116-6654
John Hattersley http://orcid.org/0000-0002-1881-8797
Derek Renshaw http://orcid.org/0000-0002-2759-0229
Michael Duncan http://orcid.org/0000-0002-2016-6580

References

[1] Marcus RL, Addison O, Kidde JP, et al. Skeletal muscle fat infiltration: impact of age, inactivity, and exercise. J Nutr Health Aging. 2010;14(5):362–366.
[2] Landi F, Cruz-Jentoft AJ, Liperoti R, et al. Sarcopenia and mortality risk in frail older persons aged 80 years and older: results from iLSIRENTE study. Age Ageing. 2013;42(2):203–209.
[3] Beaudart C, Zaaria M, Pasleau F, et al. Health outcomes of sarcopenia: a systematic review and meta-analysis. PLoS One. 2017;12(1):e0169548.
[4] Pinedo-Villanueva R, Westbury LD, Syddall HE, et al. Health care costs associated with muscle weakness: a UK population-based estimate. Calcif Tissue Int. 2019;104(2):137–144.
[5] Janssen I, Shepard DS, Katzmarzyk PT, et al. The healthcare costs of sarcopenia in the United States. J Am Geriatr Soc. 2004;52(1):80–85.
[6] Dodds R, Sayer AA. Sarcopenia and frailty: new challenges for clinical practice. Clin Med J R Coll Phys. 2016;16:455–458.
[7] Beaudart C, Rizzoli R, Bruyère O, et al. Sarcopenia: burden and challenges for public health. Arch. Public Heal. 2014;72:45.
[8] Cruz-Jentoft AJ, Bahat G, Bauer J, Writing Group for the European Working Group on Sarcopenia in Older People 2 (EWGSOP2), and the Extended Group for EWGSOP2, et al. Sarcopenia: Revised European consensus on definition and diagnosis. Age Ageing. 2019;48(1):16–31.
[9] Chen LK, Liu LK, Woo J, et al. Sarcopenia in Asia: consensus report of the Asian working group for sarcopenia. J Am Med Dir Assoc. 2014;15(2):95–101.
[10] Fielding RA, Vellas B, Evans WJ, et al. Sarcopenia: an undiagnosed condition in older adults. current consensus definition: prevalence, etiology, and consequences. J Am Med Dir Assoc. 2011;12(4):249–256.
[11] Studenski SA, Peters KW, Alley DE, et al. The FNIH sarcopenia project: Rationale, study description, conference recommendations, and final estimates. J Gerontol – Ser A Biol Sci Med Sci. 2014;69(5):547–558.
[12] Morley JE, Abbatecola AM, Argiles JM, Society on Sarcopenia, Cachexia and Wasting Disorders Trialist Workshop, et al. Sarcopenia with limited mobility: an international consensus. J Am Med Dir Assoc. 2011;12(6):403–409.
[13] Muscaritoli M, Anker SD, Argilés J, et al. Consensus definition of sarcopenia, cachexia and pre-cachexia: Joint document elaborated by Special Interest Groups (SIG) "cachexia-anorexia in chronic wasting diseases" and "nutrition in geriatrics. Clin Nutr. 2010;29(2):154–159.
[14] Dahlqvist JR, Vissing CR, Hedermann G, et al. Fat replacement of paraspinal muscles with aging in healthy adults. Med. Sci. Sports Exerc. 2017;49(3):595–601.
[15] Hicks GE, Simonsick EM, Harris TB, et al. Cross-sectional associations between trunk muscle composition, back pain, and physical function in the health, aging and body composition study. J Gerontol Ser A Biol Sci Med Sci. 2005;60(7):882–887.
[16] Crawford RJ, Volken T, Mhuiris ÁN, et al. Geography of lumbar paravertebral muscle fatty infiltration. Spine (Phila. Pa. 1976). 2019;44(18):1294–1302.
[17] Shahtahmassebi B, Hebert JJ, Hecimovich MD, et al. Associations between trunk muscle morphology, strength and function in older adults. Sci Rep. 2017;7(1):10907.
[18] Sions JM, Coyle PC, Velasco TO, et al. Multifidi muscle characteristics and physical function among older adults with and without chronic low back pain. Arch Phys Med Rehabil. 2017;98(1):51–57.
[19] Fortin M, Gibbons LE, Videman T, et al. Do variations in paraspinal muscle morphology and composition predict low back pain in men? Scand J Med Sci Sports. 2015;25(6):880–887.
[20] Narici MV, Maffulli N. Sarcopenia: characteristics, mechanisms and functional significance. Br Med Bull. 2010;95(1):139–159.
[21] Dahlqvist J, Vissing C, Hedermann G, et al. Paraspinal fat infiltration in healthy adults with aging. Neuromuscul. Disord. 2015;25:S273.

[22] Debiane LG, Hage C, Asare-Sawiri M, et al. Utility of lumbar central sarcopenia in predicting early functional status following lung transplantation. Chest. 2015;148(4):1083A.
[23] Kim JC, Lee SU, Jung SH, et al. Natural aging course of paraspinal muscle and back extensor strength in community-dwelling older adults (sarcopenia of spine, SarcoSpine): a prospective cohort study protocol. BMJ Open. 2019;9(9):e032443.
[24] Eguchi Y, Suzuki MM, Yamanaka H, et al. Associations between sarcopenia and degenerative lumbar scoliosis in older women. Scoliosis. 2017; 12(1):9.
[25] Hicks GE, Simonsick EM, Harris TB, et al. Trunk muscle composition as a predictor of reduced functional capacity in the health, aging and body composition study: the moderating role of back pain. J Gerontol – Ser A Biol Sci Med Sci. 2005;60(11): 1420–1424.
[26] Suri P, Kiely DK, Leveille SG, et al. Trunk muscle attributes are associated with balance and mobility in older adults: a pilot study. Pm R. 2009;1(10): 916–924.
[27] Granacher U, Gollhofer A, Hortobágyi T, et al. The importance of trunk muscle strength for balance, functional performance, and fall prevention in seniors: A systematic review. Sports Med. 2013;43(7): 627–641.
[28] Barr KP, Griggs M, Cadby T. Lumbar stabilization: core concepts and current literature, part 1. Am J Phys Med Rehabil. 2005;84(6):473–480.
[29] Meakin JR, Fulford J, Seymour R, et al. The relationship between sagittal curvature and extensor muscle volume in the lumbar spine. J Anat. 2013;222(6): 608–614.
[30] Crisco JJ, Panjabi MM. The intersegmental and multisegmental muscles of the lumbar spine: a biomechanical model comparing lateral stabilizing potential. Spine (Phila. Pa. 1976). 1991;16(7):793–799.
[31] Beneck GJ, Kulig K. Multifidus atrophy is localized and bilateral in active persons with chronic unilateral low back pain. Arch Phys Med Rehabil. 2012;93(2): 300–306.
[32] Danneels LA, Vanderstraeten GG, Cambier DC, et al. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. Eur Spine J. 2000;9(4):266–272.
[33] Chen YY, Pao JL, Liaw CK, et al. Image changes of paraspinal muscles and clinical correlations in patients with unilateral lumbar spinal stenosis. Eur Spine J. 2014;23(5):999–1006.
[34] Kalichman L, Hodges P, Li L, et al. Changes in paraspinal muscles and their association with low back pain and spinal degeneration: CT study. Eur Spine J. 2010;19(7):1136–1144.
[35] Kalichman L, Klindukhov A, Li L, et al. Indices of paraspinal muscles degeneration: reliability and association with facet joint osteoarthritis: feasibility study. Clin Spine Surg. 2016;29(9):465–470.
[36] Teichtahl AJ, Urquhart DM, Wang Y, et al. Fat infiltration of paraspinal muscles is associated with low back pain, disability, and structural abnormalities in community-based adults. Spine J. 2015;15(7): 1593–1601.
[37] Le Cara EC, Marcus RL, Dempsey AR, et al. Morphology versus function: the relationship between lumbar multifidus intramuscular adipose tissue and muscle function among patients with low back pain. Arch Phys Med Rehabil. 2014;95(10): 1846–1852.
[38] Shahidi B, Parra CL, Berry DB, et al. Contribution of lumbar spine pathology and age to paraspinal muscle size and fatty infiltration. Spine (Phila. Pa. 1976). 2017;42(8):616–623.
[39] Valentin S, Licka T, Elliott J. Age and side-related morphometric MRI evaluation of trunk muscles in people without back pain. Man Ther. 2015;20(1): 90–95.
[40] Hebert JJ, Kjaer P, Fritz JM, et al. The relationship of lumbar multifidus muscle morphology to previous, current, and future low back pain: a 9-year population-based prospective cohort study. Spine (Phila Pa 1976)). 2014;39(17):1417–1425.
[41] Crawford RJ, Filli L, Elliott JM, et al. Age- and level-dependence of fatty infiltration in lumbar paravertebral muscles of healthy volunteers. AJNR Am J Neuroradiol. 2016;37(4):742–748.
[42] Fortin M, Videman T, Gibbons LE, et al. Paraspinal muscle morphology and composition: a 15-yr longitudinal magnetic resonance imaging study. Med Sci Sports Exerc. 2014;46(5):893–901.
[43] Dallaway A, Kite C, Griffen C, et al. Age-related degeneration of the lumbar paravertebral muscles: Systematic review and three-level meta-regression. Exp Gerontol. 2020;133:110856.
[44] Peltonen JE, Taimela S, Erkintalo M, et al. Back extensor and psoas muscle cross-sectional area, prior physical training, and trunk muscle strength – a longitudinal study in adolescent girls. Eur J Appl Physiol Occup Physiol. 1997;77(1-2):66–71.
[45] Gibbons LE, Videman T, Battié MC, et al. Determinants of paraspinal muscle cross-sectional area in male monozygotic twins. Phys Ther. 1998; 78(6):602–610.
[46] Kader DF, Wardlaw D, Smith FW. Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. Clin. Radiol. 2000;55(2): 145–149.
[47] Ekin EE, Yıldız HK, Mutlu H. Age and sex-based distribution of lumbar multifidus muscle atrophy and coexistence of disc hernia: an MRI study of 2028 patients. Diagn Interv Radiol. 2016;22(3):273–276.
[48] Ikezoe T, Nakamura M, Shima H, et al. Association between walking ability and trunk and lower-limb muscle atrophy in institutionalized elderly women: a longitudinal pilot study. J. Physiol. Anthropol. 2015; 34:1–6.
[49] Kalichman L, Carmeli E, Been E. The association between imaging parameters of the paraspinal muscles, spinal degeneration, and low back pain. Biomed Res Int. 2017;2017:2562957–2562914.
[50] Anderson DE, Quinn E, Parker E, et al. Associations of computed tomography-based trunk muscle size

and density with balance and falls in older adults. GERONA. 2016;71(6):811–816.
[51] Crawford RJ, Volken T, Valentin S, et al. Rate of lumbar paravertebral muscle fat infiltration versus spinal degeneration in asymptomatic populations: an age-aggregated cross-sectional simulation study. Scoliosis Spinal Disord. 2016;11:21–29.
[52] Määttä JH, Karppinen JI, Luk KDK, et al. Phenotype profiling of Modic changes of the lumbar spine and its association with other MRI phenotypes: a large-scale population-based study. Spine J. 2015;15(9): 1933–1942.
[53] Schweitzer L, Geisler C, Pourhassan M, et al. Estimation of skeletal muscle mass and visceral adipose tissue volume by a single magnetic resonance imaging slice in healthy elderly adults. J Nutr. 2016; 146(10):2143–2148.
[54] Sions JM, Elliott JM, Pohlig RT, et al. Trunk muscle characteristics of the multifidi, erector spinae, psoas, and quadratus lumborum in older adults with and without chronic low back pain. J Orthop Sports Phys Ther. 2017;47(3):173–179.
[55] Boom HPW, van Spronsen PH, van Ginkel FC, et al. A comparison of human jaw muscle cross-sectional area and volume in long- and short-face subjects, using MRI. Arch Oral Biol. 2008;53(3):273–281.
[56] Ploumis A, Michailidis N, Christodoulou P, et al. Ipsilateral atrophy of paraspinal and psoas muscle in unilateral back pain patients with monosegmental degenerative disc disease. BJR. 2011;84(1004): 709–713.
[57] Min JH, Choi HS, Ihl Rhee W, et al. Association between radiculopathy and lumbar multifidus atrophy in magnetic resonance imaging. BMR. 2013; 26(2):175–181.
[58] Baracos VE. Psoas as a sentinel muscle for sarcopenia: a flawed premise. J Cachexia Sarcopenia Muscle. 2017;8(4):527–528.
[59] von Elm E, Altman DG, Egger M, STROBE Initiative, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–1457.
[60] Fritz JM, Irrgang JJ. A comparison of a modified Oswestry Low Back Pain Disability Questionnaire and the Quebec Back Pain Disability Scale. Phys Ther. 2001;81(2):776–788.
[61] Craig CL, Marshall AL, Sjöström M, et al. International physical activity questionnaire: 12-Country reliability and validity. Med Sci Sports Exerc. 2003;35(8):1381–1395.
[62] Roberts HC, Denison HJ, Martin HJ, et al. A review of the measurement of grip strength in clinical and epidemiological studies: towards a standardised approach. Age Ageing. 2011;40(4):423–429.
[63] Yushkevich PA, Piven J, Hazlett HC, et al. User-guided 3D active contour segmentation of anatomical structures: significantly improved efficiency and reliability. Neuroimage. 2006;31(3):1116–1128.
[64] Akagi R, Takai Y, Ohta M, et al. Muscle volume compared to cross-sectional area is more appropriate for evaluating muscle strength in young and elderly individuals. Age Ageing. 2009;38(5):564–569.
[65] Blazevich AJ, Coleman DR, Horne S, et al. Anatomical predictors of maximum isometric and concentric knee extensor moment. Eur J Appl Physiol. 2009; 105(6):869–878.
[66] Deng X, Zhu Y, Wang S, et al. CT and MRI determination of intermuscular space within lumbar paraspinal muscles at different intervertebral disc levels. PLoS One. 2015;10(10):e0140315.
[67] D'Hooge R, Cagnie B, Crombez G, et al. Increased intramuscular fatty infiltration without differences in lumbar muscle cross-sectional area during remission of unilateral recurrent low back pain. Man Ther. 2012;17:584–588.
[68] Hyun S-JJ, Bae C-WW, Lee S-HH, et al. Fatty degeneration of paraspinal muscle in patients with the degenerative lumbar kyphosis: a new evaluation method of quantitative digital analysis using MRI and CT scan. Clin Spine Surg. 2016;29(10):441–447.
[69] Gibbons LE, Videman T, Battié MC, et al. Isokinetic and psychophysical lifting strength, static back muscle endurance, and magnetic resonance imaging of the paraspinal muscles as predictors of low back pain in men. Scand J Rehabil Med. 1997;29:187–191.
[70] Teichtahl AJ, Urquhart DM, Wang Y, et al. Physical inactivity is associated with narrower lumbar intervertebral discs, high fat content of paraspinal muscles and low back pain and disability. Arthritis Res Ther. 2015;17:114.
[71] Hong JY, Suh SW, Modi HN, et al. Reliability analysis for radiographic measures of lumbar lordosis in adult scoliosis: a case-control study comparing 6 methods. Eur Spine J. 2010;19(9):1551–1557.
[72] Walsh JME, Pressman AR, Cauley JA, et al. Predictors of physical activity in community-dwelling elderly white women. J Gen Intern Med. 2001;16(11): 721–727.
[73] Migueles JH, Cadenas-Sanchez C, Ekelund U, et al. Accelerometer data collection and processing criteria to assess physical activity and other outcomes: a systematic review and practical considerations. Sports Med. 2017;47(9):1821–1845.
[74] Diaz KM, Krupka DJ, Chang MJ, et al. Wrist-based cut-points for moderate- and vigorous-intensity physical activity for the Actical accelerometer in adults. J. Sports Sci. 2018;36(2):206–212.
[75] Huang C, Townshend JRG. A stepwise regression tree for nonlinear approximation: applications to estimating subpixel land cover. Int. J. Remote Sens. 2003;24(1):75–90.
[76] Gueugneau M, Coudy-Gandilhon C, Théron L, et al. Skeletal muscle lipid content and oxidative activity in relation to muscle fiber type in aging and metabolic syndrome. J Gerontol A Biol Sci Med Sci. 2015; 70(5):566–576.
[77] Choi SJ, Files DC, Zhang T, et al. Intramyocellular lipid and impaired myofiber contraction in normal weight and obese older adults. J Gerontol A Biol Sci Med Sci. 2016;71(4):557–564.
[78] St-Jean-Pelletier F, Pion CH, Leduc-Gaudet J-PP, et al. The impact of ageing, physical activity, and

pre-frailty on skeletal muscle phenotype, mitochondrial content, and intramyocellular lipids in men. J Cachexia Sarcopenia Muscle. 2017;8(2):213–228.
[79] Novotny SA, Warren GL, Hamrick MW. Aging and the muscle-bone relationship. Physiology (Bethesda). 2015;30(1):8–16.
[80] Lexell J, Taylor CC, Sjöström M. What is the cause of the ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year-old men. J Neurol Sci. 1988;84(2-3):275–294.
[81] Ng JKF, Richardson CA, Kippers V, et al. Relationship between muscle fiber composition and functional capacity of back muscles in healthy subjects and patients with back pain. J Orthop Sports Phys Ther. 1998;27(6):389–402.
[82] Mannion AF, Dumas GA, Cooper RG, et al. Muscle fibre size and type distribution in thoracic and lumbar regions of erector spinae in healthy subjects without low back pain: normal values and sex differences. J Anatomy. 1997;190(4):505–513.
[83] Parkkola R, Alanen A, Kalimo H, et al. MR relaxation times and fiber type predominance of the psoas and multifidus muscle: an autopsy study. Acta Radiol. 1993;34(1):16–19.
[84] Kimura T. Composition of psoas major muscle fibers compared among humans, orangutans, and monkeys. Z Morphol Anthropol. 2002;83:305–314.
[85] Sirca A, Kostevc V. The fibre type composition of thoracic and lumbar paravertebral muscles in man. J Anat. 1985;141:131–137.
[86] Rantanen J, Rissanen A, Kalimo H. Lumbar muscle fiber size and type distribution in normal subjects. Eur Spine J. 1994;3(6):331–335.
[87] Kita T, Takayama K, Kanematsu F, et al. Aging-associated changes in paraspinal muscles and psoas muscles. ESSR. 2013;2013:92–93.
[88] Hedermann G, Dahlqvist JR, Løkken N, et al. Progressive fat replacement of muscle contributes to the disease mechanism of patients with single, large-scale deletions of mitochondrial DNA. Neuromuscul Disord. 2018;28(5):408–413.
[89] Lee SH, Park SW, Kim YB, et al. The fatty degeneration of lumbar paraspinal muscles on computed tomography scan according to age and disc level. Spine J. 2017;17(1):81–87.
[90] Crawford RJ, Elliott JM, Volken T. Change in fatty infiltration of lumbar multifidus, erector spinae, and psoas muscles in asymptomatic adults of Asian or Caucasian ethnicities. Eur Spine J. 2017;26(12): 3059–3067.
[91] Johannesdottir F, Allaire B, Anderson DE, et al. Population-based study of age- and sex-related differences in muscle density and size in thoracic and lumbar spine: the Framingham study. Osteoporos Int. 2018;29(7):1569–1580.
[92] Lorbergs AL, Allaire BT, Yang L, et al. A longitudinal study of trunk muscle properties and severity of thoracic kyphosis in women and men: The Framingham study. J Gerontol Ser A Biol Sci Med Sci. 2019;74(3):420–427.
[93] Kim H, Lee CK, Yeom JS, et al. Asymmetry of the cross-sectional area of paravertebral and psoas muscle in patients with degenerative scoliosis. Eur Spine J. 2013;22:1332–1338.
[94] Zhang Y, Guo J, Duanmu Y, et al. Quantitative analysis of modified functional muscle-bone unit and back muscle density in patients with lumbar vertebral fracture in Chinese elderly men: a case-control study. Aging Clin Exp Res. 2019;31(5):637–644.
[95] Aboufazeli M, Akbari M, Jamshidi AA, et al. Comparison of selective local and global muscle thicknesses in females with and without chronic low back pain. Ortop Traumatol Rehabil. 2018;20(3): 197–204.
[96] Paddon-Jones D, Sheffield-Moore M, Cree MG, et al. Atrophy and impaired muscle protein synthesis during prolonged inactivity and stress. J Clin Endocrinol Metab. 2006;91(12):4836–4841.
[97] Fortney SM, Schneider VS, Greenleaf JE. The physiology of bed rest. Hoboken, NJ: John Wiley & Sons, Inc.; 2011.
[98] Rogers MA, Evans WJ. Changes in skeletal muscle with aging: effects of exercise training. Exerc Sport Sci Rev. 1993;21:65–102.
[99] Roubenoff R, Hughes VA. Sarcopenia: current concepts. J Gerontol A Biol Sci Med Sci. 2000;55(12): M716–724.
[100] Wall BT, Dirks ML, Van Loon LJC. Skeletal muscle atrophy during short-term disuse: Implications for age-related sarcopenia. Ageing Res Rev. 2013;12(4): 898–906.
[101] Wang Y, Pessin JE. Mechanisms for fiber-type specificity of skeletal muscle atrophy. Curr Opin Clin Nutr Metab Care. 2013;16(3):243–250.
[102] Kalimo H, Rantanen J, Viljanen T, et al. Lumbar muscles: structure and function. Ann Med. 1989; 21(5):353–359.
[103] Daggfeldt K, Huang QM, Thorstensson A. The visible human anatomy of the lumbar erector spinae. Spine (Phila. Pa. 1976). 2000;25:2719–2725.
[104] Phillips S, Mercer S, Bogduk N. Anatomy and biomechanics of quadratus lumborum. Proc Inst Mech Eng H. 2008;222(2):151–159.
[105] Hendershot BD, Wolf EJ. Three-dimensional joint reaction forces and moments at the low back during over-ground walking in persons with unilateral lower-extremity amputation. Clin Biomech (Bristol, Avon)). 2014;29(3):235–242.
[106] Van Emmerik REA, McDermott WJ, Haddad JM, et al. Age-related changes in upper body adaptation to walking speed in human locomotion. Gait Posture. 2005;22(3):233–239.
[107] Schmid S, Bruhin B, Ignasiak D, et al. Spinal kinematics during gait in healthy individuals across different age groups. Hum Mov Sci. 2017;54:73–81.
[108] Stevens VK, Parlevliet TG, Coorevits PL, et al. The effect of increasing resistance on trunk muscle activity during extension and flexion exercises on training devices. J Electromyogr Kinesiol. 2008;18(3): 434–445.
[109] Le Huec JC, Richards J, Tsoupras A, et al. The mechanism in junctional failure of thoraco-lumbar fusions.

Part I: Biomechanical analysis of mechanisms responsible of vertebral overstress and description of the cervical inclination angle (CIA). Eur Spine J. 2018; 27(Suppl 1):129–138.
[110] Meakin JR, Aspden RM. Modeling the effect of variation in sagittal curvature on the force required to produce a follower load in the lumbar spine. J Mech Med Biol. 2012;12(01):1250013.
[111] Patwardhan AG, Havey RM, Meade KP, et al. A follower load increases the load-carrying capacity of the lumbar spine in compression. Spine. 1999;24: 1003–1009.
[112] Aspden RM. The spine as an arch: A new mathematical model. Spine. 1989;14:266–274.
[113] Mäki T, Oura P, Paananen M, et al. Longitudinal analysis of paraspinal muscle cross-sectional area during early adulthood – a 10-year follow-up MRI study. Sci Rep. 2019;9:1–8.
[114] Cofré LE, Lythgo N, Morgan D, et al. Aging modifies joint power and work when gait speeds are matched. Gait Posture. 2011;33(3):484–489.
[115] DeVita P, Hortobagyi T. Age causes a redistribution of joint torques and powers during gait. J ApplPhysiol. 2000;88(5):1804–1811.
[116] Penning L. Psoas muscle and lumbar spine stability: a concept uniting existing controversies. Critical review and hypothesis. Eur Spine J. 2000;9(6):577–585.
[117] Juker D, McGill S, Kropf P. Quantitative intramuscular myoelectric activity of lumbar portions of psoas and the abdominal wall during cycling. J Appl Biomech. 1998;14(4):428–438.
[118] Bogduk N, Pearcy M, Hadfield G. Anatomy and biomechanics of psoas major. Clin Biomech (Bristol, Avon). 1992;7(2):109–119.
[119] Santaguida PL, McGill SM. The psoas major muscle: a three-dimensional geometric study. J Biomech. 1995;28:343–345.
[120] Yoshio M, Murakami G, Sato T, et al. The function of the psoas major muscle: passive kinetics and morphological studies using donated cadavers. J Orthop Sci. 2002;7(2):199–207.
[121] Trumbull A, Subramanian G, Yildirim-Ayan E. Mechanoresponsive musculoskeletal tissue differentiation of adipose-derived stem cells. Biomed. Eng. Online. 2016;15:43.
[122] Anderson DE, D'Agostino JM, Bruno AG, et al. Variations of CT-based trunk muscle attenuation by age, sex, and specific muscle. J Gerontol Ser A Biol Sci Med Sci. 2013;68(3):317–323.
[123] Heo J-Y, Park J-H, Lim S-A, et al. Feasibility of using fat degeneration of lumbar extensor muscle as an alternative diagnostic criterion for sarcopenia in patients with osteoporotic vertebral fractures. Asian Spine J. 2019. Available from: http://asianspinejournal.org/journal/view.php?doi=10.31616/asj.2019.0044.
[124] Fortin M, Yuan Y, Battié MC. Factors associated with paraspinal muscle asymmetry in size and composition in a general population sample of men. Phys Ther. 2013;93(11):1540–1550.
[125] Ma J, Singh SK, Kumar AJ, et al. T2-weighted spine imaging with a fast three-point Dixon technique: comparison with chemical shift selective fat suppression. J Magn Reson Imaging. 2004;20(6):1025–1029.
[126] Pezolato A, De Vasconcelos EE, Defino HLA, et al. Fat infiltration in the lumbar multifidus and erector spinae muscles in subjects with sway-back posture. Eur Spine J. 2012;21(11):2158–2164.

Appendix A.

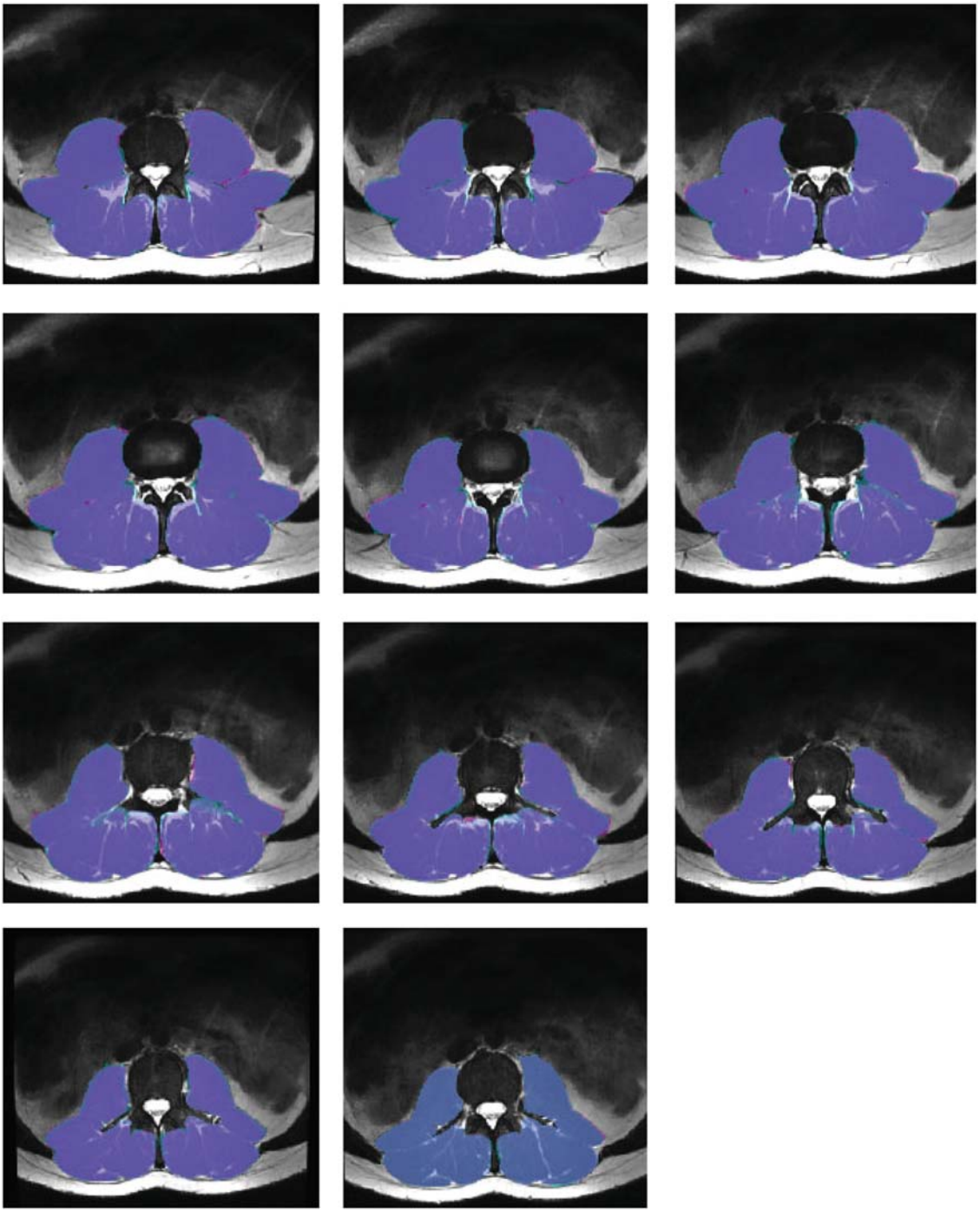
Intra-observer reliability for the MRI analysis outcome measures presented as intraclass correlation coefficient (ICC) with 95% confidence intervals [95% CI], root mean squared difference (RMSD) and mean residual difference (MRD) expressed as a percentage

	ICC [95% CI]		RMSD	MRD (%)
Volume (cm^3)				
PS	1.000	[0.996, 1.000]	0.45	0.3
QL	0.997	[0.966, 1.000]	1.49	2.6
ES	0.999	[0.986, 1.000]	1.58	1.0
MF	0.995	[0.943, 1.000]	0.91	1.5
NMV (a.u.)				
PS	0.998	[0.976, 1.000]	0.07	1.2
QL	0.994	[0.940, 1.000]	0.09	2.9
ES	0.997	[0.970, 1.000]	0.11	1.2
MF	0.995	[0.944, 1.000]	0.05	1.7
MFI (%)				
PS	0.918	[0.075, 0.995]	0.58	3.4
QL	0.990	[0.895, 0.999]	0.98	7.9
ES	0.998	[0.973, 1.000]	0.66	2.0
MF	0.999	[0.978, 1.000]	0.74	1.7
Vertebral height (cm)	0.982	[0.710, 0.999]	0.2	1.0
LLA (°)	0.989	[0.844, 0.999]	1.9	3.3

ES: erector spinae; MF: multifidus; MFI: muscle fat infiltrate; NMV: normalized muscle volume; LLA: lumbar lordotic angle; PS: psoas; QL: quadratus lumborum.

Appendix B.

Segmentation agreement maps showing the test (blue) and retest (pink) segmentations. Purple areas show the cross-over between test and retest measurements



Appendix C.

Comparison between (a) Dixon fat fraction image and (b) T2-weighted image. The fat fraction image shows noise increasing posteriorly-to-anteriorly