Beneficial intervertebral disc and muscle adaptations in high-volume road cyclists

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ABSTRACT

PURPOSE: Cycling is widely practiced as a mode of transportation, a leisurely pursuit and a competitive sport. Approximately half of cyclists experience low back pain. Yet, there has been limited study of spine tissue adaptations due to cycling.

METHODS: To investigate potential risk factors for spinal pain, we compared 18 high-volume cyclists (>150 km per week for ≥5 years) to 18 height-matched non-sporting referents. Participants had no history of spinal pathology. Magnetic resonance imaging was used to quantify intervertebral disc (IVD) morphology and hydration; and psoas, erector spinae, quadratus lumborum and multifidus muscle size and fat content. Endurance of trunk muscles (flexors and extensors) were measured and physical activity levels assessed objectively using accelerometry.

RESULTS: Cyclists IVD showed prolonged T2-time (+10.0(17.3)%; $p=0.021$), implying better IVD hydration and glycosaminoglycan content, compared to referents. Lower thoracic and upper lumbar IVD T2 time were longer in cyclists ($p\leq0.029$) but not at the lower lumbar spine. T2-time differences were larger in the nucleus pulposus compared to the annulus fibrosus. Cyclists showed larger psoas muscles with less fat content compared to referents. Cyclists also exhibited longer isometric trunk endurance times ($p\leq0.036$) and higher physical activity levels (osteogenic index, $p=0.038$).

CONCLUSION: Despite previous studies reporting higher than average prevalence of back pain in cyclists, the high-volume road cyclists in our cohort showed no anatomical or functional deficiency in spinal structures. In contrast, we found evidence for beneficial adaptations to the intervertebral discs and psoas muscles in high-volume cyclists compared to referents. These data support the notion that cycling is not detrimental to the spine; rather, in contrast, may be associated with beneficial changes at the spine.

Keywords: exercise; back pain; musculoskeletal; cycling

INTRODUCTION

Cycling is globally practiced as a mode of transportation, a leisurely pursuit, for cross-training and as competitive sport. In Australia (1), approximately 17% of the population participate in cycling while 46% of the United States population is cycling at least 25 days per year (2). Notwithstanding traffic accidents (3) and exposure to pollution (4), cycling is perceived as a safe and positive intervention. Rehabilitation protocols introduce cycling as exercise as part of the rehabilitation program and also stationary bicycle training for general fitness. However, data available in the literature portray an overall negative spinal impact of cycling. The incidence and prevalence of spinal pain is reported to be higher in road cyclists, with studies reporting that approximately half of cyclists had low back pain (5, 6). Despite this, there is limited data on the point prevalence of back pain in cyclists, with a narrative review of the literature estimating this to be 10-60% in cyclists (7). This compares to similar estimates of 1-60% in the wider community (8). Cycling has been incriminated as a causative factor for back pain in triathletes (9). Some investigators pointed to the sustained flexed posture at the lumbar spine during road cycling as the reason for the adverse spinal effects on cycling (5, 10). Other investigators have commented that fatigue of the trunk extensors, shifting of load onto passive spinal structures with viscoelastic creep during sustained trunk flexion, nutrient flow restriction to the intervertebral discs and/or overactivity of trunk extensors may damage the cyclist's spine (11). Whilst there is evidence (12) for smaller 'core' muscles in cyclists with back pain, data is otherwise limited on strength (13) and flexibility (14) imbalances in cyclists. The objective of the current study was to comprehensively investigate the lumbar spinal tissues of high-volume road cyclists (>150 km per week for minimum five years). Based on the literature, we tested the hypotheses that high-volume road cyclists would show (a) subclinical signs of intervertebral disc degeneration (reduced height and hydration) on magnetic resonance imaging

(MRI) (15), (b) smaller core muscles (psoas, erector spinae, quadratus lumborum and multifidus) on MRI, (c) increased core muscle fat content on MRI and 4) trunk extensor muscle fatigue or imbalance with trunk flexor muscles measured by isometric endurance.

METHODS

Ethical approval and subjects

The study was approved by the Deakin University Faculty of Health human ethics advisory group. This study was conducted as a pre-planned sub-analysis of a wider project (16–19) examining the impact of physical activity on the spine. All subjects gave their informed written consent prior to participation in the study. To avoid the impact of normal ageing on the spine tissues, only individuals aged 25 to 35 years of age were included. Exclusion criteria included current spinal pain, history of traumatic injury to the spine, history of spinal surgery, known scoliosis for which prior medical consultation was sought, current or prior smoker, known claustrophobia, and possible pregnancy. We included cyclists who reported a minimum of 150 km cycled per week over the last five years, with participation in other sports or exercises limited to once per week. Included in the non-sporting referent group were individuals who reported no regular sport or exercise in the last five years, currently engaged in less than 150 minutes of moderate activity per week defined as a "causing an individual to breathe harder than normal" (20) and walked less than 15 minutes to or from their place of work. Due to the influence of body height on IVD height, the referents were matched to the cyclists within two cm of body height. Thirty-six participants were included in the study (Table 1).

Testing and scanning protocol

Subjects were instructed to avoid exercise on the day of their MR scan. Due to diurnal variation in IVD water content (21), all imaging was performed after midday. Upon arriving at the MR scan facility, participants sat for a minimum of 20 minutes prior to entering the scanner. During this time participants completed questionnaires detailing their gender, current physical activity levels and body height. The cyclists also reported average distance ridden per week, hours ridden per week and number of years of participation.

To quantify IVD morphology and T2-time, a spin-echo multi-echo sequences on a 3T Phillips Ingenia scanner (Amsterdam, Netherlands) was used with spinal coils to collect images at eight echo times (15.75, 36.75, 57.75, 78.75, 99.75, 120.75, 141.75 and 162.75 ms) from 13 sagittal anatomical slices each (thickness 3 mm, interslice distance: 1.5 mm, repetition time: 2000 ms, field of view: 281 x 281 mm, image resolution: 0.366 mm per pixel) encompassing the lower spine from T11 to the sacrum. For radiological categorisation of IVD degeneration (Pfirrmann grade (22)), a sagittal plane T2-weighted sequence (15 slices, slice thickness: 3 mm, interslice distance: 1.5 mm, repetition time: 2600 ms, echo time: 70 ms, field of view: 357 mm x 357 mm, resolution: 0.532 mm per pixel) was acquired. To quantify muscle morphology and fat content, a paraxial T1-weighted scan (repetition time: 800 ms, echo time: 9 ms, slice thickness: 4 mm, interslice distance: 2 mm, field of view: 260 x 260 mm, image resolution: 0.270 mm per pixel) was performed with five groups of three slices each positioned at each vertebral body L1 to L5 and oriented parallel to the superior vertebral end-plates. Data were exported to a laptop for offline processing.

After MR scanning, body mass was measured using an electric bathroom scale with two digits precision. Participants then completed an isometric trunk flexor and extensor endurance test

following a previous published protocol (23), but without enforcing a five minute maximum. Each test was performed until failure and this time was measured (in seconds) on a stop watch. The same operator performed all testing and gave feedback every 30 seconds during the tests on correct body posture throughout. Afterwards, the subjects were given a hip-mounted ActiGraph model GT3X+ (Pensacola, FL), to measure habitual physical activity and were instructed to wear the ActiGraph continuously for eight days while awake except for water-based activities (e.g. swimming and bathing). Acceleration data were collected at 100 Hz with a ± six g range, filter set to 'normal' and 12 bit analog to digital conversion.

Offline image processing and analysis

To ensure blinding of the examiner, each subject was assigned a random numeric code (obtained from www.random.org). A radiologist determined the IVD Pfirrmann grade on sagittal T2-weighted images and this was averaged for all lumbar levels (Table 1). Three individuals had a supernumerary lumbar vertebral segment and the additional IVDs (designated L6/S1) were not included in the analyses.

ImageJ 1.38x (http://rsb.info.nih.gov/ij/) was used to perform all quantitative MR measures. In the sagittal spin-echo multi-echo images every IVD from T11/T12 to L5/S1 was measured. After segmenting the IVD, a custom written ImageJ plugin ("ROI Analyzer"; https://github.com/tjrantal/RoiAnalyzer and https://sites.google.com/site/daniellbelavy/home/roianalyser) was used to rotate the IVDs to horizontal and to measure their area, height and width. The IVD volume was calculated by linear interpolation of the area data from all slices. The slice number best centred according to the spinous process of each vertebrae was noted. With the exception of IVD volume (Table 1),

the morphometric data from three images at each lumbar IVD were averaged. Signal intensity was obtained of the entire IVD as well as five equidistant subregions of the IVD from anterior to posterior. T2-time was calculated via a linear fit to the natural logarithm of the image intensity in each of the eight MR echo times. Similar data were acquired for the five disc subregions and interpolated across the width of the IVD to generate 3D plots of T2-time distribution.

In each of the paraxial T1-weighted images, area of the lumbar multifidus, erector spinae, psoas and quadratus lumborum were measured bilaterally from L1 to L5 as in prior work (24). In every muscle, the signal intensity was measured. Similar to a method developed for T1 weighted muscle imaging in the cervical spine (25), the signal intensity inside the muscle was divided by an internal body fat reference to obtain an intramuscular fat proportion. We used two internal body fat references: subcutaneous fat and visceral fat anterolateral to the psoas muscle (Figure 1). The fat percentage (100%*muscle signal intensity/fat reference signal intensity) was calculated. The muscle morphometric data as well as the two estimates of muscle fat content were averaged from left and right sides at each level and also averaged between all lumbar levels.

Accelerometry analysis

Accelerometer data files were downloaded using ActiLife software (version 6.13.1). Raw data files were converted to 15-second epoch files by ActiLife software. These files contained vertical axis count data that were processed using a customised Excel macro. Non-wear time was defined as sustained 60-minute periods of consecutive zeroes (26) and established cut-off points were used to calculate sedentary time (26), light physical activity time and moderate-to-

vigorous physical activity time (sum of moderate and vigorous-intensity physical activity) (27). The total time spent in these intensities were obtained for each valid day (defined as ≥10 hours/day) and averaged across all valid days. To be included in the analyses, a minimum of three valid days. Fourteen cyclists and sixteen non-sporting referents returned their ActiGraph and had sufficient data for analysis.

The osteogenic index is a measure of high impact loading (28, 29). We calculated the osteogenic index from the raw ActiGraph data with a custom-written Matlab script (R2015b, Mathworks, Inc., Natick, MA, USA) according to Ahola and colleagues (30). In brief, resultant acceleration was calculated from the 3-dimensional data. No smoothing was applied to the recorded signal. Data were analysed in non-overlapping 24-hour epochs using the device-recorded time-stamps to start the first epoch from 00:00 of the first wear day, and ending at the 24:00 of the second to last wear-day and a daily osteogenic index was subsequently calculated for each 24-hour epoch. The daily osteogenic index of a particular epoch was calculated by identifying each individual peak on the resultant acceleration over 1.3 g. Subsequently, the maximum acceleration of each peak was added to an array resulting in an array of maximum accelerations of each of the peaks. Thirty-two histogram bins were then created from 1.3 to 10.8 g with any value higher than 10.8 included in the final bin (30), and a histogram of the maximum acceleration array was produced. Finally, the daily osteogenic index was calculated as $OI = \sum_{j=1}^{32} a_j ln(N_j + 1)$ where a = the lower limit of the histogram bin, j = the index of the histogram bin, N = count of peaks in a histogram bin and the average value per subject was used in further analysis.

Statistical analyses

The “R” statistical environment (version 2.10.1, www.r-project.org) was used for all statistical analyses. For continuous variables, T-tests were performed comparing the cyclist and non-sporting referent groups. Effect size was calculated as the mean of the cycling group minus the mean of the control group, divided by the pooled standard deviation. The group*gender interaction was examined on analysis of variance for the functional, physical activity and average lumbar spine MR variables. An alpha-level of 0.05 was taken for statistical significance.

RESULTS

Sitting time before entering the MR scanner was mean(SD) 44.8(20.9) min and comparable between cyclists and referents (p=0.42). Despite being height-matched, cyclists showed 0.75(1.08) mm (p=0.006) higher IVDs than referents (Table 2). Cyclists showed a 10.5(18.3) ms longer average lumbar intervertebral disc T2-time compared to referents (p=0.021; Table 2). This effect was most prominent at the lower thoracic and upper lumbar levels and also in the central region of the disc (Figures 2; Figure 3). Cyclists had longer IVD T2-times at T11/12 (123.8[20.9] ms vs 100.3[20.9] ms in referents; p=0.003), at T12/L1 (118.9[19.8] ms vs 96.7[19.8] ms; p=0.003), at L1/2 (109.3[15.0] ms vs 95.2[15.0] ms; p=0.011) and at L2/3 (104.8[12.2] ms vs 95.5[12.2]; p=0.029). No significant differences were observed between the groups at L3/4, L4/5, and L5/S1 (all p>0.2). No significant group*gender interactions were observed.

Cyclists' average spinal psoas muscle size was greater +118(365) mm^2 and psoas muscle cross-sectional area at L5 was greater +304(581) mm^2 compared to controls (NS and p=0.034, respectively; Table 3). Cyclists' spinal psoas muscle also showed less fat content that controls (-3[5]%; $p \leq 0.035$; Table 3). Cyclists' quadratus lumborum at L1 ($p \leq 0.012$) and erector spinae at L4 were also less adipose than controls (Table 3). Cyclists had longer trunk extension trunk flexion endurance times +66(128) seconds (p=0.036) and +90(142) seconds (p=0.011) respectively, compared to referents (Table 1). Cyclist accelerometry data demonstrated they were more physically active, with more high-impact loading (osteogenic index; Table 1).

DISCUSSION

We found that cyclists who did not report low back pain had greater IVD height, better IVD hydration, hypertrophy of the psoas muscle (trunk flexor), similar lumbar extensor muscle size, lower muscle fat content and higher isometric muscle endurance than non-sporting controls who also did not have a history of spinal pathology. These results contrast with prior reports (11–14) attributing to cycling no spinal benefit or worse listing cycling as a risk factor for spinal pain. As such, our findings refute the hypotheses (11) that high volume cycling is associated with detrimental effects on IVD or on trunk muscle function.

Intervertebral disc degeneration is associated with higher incidence and severity of low back pain (31, 32). Intervertebral disc degeneration is characterised (33) by loss of water and glycosaminoglycans from the central disc nucleus pulposus with subsequent loss of water signal intensity on imaging, reductions in intervertebral disc height, loss of separation between the nucleus pulposus and the annulus fibrosus, and a reduction in hydrostatic pressure inside the disc. Whilst radiological grading (22) is commonly used for the quantification of disc degeneration clinically, more sensitive measures, such as the measurement of T2 relaxation time (15) as used in the current study, can detect subclinical decreases in IVD hydration.

We found that high-volume cyclists had better intervertebral disc tissue quality than otherwise healthy, but non-sporting, people. This was characterised by greater disc height and longer T2-time (i.e. better hydration and glycosaminoglycan content), particularly in the nucleus pulposus. The nucleus pulposus develops hydrostatic pressure during spinal loading, with the collagen rings of the annulus fibrosus acting as a restraint. We are unaware of prior studies on the impact of cycling on the intervertebral disc. In contrast to current hypotheses in the

literature (11) on the causes of back pain in cyclists, we found no evidence of a detrimental impact of high-volume road cycling on low thoracic and lumbar IVDs. Quite the opposite, the current findings show that this sample of high-volume road cyclists without back pain had better IVD quality than non-sporting people without back pain.

The lumbar multifidus muscle is considered a 'core stabiliser' of the spine (34). In cyclists with low back pain, one study (12) reported atrophy of ‘stabiliser’ muscles, including multifidus, compared to cyclists without back pain. In the current study, cyclist multifidus, lumbar erector spinae and quadratus lumborum showed similar sizes to healthy non-sporting referents. We observed a hypertrophy of the psoas muscles with a lower fat content in cyclists. Psoas hypertrophy has been reported in athletes in association with training loads (35). For example, athletes in unilateral kicking sports have greater psoas muscle size on their dominant side (36). Muscle fat accumulates with age (37) and lumbar musculature fat accumulation has been reported in people with back pain (38, 39). Muscle training, in particular strength training (40), has resulted in reductions of intra-muscular fat. Low fat content can therefore be interpreted as a sign of muscle health. Our measures of decreased intramuscular fat in psoas, quadratus lumborum and erector spinae support that core spinal muscles benefitted from cycling. Spinal muscle sizes and fat content on MR are anatomical rather than functional assessments of muscle health. A more functional outcome of the effect of cycling on core muscles is the measure of trunk muscle endurance. Consistent with the anatomical images, cyclists had greater trunk muscle endurance than non-sporting controls. As such, again, the current study decisively departs from literature attributing a detrimental impact of cycling on the core muscles of the spine (11).

We intentionally excluded cyclists with back pain to assess whether cycling *per se* may cause detrimental effects to the spine. Prior studies (5, 6) have reported that approximately half of competitive cyclists suffered from low back pain. Despite this, the point prevalence of back pain in cyclists (7) appears similar to the wider community (8). In our view, it remain open whether back pain is actually more prevalent amongst cyclists than in the wider community. We postulate, however, that back pain in cyclists may have different (ergonomic) risk factors than in the non-cyclists members of the community.

Some investigators attributed the back pain of cyclists to spinal tissue trauma (11). Consequently, mitigating efforts have focused on optimizing the cyclists spinal posture (10, 41, 42) and bicycle engineering to reduce spinal flexion (5, 43). However, Brier and Nyfield (14) had failed to find an association between trunk flexibility and back pain in cyclists. Similarly, a more recent study found that trunk flexibility and strength and bicycle engineering changes failed to predict back pain in cyclists (44). Our study involved asymptomatic high volume cyclists and controls. In line with these prior works, our findings imply that cycling *per se* does not cause the deleterious spinal changes that are typically associated with, or considered risk factors for, back pain. Factors related to cycling other than posture maintenance such as prior trauma from a road accident or training injuries may need to be investigated. Bicycle setup should continue to be considered more deeply. The current cross-sectional study results support a broad scope prospective study to identify risk factors for back pain in cyclists with a focus on factors beyond the spinal tissues alone.

The current work has strengths and limitations. One of the strengths was to exclude people with spinal disease. The impact of back pain on muscle function, size, quality and the intervertebral disc would have constituted a confounding factor for the effect of cycling on

spinal outcomes. However, given our findings, future research should now consider comparisons between cyclists and sedentary controls both with and without back pain to further elucidate differences in spinal structure. We also restricted the age range of participants to people aged 25 to 35 years to avoid any confounding impact of age-related changes on the spine. Whilst our objective, hip worn, accelerometry data found cyclists to be more physically active, this was only significant for light physical activity and the osteogenic index for high-impact physical activity. Hip worn accelerometers are less able to pick up the vigorous lower-limb movements during cycling and the physical activity results observed in cyclists here may not relate to the actual cardiometabolic load. The muscle fat content represents an estimate based on MR signal and not an anatomical content: the relationship between fat and signal intensity on T1-weighted imaging is non-linear. As such, whilst cyclists have less psoas muscle fat signal than non-sporting controls, the histological difference in intra-muscular fat content may differ from the percentages reported.

In conclusion, high volume cyclists displayed the following spinal benefits: higher IVD with better hydration, in particular of the nucleus pulposus, similar or superior paraspinal muscle size with lower fat content compared to non-sporting controls. These data support the notion that cycling in and of itself is not detrimental to the spine; rather, in contrast, may be associated with beneficial changes at the spine.

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COMPETING INTERESTS

DLB, MQ, NDR, YL, DC, GT and TR declare no competing interests.

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AUTHOR CONTRIBUTIONS

Belavy: Secured funding. Conception and design of the experiments. Image analysis. Statistical analysis. Interpretation of the data. Drafting the article.

Quittner: Subject recruitment. Data collection. Image analysis. Approved final version of manuscript.

Ridgers: Conception and design of the experiments. Analysis of ActiGraph data. Drafting of accelerometer analysis methods and revision of the manuscript. Approved final version of manuscript.

Ling: Radiological grading of disc degeneration. Approved final version of manuscript.

Connell: Conception and design of the experiments. Approved final version of manuscript.

Trudel: Conception and design of the experiments. Interpretation of the data. Drafting of the manuscript. Approved final version of manuscript.

Rantalainen: Data analysis. Drafting of accelerometer analysis methods and revision of the manuscript. MRI analysis methods. Approved final version of manuscript.

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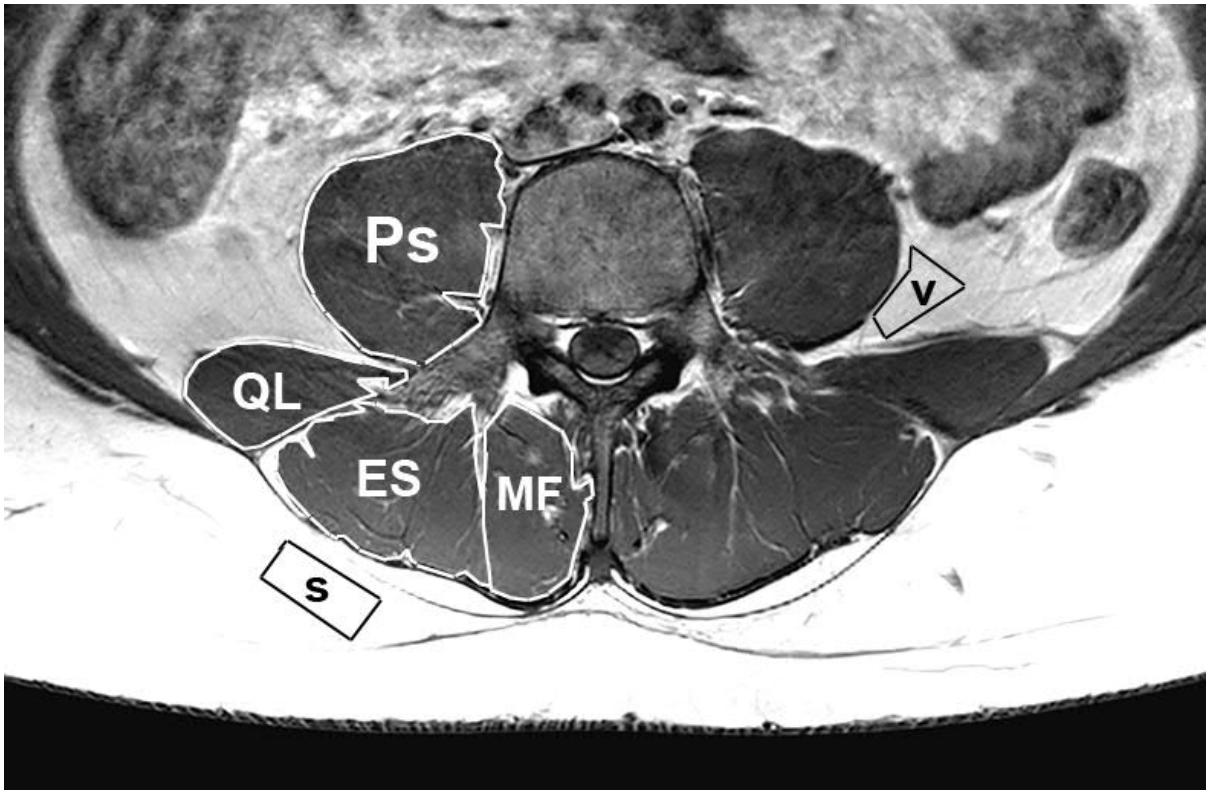
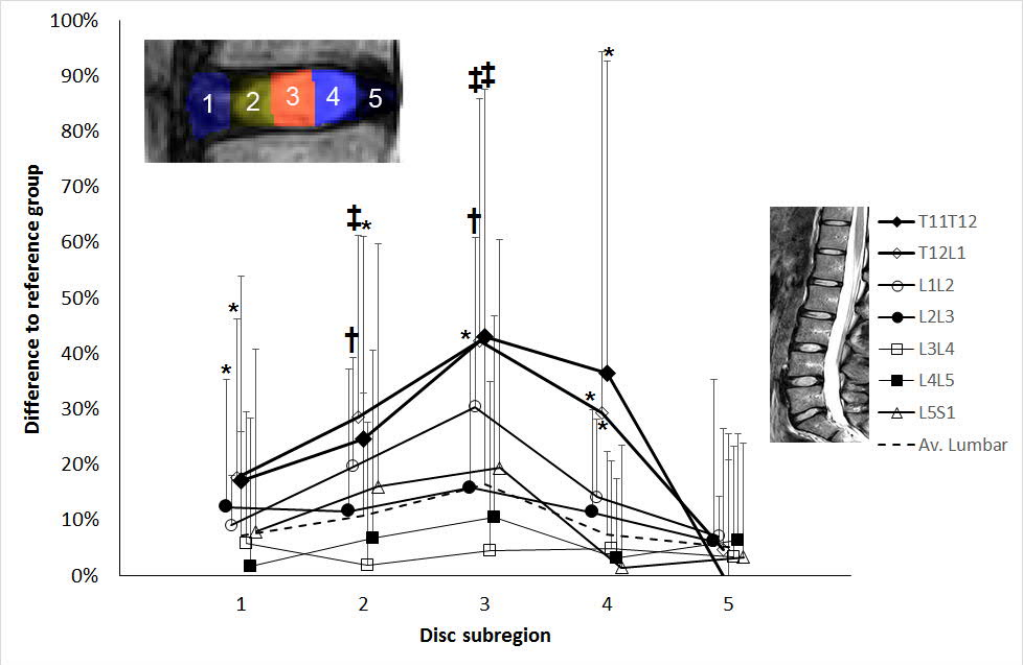
Figure 1: Analysis of T1-weighted paraxial images for muscle quantification.



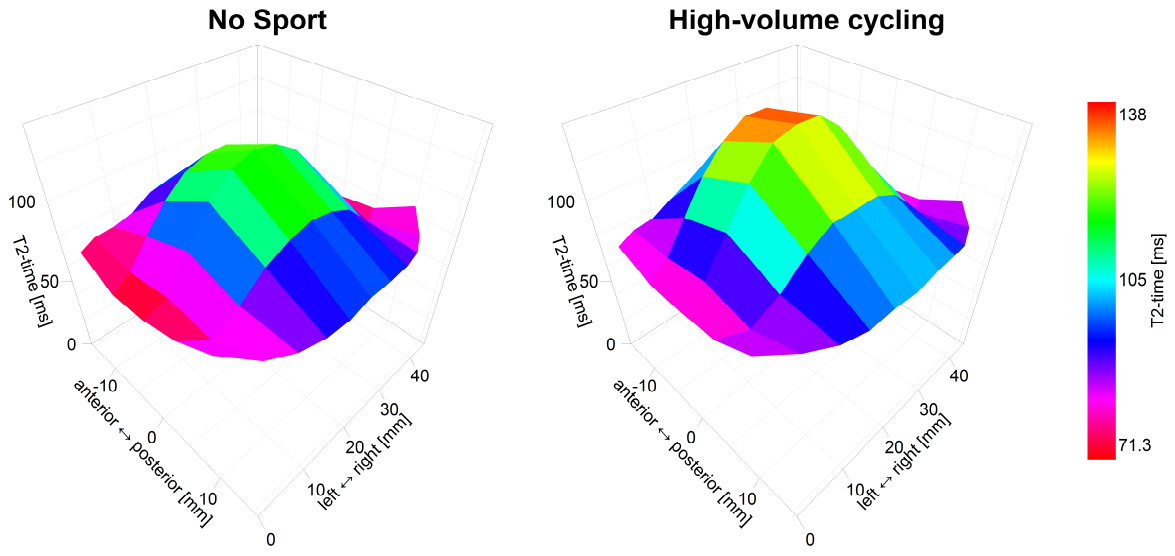
An image at the L4 vertebral level is shown. Ps: psoas, QL: quadratus lumborum, ES: erector spinae, MF: mutlfidus. The black regions of interest mark the position of the subcutaneous (s) and visceral (v) fat references.

Figure 2: Beneficial impact of cycling on intervertebral disc hydration in the nucleus pulposus and at the lower thoracic and upper lumbar spine.



Values are mean(SD) percentage difference of the cyclist group to non-sporting group in each disc subregion (see panel top left) and at each vertebral level of T2-time. *: $p<0.05$; †: $p<0.01$; ‡: $p<0.001$ and indicate significance of difference to the non-sporting group. Note that the differences to the non-sporting group are greatest in magnitude at the lower thoracic and upper lumbar levels. Also, note that effects are greatest in magnitude in the central portions of the disc (subregions 2, 3 and 4) where the nucleus is located.

Figure 3: Three dimensional representation of the effect of cycling on the intervertebral disc hydration: impact is greatest in the nucleus pulposus.



Values are mean T2-time interpolated across the volume of the IVD in each group. Data have been averaged from all lumbar discs. A longer T2-time is associated (15) with more water and glycosaminoglycan content. The intervertebral disc nucleus pulposus (NP; at centre of 3D plot) is a hydrated gel like tissue with a higher concentration of glycosaminoglycans and hence longer T2-time, surrounded by the collagenous rings of the annulus fibrosis with comparatively less glycosaminoglycans and water content (peripheral regions on 3D plot). Cyclists NP T2 values were higher than referents in all IVD regions but more so in the NP.

Table 1: Participant characteristics, isometric trunk endurance and physical activity levels.

Parameter	Non-sporting controls	High-volume cycling	Effect size
Subject characteristics			
N (N females)	18 (9)	18 (10)	
Age (yrs)	29.3(3.9)	29.9(3.8)	
Height (cm)	174.1(8.4)	174.4(8.9)	
Weight (kg)	77.0(17.5)	73.7(11.2)	
Years of cycling participation (yrs)	-	9.2(5.0)	
Duration of cycling per week (hrs/wk)	-	11.9(3.2)	
Cycling distance per week (km/wk)	-	267(100)	
Functional testing			
Trunk flexor endurance time (s)	118.4(89.2)	208.7(110.1)*	0.90
Trunk extensor endurance time (s)	172.0(98.1)	238.1(82.5)*	0.73
Ratio extensor:flexor endurance	2.0(1.3)	1.4(0.8)	-0.49
Objectively measured physical activity			
Sedentary time (min/d)	737.3(214.2)	737.7(213.4)	0.00
Light physical activity (min/d)	162.3(53.4)	204.5(52.4)*	0.80
Moderate-to-vigorous physical activity (min/d)	37.1(18.2)	47.6(26.6)	0.47
Osteogenic index	165.6(41.2)	217.5(80.4)*	0.80

Values of continuous variables are mean(SD). *: $p < 0.05$ and indicate significance of difference to the non-sporting group.

Table 2: Height-matched high-volume cyclists have greater disc height and hydration.

Parameter	No Sport	High-volume cycling	Effect size
IVD T2-time (ms)	105.6(9.9)	116.1(15.4)*	0.81
IVD height (mm)	7.0(0.7)	7.8(0.8)†	0.98
IVD anteroposterior width (mm)	25.7(2.2)	26.5(2.2)	0.39
IVD volume (cm^3)	9.6(2.1)	10.9(2.5)	0.57
Intervertebral distance (mm)	34.4(1.8)	35.1(2.4)	0.35
IVD height relative to vertebral body height	29.6(3.8)%	32.4(2.9)%*	0.82
Pfirrmann grade	2.3(0.4)	2.2(0.3)	0

Values are mean(SD) and effect size averaged across all lumbar vertebral levels. *: $p < 0.05$; †: $p < 0.01$ and indicate significance of difference to the non-sporting group. IVD: intervertebral disc. See Figure 2 for detail on differences in hydration between groups within subregions of the IVD and at different vertebral levels.

Table 3: Cyclists have greater psoas size and less intramuscular fat in psoas and quadratus lumborum.

Vertebral level	Muscle area (mm^2)			Intramuscular fat percentage					
				Relative to intra-abdominal fat			Relative to subcutaneous fat		
	No Sport	High-volume cycling	Effect size	No Sport	High-volume cycling	Effect size	No Sport	High-volume cycling	Effect size
				Psoas					
AvLx	949(237)	1068(278)	0.46	40(4)%	37(4)%*	-0.78	30(3)%	27(4)%*	-0.73
L1	207(79)	225(73)	0.24	39(5)%	31(6)%‡	-1.38	29(5)%	23(6)%†	-1.14
L2	531(159)	594(165)	0.39	39(4)%	35(5)%†	-0.96	29(3)%	25(5)%†	-0.93
L3	980(266)	1085(303)	0.37	40(5)%	38(3)%	-0.44	29(3)%	27(4)%	-0.54
L4	1365(367)	1573(421)	0.53	41(4)%	40(3)%	-0.24	30(4)%	29(4)%	-0.38

L5	1556(309)	1860(491)*	0.74	42(4)%	42(3)%	0.08	31(3)%	31(4)%	-0.16
				Erector spinae					
AvLx	1454(440)	1534(312)	0.21	41(5)%	38(3)%	-0.59	30(4)%	27(4)%	-0.63
L1	1473(422)	1564(349)	0.24	32(5)%	29(6)%	-0.66	24(5)%	21(6)%	-0.58
L2	1655(488)	1732(358)	0.18	36(6)%	33(4)%	-0.62	27(5)%	24(5)%	-0.61
L3	1581(490)	1672(317)	0.22	41(5)%	38(3)%	-0.64	30(4)%	27(4)%	-0.65
L4	1389(454)	1501(322)	0.28	45(7)%	43(3)%	-0.56	33(4)%	31(4)%*	-0.69
L5	1133(431)	1199(351)	0.17	48(8)%	48(5)%	-0.08	35(4)%	34(4)%	-0.34
				Multifidus					
AvLx	487(117)	474(101)	-0.11	44(6)%	45(5)%	0.24	33(3)%	32(4)%	-0.02
L1	217(58)	233(64)	0.27	41(5)%	43(6)%	0.46	30(3)%	31(5)%	0.23

L2	305(72)	309(107)	0.04	43(6)%	45(5)%	0.33	32(3)%	32(4)%	0.12
L3	450(98)	454(118)	0.04	45(6)%	46(5)%	0.20	33(3)%	33(3)%	-0.04
L4	638(169)	614(120)	-0.16	46(6)%	46(5)%	0.03	34(4)%	33(4)%	-0.24
L5	782(216)	761(145)	-0.11	47(6)%	47(5)%	0.08	34(4)%	34(4)%	-0.19
					Quadratus lumborum				
AvLx	362(130)	368(102)	0.05	36(3)%	35(3)%	-0.40	27(3)%	26(4)%	-0.41
L1	151(66)	162(56)	0.19	33(3)%	28(4)%†	-1.08	24(4)%	20(4)%*	-0.91
L2	276(117)	284(110)	0.07	36(3)%	34(4)%	-0.58	26(3)%	24(5)%	-0.50
L3	417(170)	431(139)	0.09	38(3)%	39(3)%	0.39	28(4)%	28(4)%	0.01
L4	566(195)	596(166)	0.17	40(3)%	41(3)%	0.26	30(4)%	29(4)%	-0.06
L5	-	-	-	-	-	-	-	-	-

Values are mean(SD) and effect size. AvLx: data averaged from all lumbar levels. *: $p < 0.05$; †:$p < 0.01$; ‡: $p < 0.001$ and indicate significance of difference to the non-sporting referents. Erector spinae intramuscular fat relative to subcutaneous fat was lower at L4 in cyclists.