

Mechanical and morphological determinants of peak power output in elite cyclists

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Title: Mechanical and Morphological Determinants of Peak Power Output in Elite Cyclists.

Running Title: Peak Power Output in Sprint Cycling

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ABSTRACT

Mechanical peak power output (PPO) is a determinant of performance in sprint cycling. The purpose of this study was to examine the relationship between PPO and putative physiological determinants of PPO in elite cyclists, and to compare sprint performance between elite sprint and endurance cyclists. Thirty-five elite cyclists (18 endurance; 17 sprint) performed duplicate sprint cycling lab tests to establish PPO and its mechanical components. Quadriceps femoris (Q_{VOL}) and hamstrings muscle volume (HAM_{VOL}) were assessed with MRI, vastus lateralis pennation angle ($P\theta_{VL}$) and fascicle length (FL_{VL}) were determined with ultrasound imaging, and neuromuscular activation of three muscles were assessed using EMG at PPO during sprint cycling. For the whole cohort there was a wide variability in PPO (range 775-2025 W) with very large, positive, bivariate relationships between PPO and Q_{VOL} ($r = 0.87$), HAM_{VOL} ($r = 0.71$) and $P\theta_{VL}$ ($r = 0.81$). Step-wise multiple regression analysis revealed that 87% of the variability in PPO between cyclists was explained by two variables Q_{VOL} (76%) and $P\theta_{VL}$ (11%). The sprint cyclists had greater PPO (+61%; $P < 0.001$ vs endurance), larger Q_{VOL} ($P < 0.001$) and BF_{VOL} ($P < 0.001$) as well as more pennate vastus lateralis muscles ($P < 0.001$). These findings emphasise the importance of quadriceps muscle morphology for sprint cycling events.

Key Words: Maximum cadence; Maximum power; Maximum torque; Muscle

1 INTRODUCTION

Peak power output can be described as the maximum power one can achieve during maximal tasks of a short duration (i.e. < 7 s).¹ Peak power output is a strong predictor of sporting performance during maximal athletic tasks such as sprint running,² jumping³ and cycling.⁴ In cycling, mechanical peak power output (PPO) relative to body mass or frontal area has been highly correlated with both acceleration^{5,6} and maximum velocity⁴, both of which are related to track sprint cycling performance. Despite these relationships being well established, the underlying physiological determinants of PPO in cycling are poorly researched and understood. Whilst lean leg volume,⁴ muscle architecture⁷ and neuromuscular activation⁸ have been suggested to influence cycling PPO, their quantitative contribution has not been examined. A better understanding of these factors will facilitate exercise prescription targeted more effectively to the key determinants of PPO and may help to maximise performance.

Energy systems and mechanical power output profiles of athletes in sprint cycling disciplines such as BMX and track sprint have been previously documented to have similarly high PPO.^{4,9,10} However, physiological comparisons between sprinters (that compete in events that are maximal, ‘all-out’ and usually last between 9 - 60 s) and endurance riders (events that last from ~4 min to in excess of 4 hours) have not examined the physiological factors that may underpin any differences in PPO.

Theoretically muscle size, specifically muscle volume, is a key predictor of neuromuscular power¹¹ and there is evidence, that, for example, quadriceps femoris volume explains a high proportion of the variance in single joint knee extension (~80%) and squat jump (90%) power.¹² It has also been suggested that muscle volume is a major predictor of PPO in sprint cycling.^{6,13} However, previous work has examined relatively crude estimates of muscle mass/volume (e.g. based on tape measures of superficial anthropometry that are not

muscle group specific) in relation to sprint cycling performance.^{4,14} Of the lower limb muscle groups, strength of the knee extensors appears to be the best predictor of cycling PPO,^{15,16} and thus, accurate assessment of quadriceps femoris muscle volume e.g. with magnetic resonance imaging (MRI), the gold standard technique,¹⁷ might be expected to be a key determinant of PPO.

Another important component of skeletal muscle mechanics and function is muscle architecture, including pennation angle and fascicle length, that can be assessed *in vivo* with ultrasound imaging. A greater pennation angle is thought to be associated with an improvement of the generation of force output for contractions against high loads by packing more sarcomeres in parallel.^{18,19} Whilst fascicle length plays an important role in determining shortening velocity of a muscle.²⁰

Furthermore, the ability to develop contractile force, and thus power, rapidly is dependent on neuromuscular activation^{21,22} and likely plays a role in cycling PPO.¹³ However, the relationship between PPO and neuromuscular activation in a large cohort of elite cyclists has not been investigated.

The primary aim of this study was to examine the relationship of a range of putative neuromuscular determinants (muscle volume, architecture and neuromuscular activation) with cycling PPO. This involved a large cohort of elite cyclists that were all familiar with performing maximum cycling efforts and drawn from different disciplines in order to ensure a wide range of sprint cycling values. The secondary aim was to compare and characterise the sprint performance and physiological measures of elite sprint and endurance cyclists. We hypothesised that muscle volume of the quadriceps femoris would be the primary predictor of sprint cycling PPO.

2 METHODS

Participants

Thirty-five elite male cyclists volunteered to take part in the study (mean \pm SD age, 22 ± 4 yr; stature, 179.1 ± 5.9 cm; mass, 77.4 ± 11.3 kg). The whole cohort included two groups: sprint ($n = 17$; age, 21 ± 3 yr; stature, 178 ± 4.0 cm; mass, 85.3 ± 9.2 kg) and endurance cyclists ($n = 18$; age, 22 ± 4 yr; stature, 179.1 ± 5.9 cm; mass, 69.1 ± 5.9 kg). The sprint included disciplines that are ‘all-out’/ maximal for ≤ 60 s, i.e. BMX ($n = 4$) and track sprinters ($n = 13$). Endurance included disciplines that were > 4 mins in duration and are not ‘all-out’, i.e. track endurance who rode team pursuit ($n = 9$), road endurance and/or road time trial ($n = 7$) and mountain bike ($n = 2$). Twenty-eight of the cyclists were currently competing internationally in their respective Union Cycliste Internationale (UCI) disciplines/categories, as well as training on a full-time basis for at least the past two years. More specifically, their collective experience and success included: 2 Olympic medals, 8 Olympic games representations, 3 Paralympic medals, 3 Paralympic games representations (as a pilot [who are able-bodied and compete individually at national and up to UCI Class 1 level events] or stoker [other than being visually impaired, they are fully able-bodied] of tandem), 10 Senior World Championship medals, 37 Senior World Championships representations, 8 Senior Para-cycling World Championship golds and 6 Senior Para-cycling World Championship medals (as pilot or stoker of tandem). The remaining seven participants who were not competing internationally were competing in ‘Elite’ category national level road cycling events ($n = 4$) or had won national level medals on the track ($n = 3$). Ethical approval was attained from Northumbria University Research Ethics Committee. Following explanation of the study design and protocol, the cyclists provided written, informed consent to prior to their participation in the study.

Study Overview

Before all experimental sessions at the laboratory, cyclists were instructed to avoid caffeine and food for 3 h prior to testing and to avoid strenuous exercise in the 24 h before each session. Cyclists made two identical cycling laboratory visits within 7 days at the same time of day (± 1 hour). Firstly, the cyclists assumed their race cycling specific position, on a custom-built ergometer that had the cast flywheel clamped, to ensure the cranks were stationary. In this position, architecture of the vastus lateralis (i.e. pennation angle [$P\theta_{VL}$] and fascicle length [FL_{VL}]) was assessed at rest prior to exercise using ultrasound²³. Subsequently, the cyclists had surface electromyography (EMG) electrodes placed on three muscles of both legs (gluteus maximus [GM], biceps femoris long head [BF], and vastus lateralis [VL]) and mounted another custom-modified isovelocity ergometer (again, the position mirrored their racing position). A standardised warm-up of 10 mins at 80 – 90 revolutions per minute (RPM) and 100 – 150 W followed by a maximal 2 s sprint at 125 RPM was completed by each cyclist. Once this was completed a series of isovelocity sprints (4 s maximal sprints at each of five velocities: 60, 115, 125, 135 and 180 RPM was performed in this order to assess PPO, as well as torque, whilst surface EMG was simultaneously recorded. On a third occasion, within 7 days of the cycling laboratory visits, MRI was used to assess quadriceps femoris and hamstrings muscle volume of both legs of each cyclist.

Muscle Architecture

For the architecture measures a custom-built cycling ergometer (United Kingdom Sports Innovation) was set-up according to the individual cyclist's track or road bike set-up. BMX riders had experience in riding road and track bikes were set-up to their track or road

bike position fitted which was typical to a track cycling set-up when assuming the dropped handle bar position (i.e. closed hip angle, flat back parallel with the floor and bent arms). The ergometer could be made isometric, as previously used.¹⁶ Before the cyclist mounted the ergometer, bib shorts were pulled up to expose their thighs in order to allow mid-thigh to be measured and marked. When the cyclists first mounted the ergometer for the ultrasound imaging, the flywheel was clamped to ensure that the crank position was fixed with the drive-side (right) crank positioned at 90° from top, dead centre (TDC). Once in this position, the cyclists were asked to take their racing position with their hands on the ‘drops’.

An ultrasound (5-10 MHz scanning width 92 mm and depth 65 mm, EUP-L53L; Hitachi EUB-8500) linear array transducer was used to capture B-mode ultrasound images. Water-soluble transmission gel was used to coat the transducer that was positioned with minimal pressure over the skin. Images were captured with the transducer placed on the medial, longitudinal line of the muscle while positioned on the skin over the VL at 50% of femur length (from the knee joint space to the greater trochanter) to correspond with the area of greatest anatomical CSA²⁴. The transducer was orientated perpendicular to the skin and parallel to the fascicular path. Parallel fascicle alignment was presumed when transducer orientation produced an image whereby the aponeuroses and the fascicle perimysium trajectory were clearly identified with no visible fascicle distortion at the image edges. Once the images were captured, the cyclists were instructed to switch lead legs and have the non-drive side (left) crank positioned at 90° from TDC and the process was repeated with the left VL. Images were later imported into analysis software (ImageJ, v.1.46; National Institutes of Health, Bethesda, MD, USA) to measure FL_{VL} and $P\theta_{VL}$. The FL_{VL} was measured as the length of the fascicular path between the superficial and deep aponeurosis. The manual (fascicular line tracing) linear extrapolation approach was adopted when the full fascicle length could not be seen within the ultrasound image. The $P\theta_{VL}$ was measured as the angle

between the fascicular path and the insertion of fascicles into the deep aponeurosis. Three different ultrasound images of each leg were recorded and analysed during each visit before first averaging the measured values from each session, and then averaging across the two sessions. The intra-rater repeatability of measures $P\theta_{VL}$ had CV of 4.1% and ICC of 0.86. and FL_{VL} had a CV of 1.9% and ICC of 0.98 and within-participant repeatability $P\theta_{VL}$ had CV of 2.9% and ICC of 0.91. and FL_{VL} had a CV of 1.3% and ICC of 0.97.

Electromyography

A wireless, surface EMG system (Delsys Trigno® Wireless EMG systems, Boston, MA, USA) was used to ascertain muscle activation by measuring EMG amplitude. Once muscle architecture assessment was complete, EMG electrodes were placed (in accordance with standard SENIAM recommendations²⁵ on each leg over the GM, BF and VL. Each location was shaved, lightly abraded, and then cleaned with a sterilised alcohol wipe. To ensure optimal electrical conductance, the electrodes were then applied using self-adhesive interfaces (Delsys Trigno®, Boston, MA, USA), each site was marked with a semi-permanent marker to ensure consistent placement across sessions. Surface EMG signals were sampled at 2,000 Hz, amplified ($\times 1000$), band-pass filtered (20-450 Hz) using an external analogue-to-digital data acquisition system (Micro 1401, Cambridge Electronic Design, Cambridge, UK) and a PC utilizing Spike2 software (version 7.11, CED, Cambridge, UK).

EMG signal amplitudes were calculated as the root mean square (rmsEMG) over an epoch equivalent to one quarter of the full crank cycle for all isovelocity cadences (e.g. 250 ms at 60 RPM). Peak rmsEMG values of each muscle (GM, VL, BF) were averaged over the first three full crank revolutions (TDC to TDC) of each sprint/velocity. The peak rmsEMG amplitude during the 60 RPM isovelocity sprint was used to normalise peak rmsEMG from the isovelocity sprint with the highest measured PPO (peak rms EMG_{PPO}) i.e. peak

$\text{rmsEMG}_{\text{PPO}}/\text{peak rmsEMG}_{60}$ and used as criterion values of activation of each muscle at PPO (GM_{ACT} , VL_{ACT} , BF_{ACT}). From the data previously collected in our lab the coefficient of variation for between-session peak rmsEMG reliability during isovelocity cycling at 60 RPM as a reference task for GM, VL and BF was 9.9, 13.0 and 9.5%, respectively.

Sprint Cycling Performance Test

Isovelocity sprints were performed on an SRM ergometer (Schoberer Rad Messtechnik, Jülich, Germany) that was modified to have a motor accelerate the flywheel to the prescribed cadence for isovelocity sprints. As with the custom-built ergometer used for muscle architecture, the protocol on isovelocity SRM ergometer was identically set-up to their racing positions. All efforts were performed in the saddle. The cyclists were instructed to perform all the efforts in the saddle. This were only monitored by the investigator and no restraint or strapping was used to ensure they remained attached to the saddle. Each cyclist performed each effort on 'drop' handlebars, using clipless pedals and racing attire. The original cranks were replaced with 170 mm instrumented cranks to record instantaneous torque, crank angle, and angular velocity directly as raw data for both right and left crank arms (Factor Cranks, BF1 Systems, Diss, UK), which were sampled at 200 Hz with a separate, wireless electronic data logger (BF1 Systems, Diss, UK). Torque and power was measured over each revolution and was processed offline as previously done.¹⁶ Raw data recorded from the cranks onto the wireless data logger were imported in to Spike2 and analysed offline using custom scripts to calculate mean torque, power and cadence per revolution from TDC to TDC.

Prior to performing the maximal isovelocity efforts to determine PPO, power-cadence and torque-cadence relationship, cyclists undertook a standard 10-min warm-up of submaximal cycling at a self-selected intensity (between 100–150 W) and cadence (between

80–90 RPM) with a 2 s maximal effort at 125 RPM. For the maximal isovelocity efforts, participants performed 4 s sprints at 60, 115, 125, 135 and 180 RPM. The order of cadences was selected at random and every cyclists performed the efforts in the following order: 115, 60, 135, 125 and 180 RPM. Prior to each effort, the motor was brought up to the desired velocity and participants were instructed to pedal below the pre-set cadence and reminded to ‘attack the effort as fast and as hard as possible’. Then the investigator gave a 3 s countdown and the participants performed a 4 s maximal effort at the set cadence. Each isovelocity effort was performed once per lab visit with a 5 min period of passive rest between each isovelocity sprint.

The maximum power output over three consecutive revolutions (from TDC to TDC) at each cadence was used and then averaged over both sessions. From that, power-cadence and torque-cadence relationships were established by fitting a quadratic and linear equation, respectively, by the least square method as previously used.^{4,26} The apex of the power-cadence relationship was interpolated to derive PPO (as well as PPO: mass by dividing PPO by body mass [W/Kg]) and cadence at PPO (C_{OPT}). Individual torque-cadence relationships, maximal torque (T_{MAX}) and maximal cadence (C_{MAX}) were extrapolated.

MR Imaging

On a separate occasion, within 7 days of the cycling laboratory visits, muscle volume of both legs was measured via MR imaging (1.5 T Signa HDxt; Alliance Medical Limited, Warwick, UK). T1-weighted axial images of each thigh originating at the anterior-superior iliac spine and finishing at the knee joint space (scan parameters: time of repetition = 600 ms; time to echo = 14 ms; image matrix 512 pixels \times 512 pixels; field of view 260 mm \times 260 mm; slice thickness = 5 mm; and interslice gap = 5 mm). An array of fish-oil capsules were attached using micro-pore surgical tape on and around the anterior-superior iliac spine and

knee joint space as previously done ²⁷. This was to help the operator orientate any overlapping blocks during the analysis stage. Participants were asked to refrain from exercise in the thirty-six hours before the scan and sat quietly for > 1 h before the scan. Participants lay supine with legs fully extended and strapped in position to discourage any extraneous movement that might cause image distortion.

Muscle volume was measured by an experienced operator, who was blinded to the participant's identity and performance data, using open source software (OsiriX Imaging SoftwareTM version 5.5.1, Geneva, Switzerland). Volume was calculated by measuring anatomical (CSA), in the axial plane, by manual segmentation of VL, vastus intermedius (VI), vastus medialis (VM) and rectus femoris (RF) as well as semitendinosus (ST) and semimembranosus (SM), long and short head BF. In each individual image using the 'closed polygon' tool. Manual outlining started with the most distal slice above the knee, at where the muscles were visible, and ended with the most proximal slice where the muscle was no longer visible. The total number of slices was noted and used to determine the length of the segment (length = $n \times 15$ mm, where n is the number of slices, given that MR image slices were 5 mm in thickness). On average, thirty images were analysed per thigh. Consequently, muscle volume was calculated by using Cavalieri formula: ²⁸

$$\text{Muscle Volume} = \sum_n e_i \times \text{CSA}_i$$

Where n is the number of slices used, and e_i is the distance between measured slices.

Knee extensor muscle volume (Q_{VOL}) was measured by summing the muscle volume of VL, VM, VI and RF of each leg. Hamstring muscle volume (HAM_{VOL}) was

measured by summing the muscle volume of long and short head BF, semitendinosus and semimembranosus of each leg. Both Q_{VOL} and HAM_{VOL} were averaged over both legs.

Statistical Analysis

All data is presented as mean \pm SD. A Shapiro-Wilk test of the measures showed that the data was normally distributed and suitable for parametric testing. Data from all thirty-five cyclists were used to perform bivariate correlations and subsequent regression analysis with the physiological measures. Initially, Pearson's product-moment correlations (r) were employed to examine the relationship between individual physiological variables and the criterion variable (PPO). The following criteria were adopted to interpret the magnitude of the relationship between test measures: <0.1 trivial, 0.1 to 0.3 small, >0.3 to 0.5 moderate, >0.5 to 0.7 large, >0.7 to 0.9 very large, and >0.9 to 1.0 almost perfect²⁹. In addition, the overall coefficient of determination (R^2) for the set of physiological measures with PPO was also calculated.

Variables that were significantly correlated with PPO were included in the step-wise regression analysis to predict PPO. With this set of predictors, our collinearity diagnostic exploration resulted in variance inflation factors of $2.0 - 5.0$ and tolerance of $0.20 - 0.80$, which indicate acceptable levels of multicollinearity.³⁰

The sprint and endurance groups within the whole cohort were compared using an independent-samples t-test for sprint performance measures (i.e. PPO, PPO: mass, C_{OPT} , T_{MAX} and C_{MAX}) and physiological measures (i.e. Q_{VOL} , HAM_{VOL} , $P\theta_{VL}$, FL_{VL} , GM_{ACT} , VL_{ACT} , BF_{ACT}). In addition, independent-samples t-tests were also used to compare the volume of individual quadriceps femoris (i.e. VL, VI, VM and RF) and hamstrings (biceps femoris short head, biceps femoris long head, ST and SM) muscles. Finally, the relative/proportional volume (percentage) of the whole muscle group accounted for by each

individual muscle within the quadriceps and hamstrings muscle groups was compared between sprint and endurance cyclists.

All physiological measures (mentioned above) were averaged over both limbs and then both sessions (with the exception of MR imaging). The level of statistical significance was set at $P < 0.05$ for all tests. All statistics were calculated using SPSS (IBM Corp. Version 24.0. Armonk, USA).

3 RESULTS

Collectively for all thirty-five riders, the average \pm SD, range (i.e. maximum and minimum) and fold variability (multiple between maximum and minimum value) of the performance and physiological measures are presented in Table 1. Very large, positive bivariate relationships were found between Q_{VOL} ($r = 0.87$; $P < 0.001$), HAM_{VOL} ($r = 0.71$; $P < 0.001$) and $P\theta_{VL}$ ($r = 0.81$; $P < 0.001$) with cycling PPO (Table 2; Figure 1). The remaining measures (FL_{VL} , VL_{ACT} , BF_{ACT} and GM_{ACT}) were unrelated to PPO. Subsequently, step-wise multiple regression analysis was done using the three significant predictor variables from the bivariate correlations (Q_{VOL} , HAM_{VOL} , $P\theta_{VL}$) to examine their combined relationship with PPO. The regression analysis found 87% of the variability in PPO between cyclists ($F_{(2, 28)} = 72.83$, $P < 0.001$) was explained by two variables Q_{VOL} (76%) and $P\theta_{VL}$ (11%).

The comparison of power-cadence and torque-cadence relationships between the groups of sprint and endurance cyclists (Figure 2) showed that sprint cyclists had substantially higher PPO ($\sim +579$ W; +47%; $P < 0.001$), PPO: Mass ($\sim +4.3$ W:Kg; +27%; $P < 0.001$), C_{OPT} ($\sim +11$ RPM; 8%; $P < 0.05$), T_{MAX} ($\sim +62$ N·m; +35%; $P < 0.001$) and C_{MAX} ($\sim +31$ RPM; +11%; $P < 0.05$; Table 3). In terms of the physiological measures sprint cyclists had significantly larger Q_{VOL} , HAM_{VOL} , volume of all the individual muscles of both groups and $P\theta_{VL}$ (all $P < 0.001$; Table 3; Figure 3). In terms of proportional volume of the

individual muscles, all four quadriceps (VL, VM, VI & RF) as well as three of the hamstrings (ST, SM, BF-1), were similar for sprint and endurance cyclists, however biceps femoris short head was proportionately larger in endurance vs sprint cyclists (17.5 ± 3.1 vs. $15.1 \pm 2.8\%$; $P = 0.0219$).

No significant differences were seen between groups when FL_{VL} , VL_{ACT} , and GM_{ACT} were examined whilst the endurance cyclists exhibited higher BF_{ACT} ($P < 0.05$) during sprint cycling.

4 DISCUSSION

To the author's knowledge, this is the first study to forensically investigate the physiological attributes that determine PPO in an elite, highly-trained cycling cohort. The main finding of this study was the very large, positive relationships between Q_{VOL} , HAM_{VOL} and $P\theta_{VL}$ with PPO, with multiple regression showing that in combination Q_{VOL} and $P\theta_{VL}$, 87% of the variability in PPO between cyclists could be explained. These findings appear to agree with the hypothesis that muscle volume of the quadricep femoris are the biggest (but not the only) predictors of PPO in sprint cycling. This demonstrates the importance of muscle morphology for sprint cycling performance. In contrast the remaining variables, particularly neuromuscular activation of three hip and knee joint muscles, showed no relationships with PPO. The secondary finding was that as expected elite sprint cyclists had substantially higher power-cadence and torque-cadence relationships (i.e. PPO, PPO: mass, C_{OPT} , T_{MAX} and C_{MAX}) than endurance cyclists and this was underpinned by greater Q_{VOL} (+52%), HAM_{VOL} (+52%) and $P\theta_{VL}$ (+20%).

The sprint performance measures in the current study were similar to previous reports. For example the sprint group recorded PPO of 1521 ± 186 W that was within 80 W of three previous studies using similar although somewhat smaller elite cohorts.^{4,31,32} The endurance

group in the current study had PPO of 942 ± 136 W, similar to untrained cyclists (941 ± 124 W)⁷ and somewhat lower than a previous elite endurance cohort (1122 ± 65 W).³² Of the physiological measures, the endurance cyclists had muscle volume and $P\theta_{VL}$ similar to the untrained groups.²⁷ The endurance group had muscle volume similar to long-term resistance trained participants²⁷ and $P\theta_{VL}$ similar to sprint runners.³³

Morphological Determinants of Sprint Cycling Performance

Q_{VOL} , HAM_{VOL} and $P\theta_{VL}$ all showed very large, positive bivariate relationships with PPO, and multiple regression analysis found Q_{VOL} and $P\theta_{VL}$ explained 87% of the variance in PPO, whereas the neuromuscular activation measures were unrelated to PPO. For the first time and in elite cyclists, this study shows that PPO is overwhelmingly determined by muscle morphology, particularly the size and pennation angle of the quadriceps, rather than the ability of the nervous system to activate the muscles.

Using MR imaging, a ‘gold standard’ method for determining muscle volume,¹⁷ we found Q_{VOL} i.e. the amount of skeletal muscle, alone explained 76% of the variance in PPO between cyclist, which makes this variable a desirable attribute for competitive (sprint) cyclists. Whilst no previous studies have carefully imaged the quadriceps and hamstrings muscles in relation to sprint cycling performance some crude estimates lower body/thigh muscle mass have been found to be moderately/strongly related to cycling PPO.^{4,7,34} Our findings for the predominant influence of Q_{VOL} on PPO, reinforce the importance of muscle size for neuromuscular power and that cyclists and their coaches be especially attentive to training and nutrition strategies to enhance Q_{VOL} . In particular resistance training is well known to stimulate hypertrophy and increased muscle volume. Elite sprint cyclists in the current study had slightly smaller Q_{VOL} than a long-term (mean 4-years) resistance trained, but not elite, cohort assessed with an almost identical MRI protocol.²⁷ This could be due to

the concurrent nature of performing both sprint cycling training in conjunction with resistance exercises that could attenuate hypertrophic responses.³⁵

$P\theta_{VL}$ was a strong correlate of PPO in the current study ($r = 0.81$) and given the relationship of muscle volume and PPO this might have been expected as pennation angle is known to be associated with muscle size indices (e.g.³⁶) and this was also the case in the current study (Q_{VOL} vs $P\theta_{VL}$ $r = 0.78$). However, what was perhaps more surprising was that $P\theta_{VL}$ was an independent predictor of PPO in addition to Q_{VOL} within the regression analysis. Suggesting that a high $P\theta_{VL}$ is advantageous for neuromuscular power even after muscle volume has been accounted for. This may reflect a net positive balance of advantages and disadvantages of increasing $P\theta_{VL}$ at the relatively low angles found in this study ($< 20^\circ$). For PPO, theoretically greater $P\theta_{VL}$ has the advantage of greater PCSA and thus higher force production capacity but potential disadvantages of loss of force transmission to the tendon and/or reduction in fascicle length and thus shortening velocity. In the current study PPO was unrelated to FL, which is somewhat contrary to the findings of a positive association of FL with sprint running (100 m) performance.³³ Furthermore, FL_{VL} was also unrelated to $P\theta_{VL}$ ($r = -0.23$), suggesting no negative consequence of increasing $P\theta_{VL}$ on FL_{VL} .

Data in this experiment indicated that peak muscle activation recorded with surface EMG exhibited no relationship with PPO, and thus was not a meaningful determinant of PPO in elite cyclists. Therefore, it is possible that more accurate and sensitive measures of neuromuscular activation, perhaps including surface EMG from more muscles and multiple sites per muscle³⁷, as well as alternative normalisation techniques,³⁸ might reveal a greater role for activation in determining PPO. However, given that muscle morphology explained 87% of the variability in PPO the unexplained variance was relatively small (13%) and the contribution of other independent factors, including neuromuscular activation appears limited for this performance task.

Magnitude of Morphological measures between Sprint cyclists and endurance cyclists

Sprint cyclists were greater in every measure of the sprint cycling performance test (i.e. PPO +61%, PPO: MASS +32%, T_{MAX} +43%, C_{OPT} +9% and C_{MAX} +12%) in comparison to endurance riders which is likely to be the consequence of sprint riders being greater in all the morphological measures that had a positive and significant relationship with PPO (i.e. Q_{VOL} , HAM_{VOL} and $P\theta_{VL}$). This perhaps gives further weight that the mechanisms of PPO.

The greater PPO of sprint cyclists (+61% vs endurance cyclists) appeared to be primarily due to their higher T_{MAX} (+43%), as opposed to a smaller difference in C_{OPT} (+9%). The greater T_{MAX} of the sprint cyclists was likely due to their greater Q_{VOL} and HAM_{VOL} as a greater muscle volume provides more sarcomeres in parallel that can exert a greater force/torque (the relationship between the sum of Q_{VOL} and HAM_{VOL} with PPO: $r = 0.81$). No differences were seen for FL_{VL} which further suggested that it may not be an important morphological determinant of sprint cycling ability.

The focus of this study has been the relationships between muscle morphology (size and architecture) with cycling PPO. The portion of the PPO differences that were not accounted for in this study could be explained by either, or a combination of, muscle fibre-type composition and muscle fibre contraction speed. With respect to skeletal muscle fibre type composition, it is thought that a higher proportion of type II muscle fibres have a substantial influence on muscle fibre PPO, primarily due to their higher maximum shortening velocity which is underpinned by the higher quantities of ATPase³⁹. The higher PPO as well as C_{OPT} and C_{MAX} in sprint cyclists could, in part, be attributed to a greater proportion of type II muscle fibres. Whilst there is relatively limited data in humans, one small study (n=10) reported a strong positive relationship ($r = 0.88$; $R^2 = 0.77\%$) between the proportion or number of fast-twitch fibres and C_{OPT} . As such, it is possible that muscle fibre type

composition, and thus maximum shortening velocity, could contribute to higher PPO of sprint cyclists, but needs to be examined further in future studies in athletic cohorts, although this is unlikely to be achieved in an elite athletic cohort of this level. This idea is underpinned by first principle models from Hill ^{41,42} and Huxley ⁴³, suggesting that fibre speeds (rather than fibre type) also could account for some differences in PPO in sprint cycling. Force-velocity characteristics of muscle are based on kinetics of cycling interaction between myosin cross-bridges and actin filaments within sarcomeres of muscle. Higher muscle fibre speeds have higher cross-bridge cycling rates that underpin muscle fibre speeds which could explain the higher torque offsets (and C_{OPT}) in sprint cyclists across the cadence ranges in comparison to the endurance cyclists.

The sprint cyclists also measured higher muscle volume in each individual muscle of the quadriceps femoris and hamstrings. Proportional muscle volume was also similar between sprint and endurance cyclists in all four individual quadriceps muscles and three out of four hamstrings muscles. The exception was that the bicep femoris short head, the smallest muscle examined in this study, which was proportionately larger in endurance than sprint cyclists. Therefore, only very limited evidence for muscle-specific regional hypertrophy / muscle mass distribution in one of the 8 muscles examined was seen. A previous study by Ema and colleagues (2016) suggested that in comparison to untrained men, experienced cyclists who were club level track cyclists who competed in sprint events had higher muscle volumes of the bicep femoris short head and semitendinosus. But no difference in the percentage of each individual muscle in the hamstrings were seen. The finding in the current study could suggest that the short head of the bicep femoris short head may have more of a role to play in endurance cycling than in sprint cycling.

Limitations of the Study

Although the data collection within the current study was extensive, there were a number of limitations associated with the methodology. Firstly, the selection of two different, highly specialised and distinct groups of cyclists may have created coincidental or exaggerated relationships by having big differences between groups for a whole cluster of variables (both assessed variables e.g. PPO and muscle volume, but also potentially unassessed variables e.g. fibre type composition, tendon stiffness). Therefore, the strength of the relationships between predictor and outcome variables in this study may actually be reflective of a range of predictor variables. Secondly, correlation does not demonstrate causality, and therefore whilst there were very strong relationships between Q_{VOL} and $P\theta_{VL}$ with PPO, and these factors in combination explained 87% of the variation of PPO, this does not necessarily mean that changes in one or both predictor variables will cause a proportionate increase in PPO. On the basis of the interesting findings of the current work it is recommended that future studies use a wider range of predictor variables (e.g. fibre type composition, or surrogate measures of contractile properties, and tendon stiffness) for cross-sectional analyses and particularly that intervention studies examine the effect of changing muscle morphology on cycling PPO.

This study largely focused on measures in the thigh as previous studies suggest that the physiological determinants of PPO are largely rooted there.^{15,16} Other muscle groups that almost certainly contribute to cycling PPO such as gluteus maximus and plantar flexors^{31,45} were not assessed. Assessment of other major muscle groups would have given a more complete understanding of physiological determinants of PPO. Furthermore, the VL is a major muscle in PPO production⁸ and assessment of VL muscle architecture has been used extensively.⁴⁶ However, these images only capture a superficial, two-dimensional representation of the muscle, which may not be representative of the whole muscle or other groups of muscles. A future study could examine multiple muscles involved in cycling), and

a range of sites within each muscle in order to further investigate the relationship of muscle architecture and PPO.

5 PERSPECTIVES

These new data showed quadriceps femoris muscle volume and pennation angle accounted for 76 and 11 %, respectively, of the variance of PPO in elite cyclists. These findings emphasise the importance of quadriceps muscle morphology for sprint cycling events and reinforce that cyclists and their coaches should be attentive to maximising these characteristics during their preparation and training. In addition, sprint cyclists achieved higher PPO than endurance cyclists, with T_{MAX} appearing to be the primary explanation for their greater PPO, which was likely because of their greater muscle morphology (Q_{VOL} , HAM_{VOL} and $P\theta_{VL}$). Additionally, future studies would need to measure changes in these morphological measures (throughout the course of a season) and whether these can predict changes in PPO and/or power- and torque-cadence relationships.

CONFLICT OF INTEREST

All authors declare that there is no conflict of interest. The authors alone are responsible for the content and writing of the manuscript.

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Table 1: Sprint cycling performance and physiological measures for thirty-five elite cyclists. Data are mean \pm SD, range and fold variability for minimum to maximum values.: Peak power output (PPO), optimal cadence (C_{OPT}), extrapolated maximum torque (T_{MAX}), extrapolated maximal cadence (C_{MAX}), quadriceps muscle volume (Q_{VOL}), hamstring muscle volume (HAM_{VOL}), pennation angle of the vastus lateralis (PA_{VL}) and fascicle length of vastus lateralis (FL_{VL}).

	Mean \pm SD	Range (max - min)	Fold variability
PPO (W)	1240 \pm 335	2025 - 775	x2.6
C_{OPT} (RPM)	131 \pm 12	161 - 112	x1.3
T_{MAX} (N.m)	175 \pm 37	236 - 117	x2.0
C_{MAX} (RPM)	267 \pm 31	362 - 221	x1.6
Q_{VOL} (cm³)	2268 \pm 582	3343 - 1347	x2.5
HAM_{VOL} (cm³)	804 \pm 206	1263 - 348	x3.6
$P\theta_{VL}$ (°)	15.6 \pm 2.0	18.8 - 11.7	x1.6
FL_{VL} (cm)	7.6 \pm 0.7	9.0 - 6.5	x1.4
GM_{ACT} (%)	102 \pm 16	128 – 62	x2.1
VL_{ACT} (%)	97 \pm 15	137 – 69	x2.0
BF_{ACT} (%)	95 \pm 11	120 - 75	x1.6

Table 2: Bivariate relationships (r) and associated coefficient of determination (R^2) for a range of physiological measures and the criterion measure (peak power output) in elite cyclists ($n = 35$). Knee extensor muscle volume (Q_{VOL}); knee flexor muscle volume (HAM_{VOL}); pennation angle ($P\theta_{VL}$); fascicle length (Fl); gluteus maximus (GM_{ACT}); vastus lateralis (VL_{ACT}) and bicep femoris (long head) (BF_{ACT}) muscle activation.

	r	R²	Relationship	p
Q_{VOL}	0.87	76%	Very large	<0.001
HAM_{VOL}	0.72	50%	Very large	<0.001
Pθ_{VL}	0.81	66%	Very large	<0.001
FL_{VL}	-0.15	2%	Small	0.933
GM_{ACT}	0.21	4%	Small	0.276
VL_{ACT}	-0.01	0%	Trivial	0.977
BF_{ACT}	-0.29	9%	Small	0.107

Table 3: Sprint cycling performance and physiological measures of sprint and endurance cyclists. Performance measures: peak power output (PPO), PPO normalised to body mass (PPO: Mass), optimal cadence (C_{OPT}), **extrapolated** maximal torque (T_{MAX}) and **extrapolated** maximal cadence (C_{MAX}). Physiological measures: Knee extensor muscle volume (Q_{VOL}); hamstring muscle volume (HAM_{VOL}); pennation angle ($P\theta_{VL}$); fascicle length of VL (FL_{VL}); gluteus maximus (GM_{ACT}); vastus lateralis (VL_{ACT}) and bicep femoris (long head) (BF_{ACT}) muscle activation. * denotes significantly higher than endurance ($p < 0.05$); ** denotes significantly higher than endurance ($p < 0.001$); † denotes significantly higher than sprint ($p < 0.05$).

	Sprint (n = 18)	Endurance (n = 17)
PPO (W)	1521 ± 186 **	942 ± 136

PPO: Mass (W/kg)	17.9 ± 1.9 **	13.6 ± 1.6
C_{OPT} (RPM)	136 ± 14 *	125 ± 7
T_{MAX} (N.m)	205 ± 18 **	143 ± 20
C_{MAX} (RPM)	282 ± 84 *	251 ± 18
Q_{VOL} (cm³)	2723 ± 420**	1786 ± 229
VL	961 ± 158**	429 ± 61
VM	662 ± 106**	503 ± 75
VI	762 ± 142**	662 ± 93
RF	337 ± 53**	232 ± 42
HAM_{VOL} (cm³)	994 ± 176**	655 ± 108
BF-s	141 ± 32*	115 ± 29
BF-l	255 ± 55**	171 ± 30
ST	262 ± 60**	170 ± 29
SM	285 ± 71**	199 ± 47
Pθ_{VL} (°)	17.1 ± 1.0**	14.8 ± 1.5
FL_{VL} (cm)	7.6 ± 0.6	7.5 ± 0.7
GM_{ACT} (%)	103 ± 16	101 ± 16
VL_{ACT} (%)	99 ± 18	96 ± 12
BF_{ACT} (%)	91 ± 9	98 ± 11†

Figure 1: Scatter plots showing the relationships between cycling peak power output (PPO) and different physiological measures: (a) quadriceps muscle volume (b) pennation angle, (c) fascicle length and (d) hamstrings muscle volume.

Figure 2: (a) Quadratic relationship of the power-cadence relationships and (b) inverse, linear torque-cadence relationship of sprint (red) and endurance (blue) cyclists. For (a) filled dots represent mean power output at each pre-determined cadence. Peak power output (PPO) and optimal cadence (C_{OPT}) are also highlighted. Significant differences were measured between PPO and C_{OPT} of both groups.

For (b), filled dots represent mean torque output at each pre-determined cadence. Extrapolated maximum torque (T_{MAX}) and (C_{MAX}) are shown for both groups. Significant differences were measured between T_{MAX} and C_{MAX} of both groups; Solid lines represent the mean relationship of measured cadences and dotted line represents extrapolation; shaded areas represent standard deviation at each pre-determined cadence which mirror the same relationships of respective relationships.

Figure 3: A comparison of proportional volume of individual muscles (% of whole muscle group) between sprint (n=17) and endurance cyclists (n=18) for (a) quadriceps femoris muscles: vastus intermedialis (VI), vastus medialis (VM), vastus lateralis (VL) and rectus femoris (RF) and (b) hamstrings muscles: long head bicep femoris (BF (LONG)), short head bicep femoris (BF (SHORT)), semitendinosus (ST) and semimembranosus (SM). Data presented as mean \pm SD; * denotes significant difference between proportion of muscle groups between sprinters and endurance riders.