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Severe Lumbar Disability Is Associated With Decreased Psoas Cross-Sectional Area in Degenerative Spondylolisthesis

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Abstract

Study Design: Retrospective cohort.

Objectives: Alterations in lumbar paraspinal muscle cross-sectional area (CSA) may correlate with lumbar pathology. The purpose of this study was to compare paraspinal CSA in patients with degenerative spondylolisthesis and severe lumbar disability to those with mild or moderate lumbar disability, as determined by the Oswestry Disability Index (ODI).

Methods: We retrospectively reviewed the medical records of 101 patients undergoing lumbar fusion for degenerative spondylolisthesis. Patients were divided into ODI score $\leq$40 (mild/moderate disability, MMD) and ODI score >40 (severe disability, SD) groups. The total CSA of the psoas and paraspinal muscles were measured on preoperative magnetic resonance imaging (MRI).

Results: There were 37 patients in the SD group and 64 in the MMD group. Average age and body mass index were similar between groups. For the paraspinal muscles, we were unable to demonstrate any significant differences in total CSA between the groups. Psoas muscle CSA was significantly decreased in the SD group compared with the MMD group (1010.08 vs 1178.6 mm^2, $P = .041$). Multivariate analysis found that psoas CSA in the upper quartile was significantly protective against severe disability ($P = .013$).

Conclusions: We found that patients with severe lumbar disability had no significant differences in posterior lumbar paraspinal CSA when compared with those with mild/moderate disability. However, severely disabled patients had significantly decreased psoas CSA, and larger psoas CSA was strongly protective against severe disability, suggestive of a potential association with psoas atrophy and worsening severity of lumbar pathology.

Keywords
degenerative spondyloslisthesis, paraspinal muscle morphology, lumbar disability, psoas

Introduction

There has been increasing interest in examining the relationship of lumbar paraspinal muscle morphometry and outcomes after lumbar spinal surgery, as well as investigating any association between changes in paraspinal muscle density and low back pain (LBP). The multifidus, iliocostalis, and longissiumus muscles comprise the posterior paraspinal muscle complex, and provide dynamic stability to the lower lumbar spine. Recent literature examining the posterior lumbar paraspinal muscle cross-sectional surface area (CSA), as measured on magnetic resonance imaging (MRI) or computed tomography (CT), has found some association with decreased paraspinal muscle density and worsening lumbago.[1-3] Decreased CSA of the multifidus muscle has been shown to independently predict poor outcomes after surgical intervention for lumbar spinal

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stenosis,[3] while increasing fat infiltration of the erector spinae musculature may contribute to worsened disability and the development of modic changes in the lumbar spine.[4,5] A progressive decrease in paraspinal CSA and multifidus fat infiltration may occur as part of normal aging, but may also result from underlying structural pathology that can contribute to severe LBP.[1,6]

However, the relationship of the iliopsoas muscle to the posterior paraspinal muscles, and the effect of variation in psoas CSA to lumbar disability, is also of increasing interest. The anterior psoas muscle works in concert with the posterior structures to maintain posture and stabilize the spine.[2] While psoas CSA has been shown to decrease with age and female sex, and may not correlate with severity of structural alterations on MRI in patients with isthmic spondylolisthesis,[7] the association between changes in psoas CSA and the severity of lumbar disability in patients with degenerative spondylolisthesis has not been extensively explored. We hypothesized that psoas and paraspinal CSA in patients with disabling LBP, as determined by the Oswestry Disability Index (ODI), would be significantly decreased when compared to patients without significant lumbar disability. The purpose of this study, therefore, was to compare the absolute psoas and paraspinal CSA in patients with degenerative lumbar spondylolisthesis and corresponding severe disability to similar patients with only mild or moderate disability.

Methods

After institutional review board approval, a retrospective review of outpatient medical records was performed for patients with degenerative lumbar spondylolisthesis undergoing single level posterior lumbar fusion. Inclusion criteria were any patient greater than 18 years of age at our institution diagnosed with degenerative lumbar spondylolisthesis, based on the ICD-9 code 721.42, with a preoperative lumbar spine MRI and completed preoperative patient-reported outcome scores. Patients younger than 18 years, those lacking a preoperative lumbar spine MRI or completed preoperative patient-reported outcome scores, or those with a diagnosis other than degenerative lumbar spondylolisthesis were excluded from the study. Patients who had previously undergone lumbar spinal surgery of any type were also excluded. Patients were stratified based on their preoperative ODI, with ODI score $\leq$40 comprising the mild to moderate (MMD) disability group, and ODI score >40 comprising the severe disability (SD) group. Other health-related questionnaires, including the Short Form 12 Physical (SF-12 P), Short Form 12 Mental (SF-12 M), and back pain VAS scores, were also performed. Demographic data, including age, sex, history of coronary artery disease and/or congestive heart failure, BMI, diabetes mellitus, smoking status and American Society of Anesthesiology (ASA) class was also recorded.

T1-weighted axial MRI images were evaluated independently by 2 blinded reviewers. Using Sectra Liteview Picture Archiving and Communication System (PACS) imaging measurement software (SectraAB, Linköping, Sweden), the CSA of the posterior paraspinal musculature (multifidus, iliocostalis, and longissiumus), as well as the anterior psoas CSA, were measured. The measurements were taken through the same axial cut at the midpoint of the L3-4 vertebral disc space (Figure 1). The average of the 2 measurements from each reviewer was then recorded as the CSA for that muscle. A ratio of the psoas CSA to the total posterior paraspinal CSA, as well as a psoas-to-multifidus ratio, were calculated to assess for any relative changes between posterior and anterior muscle CSA.

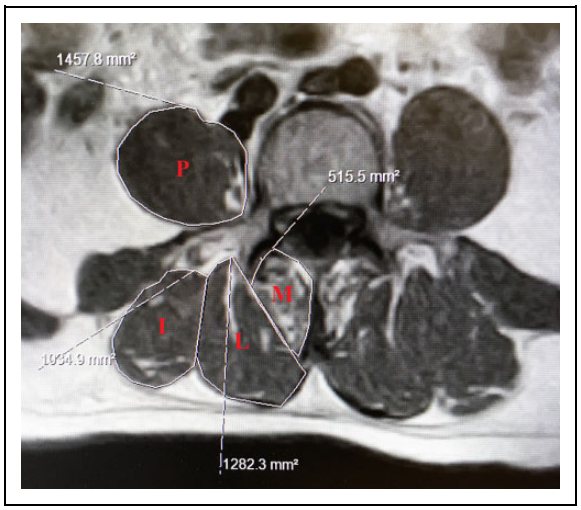

Figure 1. A representative magnetic resonance imaging (MRI) slice depicting the technique for measuring psoas and posterior paraspinal cross-sectional area. Measurements were performed at the same axial cut on T1 MRI, at the midpoint of the L3-4 vertebral disc space, by 2 blinded reviewers using standard digital imaging software. The average of both measurements was used as the final value for each patient. P, psoas; M, multifidus; L, Longissimus; I, Iliocostalis.

We used Microsoft Excel and Open Source Epidemiologic Statistics for Public Health (OpenEpi) Version 3.01 and JMP (SAS, Cary, NC, USA) for statistical tests. Student's *t* test was used to compare continuous variables between groups. Multivariable logistic regression was performed analyzing the effects of ASA (American society of Anesthesiologists) class, history of heart disease, smoking status, and psoas CSA on lumbar disability. $P \leq .05$ was considered statistically significant for all tests.

Results

A total of 101 patients met our inclusion criteria, with an average age of 60.0 years. Three patients had minimal disability (ODI <20), 34 patients had moderate disability (ODI 21-40), 48 had severe disability (ODI 41-60), and 16 had crippling and/or exaggerated pain (ODI >60). Using an ODI of 40 as a threshold to group patients as either mild/moderate or severe, there were 37 patients (36.6%) in the SD group and 64 (63.4%) in the MMD group. Average age was 59.1 and 61.6 years in the SD and MMD groups, respectively ($P = .377$). Average body mass index (BMI) was 30.6 and 31.5 kg/m^2, respectively ($P = .474$).

Table 1. Summary of Patient Demographics.

	Mild/Moderate Disability	Severe Disability	*P*
n (% male)	37 (48.6)	64 (45.3)	.746
Mean BMI (kg/m^2)	31.5	30.6	.474
Mean age, years	61.6	59.1	.377
Smokers (%)	16.2	34.4	.05*
ASA class	1.75	2.03	.005*
DM (%)	16.2	10.9	.445
CAD/CHF (%)	2.7	26.6	.003*
Mean VAS back pain	5.8	7.8	.001*
Mean SF-12 Physical	33.5	27.9	.002*
Mean SF-12 Mental	51.2	46.8	.066

Abbreviations: BMI, body mass index; ASA, American Society of Anesthesiologists; DM, diabetes mellitus; CAD, coronary artery disease; CHF, congestive heart failure; VAS, visual analogue scale.
*Indicates a statistically significant difference ($P \leq .05$).

There were no differences in the ratio of male and female patients between groups (45.3% male and 48.6% male in the SD and MMD groups, respectively, $P = .746$). A higher percentage of patients in the SD group reported active tobacco smoking (34.4% vs 16.2%, $P = .05$). Patients in the SD group had a significantly higher average ASA classification (2.03 vs 1.75, $P = .005$) and a significantly higher percentage had a history of heart disease (26.6% vs 2.7%, $P = .003$). The SD group patients also had higher average VAS back pain scores, as well as lower average SF-12P scores. This data is summarized in Table 1.

The average time between lumbar spine MRI and initial outpatient evaluation was 3.6 months. For the posterior paraspinal musculature, average iliocostalis CSA was 1093.5 mm^2 in the SD group versus 1192.2 mm^2 in the MMD group, which was not statistically significantly different ($P = .168$). The measured longissimus CSA was 685.8 versus 764.8 mm^2 in the SD and the MMD groups, respectively, which was also not significantly different ($P = .329$). The multifidus CSA was 499.1 versus 526.0 mm^2 for the SD and MMD groups, respectively, again not significantly different ($P = .125$). Psoas muscle CSA was significantly decreased in the SD group compared to the MMD group (1010.1 vs 1178.6 mm^2, $P = .041$). The overall posterior paraspinal muscle CSA was also not significant between groups, and while the ratio of psoas CSA to overall posterior paraspinal CSA was lower in the SD group, it did not reach statistical significance. When analyzing patients based on ODI subgroup (<20, 21-40, 41-60, >60), 1-way analysis of variance did not demonstrate any significant differences in average psoas CSA ($P > .05$). We also compared patients in the moderate (ODI = 21-40) and severe (ODI = 41-60) disability groups while excluding outliers in the extreme ODI groups. We found average psoas CSA remained significantly decreased in the severe ODI group when compared with the moderate disability group (1193.49 vs 992.71 mm^2, respectively, $P = .028$). Univariate contingency analysis of the distribution of psoas CSA demonstrated that the upper quartile (psoas CSA >1349.0 mm^2) was protective against severe disability (likelihood ratio = 0.272, $P = .006$).

Table 2. Muscle Cross-Sectional Area Measurement Results and Univariate Analysis.

	Mild/Moderate Disability	Severe Disability	*P*-value
Psoas CSA (mm^2)	1178.6	1010.1	.041*
Iliocostalis CSA (mm^2)	1192.2	1093.5	.168
Longissimus CSA (mm^2)	764.8	685.8	.329
Multifidus CSA (mm^2)	526.0	499.1	.125
Total posterior paraspinal CSA (mm^2)	2456.8	2278.4	.168
Psoas-posterior paraspinal CSA ratio	0.485	0.441	.087

Abbreviation: CSA, cross-sectional area.
*Indicates a statistically significant difference ($P \leq .05$).

Table 3. Multivariate Analysis Results.

Variable	Odds Ratio[a]	Range	*P*
ASA class	5.28	0.189-147.7	>.05
CAD/CHF	>200	0 to >1000	>.05
Psoas CSA (>75%ile)	0.13	0.03-0.66	.013*
Smoking status	12.9	1.19-140.2	.035*

Abbreviations: ASA, American Society of Anesthesiologists; CAD, coronary artery disease; CHF, congestive heart failure; CSA, cross-sectional area.
[a]Odds ratios are presented as the independent likelihood of having severe lumbar disability, based on Oswestry Disability Index (ODI) score >40.
*Indicates a statistically significant difference ($P \leq .05$).

A multivariable logistic regression model was then employed and found that active smoking was independently associated with severe disability (odds ratio = 12.9, range = 1.19-140.2, $P = .035$), while psoas size above the upper quartile threshold value was significantly protective against severe disability (odds ratio = 0.13, range = 0.03-0.66, $P = .013$). ASA class and history of heart disease were not associated with lumbar disability ($P > .05$). This data is summarized in Tables 2 and 3. A scatterplot of psoas CSA versus ODI score is represented in Figure 2.

Discussion

We found that in patients with degenerative lumbar spondylolisthesis and LBP leading to severe disability, as determined by preoperative ODI, the CSA of the iliopsoas muscle on MRI was significantly decreased by almost 200 mm^2 when compared with similar patients with mild or moderate disability. In addition, psoas CSA in the upper quartile range was strongly and independently protective against severe disability. While we did find that smoking status was also independently associated with severe lumbar disability, which corroborates the results of a previously published meta-analysis,[8] there were no significant differences in CSA in any of the posterior paraspinal musculature between groups. To our knowledge, this is the first

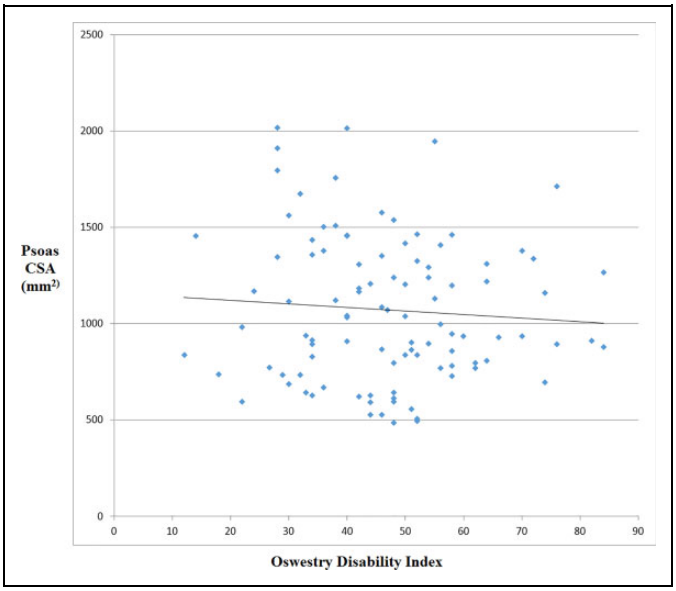

Figure 2. Graphical distribution of iliopsoas cross-sectional surface area (CSA) versus Oswestry Disability Index (ODI) score. A general trend in decreasing psoas CSA with increasing ODI is noted, but there is no strong linear correlation ($R^2 = 0.0058$).

study of its kind to describe changes in the psoas morphometry corresponding to the severity of lumbar disability in a select group of preoperative patients.

Recent literature has focused on changes in size and density of the posterior paraspinal muscles and the relationship between LBP, disability, and surgical outcomes.[3,4,9,10] In the present study, we were unable to show any significant differences in CSA of posterior paraspinal musculature between patients with severe or mild/moderate lumbar disability. In their study of LBP patients with spinal stenosis, Zotti et al[3] found that patients with multifidus CSA <8.5 cm^2 undergoing posterior lumbar decompression and had significantly worse health related outcomes scores at 1 and 2 years. Selective atrophy of the multifidus muscle has also been found in patients with isthmic spondylolisthesis.[7] However, our inability to show any significant association between posterior paraspinal CSA and lumbar disability corroborates the results of a recent systematic review, which concluded that currently published evidence is conflicting and no relationship has been definitively established.[11] In a retrospective chart analysis of 178 patients, Wan et al[10] reported significant ipsilateral atrophy of the paraspinal muscles in patients with acute and chronic LBP, including the psoas, but that significant associated fatty degeneration occurred synchronously with the loss of CSA. In support of their findings, there is increasing evidence that such fatty degeneration is related to paraspinal dysfunction and potentially to structural changes within the spinal column. A recent cross-sectional study of patients with severe LBP and ambulatory dysfunction found that lipomatous infiltration of the paraspinal musculature was significantly associated with type II Modic changes and chronic LBP.[4] Thus, it may be reasonable to posit that it is the quality of the paraspinal musculature, rather than the absolute or relative cross-sectional size of individual components alone, that is more relevant as a predictor of lumbar dysfunction.

Particular interest in changes in the CSA of the iliopsoas has also recently developed, as some previous studies have shown a relationship between LBP and CSA.[2,6,11-15] One study of patients with unilateral LBP found a significant decrease in ipsilateral multifidus and psoas CSA, which also positively correlated with the severity of self-reported pain and radiculopathic symptoms[2]; these findings were confirmed by a later study in unilateral LBP patients.[16] Arbanas et al[6] compared a cohort of patients with LBP to normal controls and found an increase in the relative psoas CSA in patients with chronic pain; however, they also found that the relative psoas CSA significantly decreased in patients with degenerative structural changes to the lumbar spine. The authors argue that these changes are likely related to decreased activation of the psoas secondary to chronic pain, and acknowledge that by normalizing psoas CSA to intervertebral disc CSA, rather than using an absolute psoas CSA, these differences may be explained.[6] Psoas size and mass have also been shown to predict poor surgical outcomes in patients undergoing spine surgery. Zakaria et al[13] showed that for both male and female patients undergoing lumbar surgery, a psoas CSA in the lowest tertile was independently predictive of having a postoperative complication, while decreases in the posterior paraspinals were not. Similarly, Bokshan et al[14] found that patients in the lowest third of measured psoas CSA were at a 3-fold increased risk of postoperative complications after thoracolumbar surgery and had a significantly lower cumulative survival. In fact, some studies have suggested that psoas CSA may function as a surrogate measurement for overall sarcopenia and health status,[14,17] though this assumption has not necessarily borne out in recent spine literature.[18] While these studies have found associations between decreased psoas CSA and poor surgical outcomes, which were not explored in the present study, to our knowledge this is the first study to show a relationship between psoas CSA and severity of lumbar symptoms and disability in patients meeting operative criteria for degenerative spondylolisthesis.

There is no clear consensus on the role of the iliopsoas muscle to the development of lumbar pain or degenerative disease.[11] The psoas muscle acts in concert with the posterior paraspinal muscles to dynamically maintain sagittal alignment and stabilize the lumbar spine, and various physical therapy regimens for lumbago typically focus on strengthening and stretching of the core stabilizers.[2] As noted previously, some authors have hypothesized that changes in CSA of the psoas occur secondary to development of structural degenerative disease, rather than as a cause of it.[6] Still others, however, have suggested that changes in recruitment of fibers from the primary lumbar stabilizers, because of lifestyle or other factors, can lead to compromised spinal stability and worsening pathology.[2,19] Additionally, age-related changes affecting the psoas and other paraspinals likely alter function, but the relationship between these changes and progression of disease is unclear. In the present study, we did not find any significant

differences in age between the cohorts, despite differences in psoas CSA, limiting the applicability of age as a potential confounding factor.

There are several limitations to this study. The retrospective design can increase the risk for observer and reporting bias. The distribution of parsapinal and iliopsoas CSA in normal, asymptomatic controls is not known or well-studied. Additionally, our study population is limited in size, which places our analysis at risk for type II error. The ODI value of 40 is somewhat arbitrary for use as a cutoff value, though we elected to do so because of the small number of patients in the minimal or crippling/exaggerated symptoms groups. As such, we were unable to show any significant differences in psoas CSA when analyzing by each ODI subgroup, which we believe is likely related to low numbers of patients in the extreme groups. We also lacked potentially valuable data, such as the duration of LBP prior to evaluation, and we did not examine postoperative outcomes. Lumbar spine MRIs were performed at separate locations, which may potentially affect image quality and introduce variability in our CSA measurements. However, the measurement process was conducted by two blinded reviewers on the same imaging software system to minimize the risk of this error. The strengths of the study include utilization of an established method of measuring the CSA of the paraspinal musculature, as well as generally similar demographic data between cohorts. Our study population represents patients with prior advanced imaging who were seeking surgical evaluation for lumbar spondylolisthesis, which likely makes our findings specifically applicable to this population. However, as a purely cross-sectional study, we are unable to make any conclusions with regard to the etiology of psoas atrophy in patients with severe disability, nor can we make any determinations with regard to causal relationships between lumbago and psoas degeneration.

Conclusions

In patients with severe lumbar disability and a diagnosis of degenerative spondylolisthesis, when compared to similar patients with mild or moderate disability, we found significantly decreased absolute CSA of the psoas muscle. In addition, multivariate analysis found that increased psoas CSA was significantly protective against severe lumbar disability. However, we were unable to show any association between posterior lumbar paraspinal CSA and lumbar disability. Future studies prospectively measuring lumbar paraspinal muscle CSA and fatty degeneration in patients with different lumbar diagnoses, or even in normal healthy controls, would be optimal. Additionally, any effects of paraspinal morphology on postoperative outcomes, especially with validated health related outcomes scores, may further elucidate any potential relationships between these measurements and symptoms.

Authors' Note

This study was performed at the Rothman Institute at Thomas Jefferson University, Philadelphia, PA. Two authors (SCW and PBM) are employees of the United States government. This work was prepared as part of their official duties and as such, there is no copyright to be transferred. The views expressed in this article are those of the authors and do not reflect the official policy of the Department of Navy, Department of Defense, or US Government.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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References

1. Aldebeyan S, Aoude A, Fortin M, et al. Predictors of discharge destination after lumbar spine fusion surgery. *Spine (Phila Pa 1976)*. 2016;41:1535-1541. doi:10.1097/BRS.0000000000001575.
2. Barker KL, Shamley DR, Jackson D. Changes in the cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. *Spine (Phila Pa 1976)*. 2004;29:E515-E519.
3. Zotti MGT, Boas FV, Clifton T, Piche M, Yoon WW, Freeman BJC. Does pre-operative magnetic resonance imaging of the lumbar multifidus muscle predict clinical outcomes following lumbar spinal decompression for symptomatic spinal stenosis? *Eur Spine J*. 2017;26:2589-2597. doi:10.1007/s00586-017-4986-x.
4. 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;15:1593-1601. doi:10.1016/j.spinee.2015.03.039.
5. 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;42:616-623. doi:10.1097/BRS.0000000000001848.
6. Arbanas J, Pavlovic I, Marijancic V, et al. MRI features of the psoas major muscle in patients with low back pain. *Eur Spine J*. 2013;22:1965-1971. doi:10.1007/s00586-013-2749-x.
7. Thakar S, Sivaraju L, Aryan S, Mohan D, Sai Kiran NA, Hegde AS. Lumbar paraspinal muscle morphometry and its correlations with demographic and radiological factors in adult isthmic spondylolisthesis: a retrospective review of 120 surgically managed cases. *J Neurosurg Spine*. 2016;24:679-685. doi:10.3171/2015.9.SPINE15705.
8. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between smoking and low back pain: a meta-analysis. *Am J Med*. 2010;123:87,e87-e35. doi:10.1016/j.amjmed.2009.05.028.
9. Fortin M, Gibbons LE, Videman T, Battie MC. Do variations in paraspinal muscle morphology and composition predict low back pain in men? *Scand J Med Sci Sports*. 2015;25:880-887. doi:10.1111/sms.12301.
10. Wan Q, Lin C, Li X, Zeng W, Ma C. MRI assessment of paraspinal muscles in patients with acute and chronic unilateral low back pain. *Br J Radiol*. 2015;88:20140546. doi:10.1259/bjr.20140546.

11. Ranger TA, Cicuttini FM, Jensen TS, et al. Is the size and composition of the paraspinal muscles associated with low back pain? A systematic review. *Spine J*. 2017;17:1729-1748. doi:10.1016/j.spinee.2017.07.002.
12. Belavy DL, Bansmann PM, Bohme G, et al. Changes in intervertebral disc morphology persist 5 mo after 21-day bed rest. *J Appl Physiol (1985)*. 2011;111:1304-1314. doi:10.1152/japplphysiol.00695.2011.
13. Zakaria HM, Schultz L, Mossa-Basha F, Griffith B, Chang V. Morphometrics as a predictor of perioperative morbidity after lumbar spine surgery. *Neurosurg Focus*. 2015;39:E5. doi:10.3171/2015.7.FOCUS15257.
14. Bokshan SL, Han AL, DePasse JM, et al. Effect of sarcopenia on postoperative morbidity and mortality after thoracolumbar spine surgery. *Orthopedics*. 2016;39:e1159-e1164. doi:10.3928/01477447-20160811-02.
15. Belavy DL, Armbrecht G, Richardson CA, Felsenberg D, Hides JA. Muscle atrophy and changes in spinal morphology: is the lumbar spine vulnerable after prolonged bed-rest? *Spine (Phila Pa 1976)*. 2011;36:137-145. doi:10.1097/BRS.0b013e3181cc93e8.
16. Ploumis A, Michailidis N, Christodoulou P, Kalaitzoglou I, Gouvas G, Beris A. Ipsilateral atrophy of paraspinal and psoas muscle in unilateral back pain patients with monosegmental degenerative disc disease. *Br J Radiol*. 2011;84:709-713. doi:10.1259/bjr/58136533.
17. Englesbe MJ, Patel SP, He K, et al. Sarcopenia and mortality after liver transplantation. *J Am Coll Surg*. 2010;211:271-278. doi:10.1016/j.jamcollsurg.2010.03.039.
18. Charest-Morin R, Street J, Zhang H, et al. Frailty and sarcopenia do not predict adverse events in an elderly population undergoing non-complex primary elective surgery for degenerative conditions of the lumbar spine. *Spine J*. 2018;18:245-254. doi:10.1016/j.spinee.2017.07.003.
19. Danneels LA, Vanderstraeten GG, Cambier DC, Witvrouw EE, De Cuyper HJ, Danneels L. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. *Eur Spine J*. 2000;9:266-272. doi:10.1007/s005860000190.