The Journal of Sports Medicine and Physical Fitness 2025 April;65(4):493-506
DOI: 10.23736/S0022-4707.24.16429-8

ORIGINAL ARTICLE

EXERCISE PHYSIOLOGY AND BIOMECHANICS

Discipline-specific adaptation patterns in respiratory and lower limb musculotendinous structures: cyclists *vs.* basketball players

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ABSTRACT

BACKGROUND: This study aimed at assessing how chronic exposure to specific exercise training (high-intensity intervals *vs.* endurance), comparing experienced basketball-players (BP, N.=16), cyclists (CY, N.=16), and non-specifically trained individuals (CN, N.=16), influences the structural and functional characteristics of both lower limb and respiratory musculotendinous structures.
METHODS: Vastus lateralis, gastrocnemius lateralis, and medialis, diaphragm muscles, as well as patellar tendon and Achilles tendon, were assessed using B-mode ultrasonography. Maximal voluntary isometric and passive torque measurements were conducted in the knee-extensors and plantar-flexors. Additionally, a subset of participants (N.=10 for each group) underwent a fatigue-inducing exercise-till-exhaustion protocol, and the strength of lower limb and respiratory muscles was evaluated immediately before and after the trial.
RESULTS: Athletes had bigger and stronger musculotendinous structures and greater endurance to fatigue than CN ($P<0.05$). BP had bigger plantar-flexors and diaphragm, greater fascicles length, more explosive plantar-flexors and respiratory muscles and bigger tendons than CY ($P<0.05$). On the other hand, CY showed greater muscle pennation angle and greater endurance to fatigue for both, lower limb, and respiratory muscles ($P<0.05$).
CONCLUSIONS: The present study emphasizes that chronic and specific exercise training leads to distinctive adaptations, not only in lower limb musculotendinous structures but also in other components such as respiratory muscles.

(*Cite this article as*: Cesanelli L, Saveikis D, Conte D, Satkunskiene D. Discipline-specific adaptation patterns in respiratory and lower limb musculotendinous structures: cyclists *vs.* basketball players. J Sports Med Phys Fitness 2025;65:493-506. DOI: 10.23736/S0022-4707.24.16429-8)

Key words: Muscles; Tendons; Diaphragm; Muscle strength; Physical functional performance.

Exercise training is acknowledged as one of the strongest stimuli for inducing musculotendinous remodeling. Indeed, the mechanical trigger derived from exercise initiates molecular signaling cascades, that under chronic exposure, ultimately determine a specific musculotendinous phenotype, reflecting the specificity of the task being performed.[1, 2] In this sense, previous studies indicate that experienced athletes, chronically exposed to specific exercise modalities, exhibit unique muscle-tendon unit (MTU) features.[3-6] For instance, prolonged engagement in weight-bearing activities compared, for example, to water sports, has been associated with larger and stiffer lower limb tendons.[5] In general, greater adaptive responses in lower limb musculotendinous structures have been reported in individuals subjected to repetitive loads (*e.g.*, runners) or intermittent loads (*e.g.*, volleyball or basketball players) compared to those in sports that do not involve such stimuli or in sedentary individuals.[7] Furthermore, exercises involving repeated stretch-shortening cycles primarily promote increases in fascicle length, while athletes engaged in sports with predominantly concentric work tend to develop greater muscle pennation angle.[3, 4] The different characteristics of the exercise, encompassing factors like the type of contractile work, acting stresses, and operating length of the MTU, has been then hypothesized to contribute to the observed differences in musculotendinous properties such as architectural features, passive mechanical properties, functional capacity, and fatiguability among different athletes.[3-6, 8] Moreover, previous studies indicate that adaptive changes extend beyond primary load-bearing musculotendinous structures engaged in specific exercise tasks, to include structures supporting them, such as the respiratory muscles.[9-15] For example, in sports where contraction of ventilatory muscles, such as the diaphragm, is essential for generating intra-abdominal pressure to support exercise execution, greater ventilatory muscle strength and increased diaphragm size have been reported.[11] Indeed, the type of sport, and the exercise demands placed on the musculoskeletal system, significantly influence the physiological adaptation of the respiratory system. Athletes involved in disciplines requiring intense, high-intensity, and powerful movements tend then to display higher ventilatory capacity and hypertrophy of the respiratory muscles.[10-14] Moreover, musculotendinous size, strength and fatiguability of the lower limbs and respiratory muscles seem to be intertwined, where one can have an influence on the other one.[8, 16-18] These findings were corroborated not only in athletes but also in individuals with conditions such as sarcopenia, where exercise interventions targeting peripheral muscles had a positive impact on respiratory function.[18] In this context, it is evident that prolonged exposure to specific exercise training can lead to distinct musculotendinous adaptations in both the structures responsible for generating the movement and the accessory ones exerting a supportive function to perform the activity. These adaptations align with the demands of exercise, ultimately enhancing physical performance. Experienced athletes dedicated to particular sports, and exposed to specific exercise modalities, provide a robust model for understanding the impact of chronic training on musculotendinous structure and function. Cycling training, characterized by endurance and concentric-dominant exercise, and basketball, marked by high-intensity training, impact forces, and continuous stretch-shortening cycles, highlight key differences in musculotendinous adaptations. Basketball requires short, powerful bouts of exercise, necessitating a rapid buildup of intra-abdominal pressure, leading to greater ventilatory muscle strength and increased diaphragm size.[4, 6] In contrast, cycling that stimulates predominantly respiratory muscle endurance, may well result in less hypertrophy and strength of these muscles compared to sports requiring intense, maximal efforts.[4, 6] Moreover, investigating how chronic exercise training may influence adaptations not only in load bearing (*i.e.*, lower limbs muscles and tendons) but also accessory (*i.e.*, the respiratory muscles) musculotendinous structures, enhances the importance of this experimental model. Indeed, exploring these aspects could elucidate the impact of the long-term exposition to different training modalities, and in turn, paving the way for implementing cross-training among different activities and sports disciplines.[19] Such approach could be valuable in targeting specific adaptations, whether in rehabilitation contexts or optimizing performance.[20] Therefore, this study aims to assess how chronic exposure to different forms of exercise (*i.e.*, high intensity intervals *vs.* endurance), comparing experienced basketball players and cyclists, and non-specifically trained individuals, influences the structural and functional characteristics of both lower limb and respiratory musculotendinous structures. Moreover, the hypothesis posits that basketball players, compared to cyclists, would exhibit larger musculotendinous structures, greater force production, but lower fatigue resistance in both lower limbs and respiratory muscles.

Materials and methods

Study design and participants

Forty-eight male participants, including 16 cyclists (CY), 16 basketball players (BP), and 16 control subjects (CN) voluntarily participated in this cross-sectional study (Table I). To be included, the athletes had to fulfil at least two of

TABLE I.—*Characteristics of the three groups.*

Variables	CY	BP	CN	F	P	η^2
Age (years)	24±3.9	22±3.2	24±4.1	2.127	0.105	0.097 (S)
Body mass (kg)	81±6.9	87±10.8	81±8.5	2.463	0.097	0.111 (S)
Height (cm)	184±3.4[a]	193±5.4[c]	183±7.1	16.517	<0.001	0.423 (L)

CY: cyclists; BP: basketball players; CN: control.
[a]$P<0.05$ CY *vs*. BP; [c]$P<0.05$ BP *vs*. CN.

the “highly trained athlete” criteria described by McKay *et al.*[21] In addition, the exclusion criteria included self-reports of knee injuries that impeded maximal muscle contraction, a history of anterior knee pain, Osgood-Schlatter disease, cardiovascular disorders, medical history of diabetes, respiratory and neuromuscular diseases, or disclosure of drug abuse. The CY group comprised athletes from two continental UCI teams and one amateur national-level team, boasting an average of 11.0±5.3 years of experience. The cyclists covered an average distance of 13,932±5,811 km per year, equating to 9 to 15 hours of training per week. Differently, the BP group consisted of players competing within a first-league team engaged in international competitions and a second-league team playing only at national level. Athletes in the BP participants had an average experience of 8.0±5.1 years and trained for 8-12 hours per week. CN participants were sourced from the local community in Lithuania, with some of them being sport science students at a public Lithuanian university. To the best of author’s knowledge, no previous studies with a similar design and investigating similar dependent variables have reported effect sizes, preventing us from precisely estimating sample sizes through power analysis. Consequently, we opted for a convenient sample of 16 participants per group, aligning with previous investigations involving similar populations and with similar research aims.[4, 6] Participants were provided with detailed information about the experimental procedures, purposes, as well as the associated risks and benefits before signing a written informed consent. The research protocol adhered to the Declaration of Helsinki and was approved by the Ethics Committee of the Lithuanian Sports University (MNL-SVA(M)-2023-556).

Testing procedures

All participants were instructed to refrain from vigorous physical activity for 24 hours prior to the testing session and to maintain their regular diet. To ensure consistency, the same group of investigators conducted all measurements. The testing protocol began with the collection of anthropometric measurements, including body mass using a Tanita-305 body-fat analyzer (Tanita Corp), skinfold thickness measurements (GIMA Skinfold Caliper, GIMA S.p.A.), and body girths (ROLLFIX® DIA Ø, Hoechstmass Balzer GMBH, Sulzbach, Germany), following the guidelines from the International Society for the Advancement of Kinanthropometry (ISAK).[22] B-mode ultrasound (Telemed, Vilnius, Lithuania) was then used to examine the vastus lateralis (VL), gastrocnemius lateralis (GL), gastrocnemius medialis (GM), diaphragm (DIA) muscles, patellar tendon (PT), and Achilles tendon (AT). Following a standard warm-up on a stationary bike, participants underwent a passive resistive protocol test and a maximal voluntary isometric contraction test for knee extensors and plantar flexors using a calibrated Biodex System 4 dynamometer (Biodex Medical Systems, Shirley, NY, USA). For the recording of muscle activity, self-adhesive preamplified electrodes (F3010 FIAB) were attached to shaved, abraded, and cleaned skin regions over the rectus femoris, vastus lateralis, gastrocnemius lateralis and soleus muscles, following SENIAM recommendations,[23, 24] with ground electrodes fixed over the patella and malleolus. Surface electromyographic (sEMG) signals were sampled at 1000 Hz and synchronized with the dynamometer through a Biopac 12-bit-to-digital converter system (EL254S; Biopac Systems, Inc., Goleta, CA, USA) and AcqKnowledge software (version 4.1; Biopac Systems). Athletes then underwent respiratory function assessments, including spirometry procedures (MetaLyzer 3B; Cortex Biophysik, Leipzig, Germany) and a maximal inspiratory (MIP) and expiratory (MEP) pressure test (RPCheck; MD Diagnostics Ltd, Kent, UK). A subset of participants (CY, N.=10; BP, N.=10; CN, N.=10) performed a fatigue protocol on a separate day. The fatigue protocol began with an incremental ramp test on a cycle ergometer (Lode Excalibur Sport, Lode BV; Groningen, the Netherlands) to measure peak oxygen consumption ($\dot{V}O_{2peak}$) (MetaLyzer 3B; Cortex Biophysik), followed by an exercise to exhaustion session. Respiratory function and maximal voluntary contraction tests were conducted again immediately (5 minutes) before and after the time trial protocol. A summary of the study procedures is provided in Figure 1.

Musculoskeletal ultrasound imaging

Images of the VL, GL, GM and DIA muscles, PT and AT were obtained using a grayscale B-mode ultrasonography linear array transducer (10- to 15-MHz transducer, Echoblaster 128, UAB; Telemed). The settings of the ultrasound system were standardized (kept identical) for all participants and recorded using EchoWave II video-based software (Telemed), following the recommendations of the European Society of Muscoloskeletal Radiology.[5] The probe was positioned at 70% of the femoral length from the knee joint space to the greater trochanter on the VL, at the midpoint of the GL and GM muscle belly, and between the 7th and 10th intercostal space in the mid-axillary line for the DIA, where the coronal view of the right hemidiaphragm was identified (Figure 2). Additionally, images of PT and AT were collected, with the knee angle fixed at

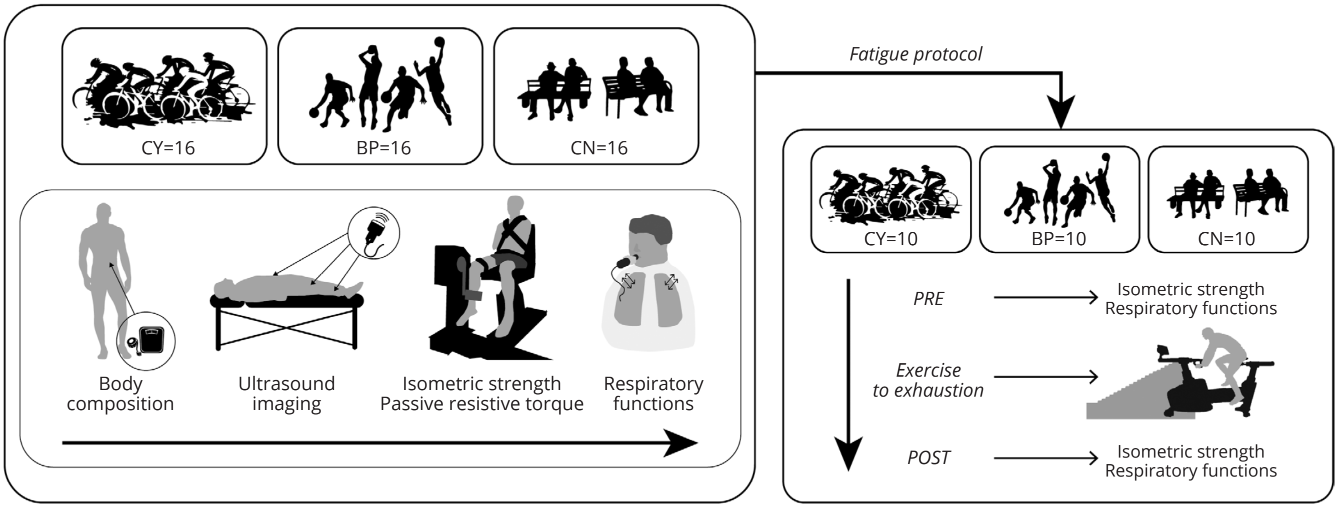

Figure 1.—Schematic representation of the study procedures: A) between groups comparison testing procedures; and B) fatigue protocol testing procedures.

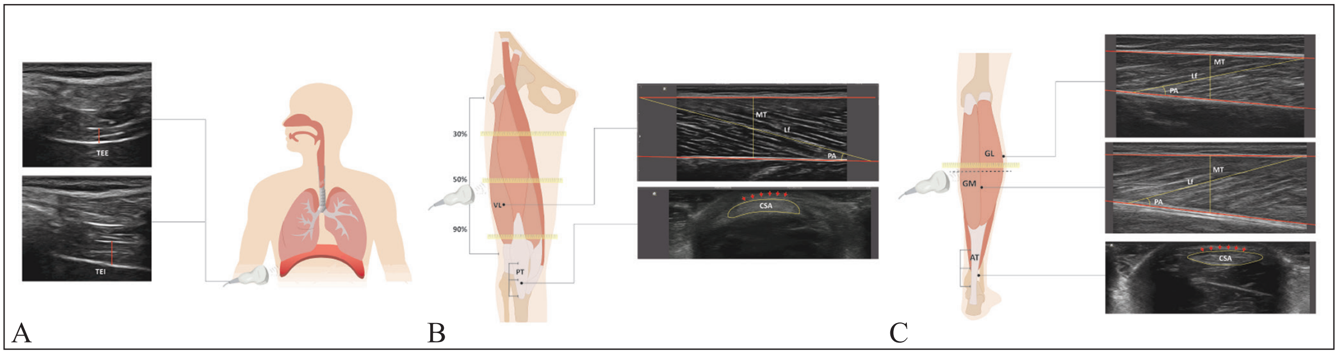

Figure 2.—Details of the ultrasound imaging procedures, featuring detailed views of various muscles and tendons. A-C) DIA muscle, VL muscle, PT, the GL and GM muscles, and the AT. Specifically, DIA ultrasound images were recorded at the zone of apposition during tidal and forced breaths, while VL, GL, GM, PT, and AT images were captured in longitudinal and transversal plane, respectively.
DIA: diaphragm; VL: vastus lateralis; PT: patellar tendon; GL: gastrocnemius lateralis; GM: gastrocnemius medialis; AT: Achilles tendon.

30° and the ankle joint angle at 90°, respectively. Images were collected in both longitudinal and transverse planes to measure the tendon thickness and cross-sectional area (CSA).[25] The images were later imported into ImageJ (version 1.46, US National Institutes of Health).[26] The Lf (fascicle length), PA (pennation angle), and MT (muscle thickness) were measured as the length of the fascicular path between the superficial and deep aponeuroses (when the transducer was not able to capture the whole length of the fascicles, the line of the fascicle was extrapolated beyond the frame of the image), the angle between the fascicular path and deep aponeurosis, and the distance between superficial and deep aponeurosis, respectively (Figure 2).[26] The PT and AT cross-sectional area (CSA_{PT} and CSA_{AT}) were analyzed at three anatomical landmarks, namely proximal, medial, and distal (Figure 2).[25] The CSA_{PT} and CSA_{AT} were measured by defining the tendon borders inferior to the first hyperechoic region between the subcutaneous tissue and the deep fascia layer in the transverse plane image. Videos of the DIA muscle were recorded with the subject in semi recumbent position and using the mid-axillary intercostal approach at the zone of apposition during tidal and forced breaths. Diaphragm thickness calculated at the functional residual capacity position ($MT_{DIA\mathit{ee}}$), at end of tidal breaths ($MT_{DIA\mathit{ei}}$) and of the maximal inspiratory efforts ($MT_{DIA\mathit{eimax}}$).[27, 28] Furthermore, the DIA thickening fraction TF=[(MT DIA end inspiration - MT DIA end espiration)/ MT DIA end espiration]×100 was calculated in both positions (tidal and maximal breaths).[27, 28] Three ultrasound images were recorded and analyzed for each participant in the different anatomical landmarks, and the measured values were averaged. MT, and CSA data were analyzed as absolute but they were also normalized by the participant's body mass raised to the power of ⅔[29] while the Lf was normalized by the participant's height raised to the power of ¼.[30] Intra-rater repeatability was calculated for all variables, and the values ranged from ICC=0.92 to ICC=0.97 and CV=2.09% to CV=4.37%.[4]

Passive resistive torque

The passive resistive torque protocol utilized a Biodex System 4 isokinetic dynamometer to measure torque resistance during passive stretch, capturing knee and ankle joint angle and angular velocity. For the knee extensors, participants lay supine with the pelvis and dominant leg straight, parallel to the floor, and securely strapped to minimize secondary movements. The opposite hip and knee were fixed at 90° flexion to limit pelvic and lumbar motion, aligning the knee axis with the dynamometer's rotational axis. With the participant fully relaxed, the dynamometer passively flexed the knee to 120° at 4° per second.[31] For the plantar flexors, participants lay prone with fully extended legs, secured by adjustable lap belts. The dominant ankle joint was strapped to a footplate connected to the dynamometer's lever arm, aligning the dynamometer's input axis with the ankle joint's rotation axis. In this configuration, and with the participant fully relaxed, the dynamometer passively flexed the ankle from 10° dorsiflexion to 30° plantarflexion at 4° per second.[32] The procedure was repeated three times with a 1-minute rest between sets. Passive stiffness of the MTU was calculated by dividing the change in passive torque by the change in ankle joint angle, expressed as Stiffness=ΔTorque/ΔROM, considering the slope of the curve between 80% and 100% of the total ROM and after correcting for gravity by using a custom-made Excel spreadsheet for calculations. Stiffness values were normalized by the athletes' body mass raised to the power of ⅔.[29] Passive resistive torque measurements were deemed valid only if sEMG activity was <5% of the maximum sEMG value (sEMGmax) recorded during the maximal voluntary contraction test.

Maximal voluntary isometric contractions

For the maximal voluntary isometric contraction test, athletes were securely strapped to a Biodex System 4 dynamometer using transverse belts. For the knee extensors, athletes were seated with 90° hip and knee flexion and aligned the femoral lateral epicondyle with the dynamometer axis. The dominant leg was fixed above the medial malleolus, minimizing joint movement and vertical displacement. For the plantar flexors, the athletes were lying prone, legs fully extended, with the thighs, the hips and the shoulders secured. The dominant ankle joint was securely strapped to a footplate connected to the level arm of the dynamometer. The input axis of the dynamometer was carefully adjusted to the axis of rotation of the ankle joint. Athletes were signaled to contract as fast and forcefully as possible upon supervisor instruction, receiving robust verbal encouragement during the test.[33] The athletes executed three maximal contractions, each lasting 2 seconds, with a 2-min recovery period between each contraction. For the knee extensors the contractions were performed, respectively at 80°, 90° and 100° knee joint angles, while for the plantar flexors at 90°, 80° and 70° ankle joint angles. The maximal voluntary torque (MVT) represented the peak isometric torque (Nm) during the contraction. Contractile rate of torque development (RTD) was calculated as average slope (Nm/s) in early (RTD_E) (0-100 ms) and late (RTD_L) (0-300 ms) phases.[33] Contraction onset was defined as knee extensor torque surpassing 2.5% of the baseline-to-peak torque difference.[33] The sampling frequency was set at 100 Hz by the dynamometer acquisition hardware. All values were normalized by the athletes' body mass raised to the power of ⅔.[29] The MVT and RTD data obtained from the best of the three repetitions of the highest voluntary contraction were analyzed. The sEMG signal of the VL, rectus femoris, GL and soleus muscles were recorded and the sEMGmax value quantified by calculating the root mean squared over a 0.05-sec period around the peak torque achieved during the maximal voluntary contractions. Additionally, 5% of the maximum sEMG value was determined to analyze muscle activity during the passive-resistive torque test, assessing the validity of the measurement.[34]

Respiratory function

Pulmonary function was assessed by conducting maximal flow volume loops[35] using a stationary spiroergometric device (MetaLyzer 3B, Cortex Biophysik), calibrated before each test according to the manufacturer's guidelines. Maximal static inspiratory (MIP) and expiratory (MEP) pressure, as well as the maximal rate of inspiratory (MRIPD) and expiratory (MREPD) pressure development – defined as the average slope between the onset of pressure (pressure surpassing 2.5% of the baseline-to-peak pressure difference) and its peak (cmH_2O/s) – were measured as indexes of respiratory muscle strength. This was done using a handheld mouth pressure meter fitted with a flanged mouthpiece (RPCheck, MD Diagnostics Ltd). Inspiratory and expiratory maneuvers were performed in a sitting upright position, initiated from residual volume and total lung capacity, respectively, and sustained for at least 1-sec. At least three valid attempts were recorded, and the maximum value from these attempts was used for subsequent analysis.

Fatigue protocol

Ten subjects from each group (CY, BP and CN) willingly participated in the fatigue protocol, with at least one week between the preceding testing procedures. To determine $\dot{V}O_{2peak}$, participants started with a standardized warm-up at 120 W for 5 minutes, followed by an incremental ramp test protocol (20 W/min) on an electromagnetically braked ergometer (Lode Excalibur Sport, Lode BV).[36, 37] Athletes had the freedom to choose their pedaling cadence, and the ergometer's configuration was customized to individual preferences and requirements. Real-time analysis of gas exchange was carried out using a stationary spiroergometric device operating in breath-by-breath mode (MetaLyzer 3B; Cortex Biophysik), with the gas analyzer calibrated before each session. Heart rate was continuously monitored via a chest strap heart rate monitor (Polar H10, Polar Electro). The test was considered concluded if the cadence dropped by more than 10 rpm from the average maintained throughout the test for a duration exceeding 10-sec, even with consistent verbal encouragement. The highest attained VO_2, quantified both absolutely and relative to the participant's body mass, was determined from each test as the highest consecutive 20-sec average value achieved during the test.[37] From the $\dot{V}O_{2peak}$ test, an exercise to exhaustion (ETE) protocol was planned, considering the 90% power output from $\dot{V}O_{2peak}$ power. To evaluate perceived exertion in the three groups after the ETE, all subjects completed the session-RPE scale (BORG-CR10) with values ranging from 0 (no exertion at all) to 10 (maximal exertion).[38] The ETE was performed at least 48 hours after the $\dot{V}O_{2peak}$ test. Five minutes before and after the ETE test, all athletes underwent respiratory function and maximal voluntary isometric contraction tests (for both knee extensors and plantar flexors) following the previously described procedures. Test completion times were monitored to ensure adherence to the fixed time window for all procedures. Delta (Δ) values were used for analyses, with an average value considered for the knee extensors and plantar flexors in isometric torque production from the different joint angles.

Statistical analysis

The data analysis was conducted using IBM SPSS Statistics (version 21.0; IBM Corp., Armonk, NY, USA), and graphical representations were created using GraphPad Prism (version 7.0; GraphPad Software, San Diego, CA, USA). Descriptive statistics (mean ± standard deviation [SD]) were calculated for each variable, and the normality of the sample distribution was assessed using the Shapiro-Wilk Test. Accordingly, a one-way analysis of variance (ANOVA) was carried out to determine if there were significant differences in the mean values of all tested variables among the three groups (CY, BP, and CN). In the event of a significant between-group effect, a Tukey-Corrected *post-hoc* Test was employed to evaluate the statistical significance of differences between mean values across the three groups. Furthermore, a 2×3 Mixed ANOVA was employed to evaluate the effect of the fatigue protocol (pre to post; time effect) between the three groups (CY, BP, and CN; group effect). Effect size was determined based on Cohen's guidelines,[39] with standardized effect (η^2) being small (S) for $\eta^2>0.1$, medium (M) for $\eta^2>0.25$, and large (L) for $\eta^2>0.4$, and with the standardized mean difference (*d*) for the pairwise comparisons interpreted as trivial, <0.20; small, 0.20 to 0.59; moderate, 0.60 to 1.19; large, 1.20 to 1.99; and very large, ≥2.00.[40] The critical level of statistical significance was set at a α level of 0.05.

Results

The characteristics of the subjects are reported in Table I. Body mass and height were higher in BP compared to CY ($P<0.05$) (Table I). The body fat percentage and body girths did not differ significantly between CY and BP groups, while the CN group showed significantly greater body fat and reduced lower limb girths (Supplementary Digital Material 1: Supplementary Table I).

Musculotendinous morphology

Significant between-group differences were observed for VL architecture: MT_{VL} ($F=18.462$; $P<0.001$; $\eta^2=0.451$; L), Lf_{VL} ($F=9.776$; $P<0.001$; $\eta^2=0.303$; M) and PA_{VL} ($F=28.236$; $P<0.001$; $\eta^2=0.557$; L). More in detail, BP and CY exhibited greater values than CN ($P<0.001$; $d=1.44$; large) with CY showing greater PA than BP and CN ($P<0.001$; $d=1.65$; large) (Figure 3). Significant between-group differences were observed for GL and GM archi-

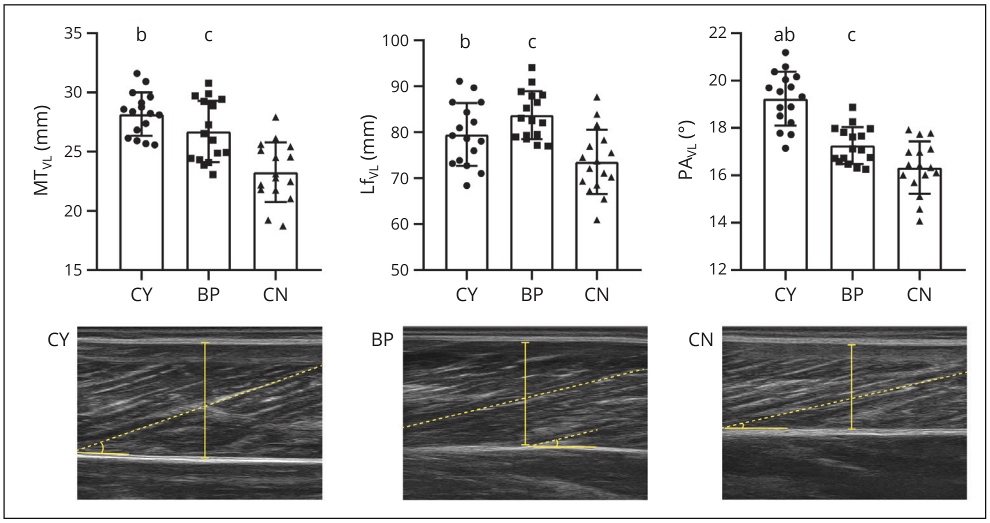

Figure 3.—Differences in MT$_{VL}$, Lf$_{VL}$ and PA$_{VL}$ between the three groups.
VL: vastus lateralis; MT$_{VL}$: VL muscle thickness; Lf$_{VL}$: fascicle length; PA$_{VL}$: pennation angle; CY: cyclists; BP: basketball players; CN: control.
^{a}P<0.05 CY *vs*. BP; ^{b}P<0.05 CY *vs*. CN; ^{c}P<0.05 BP *vs*. CN.

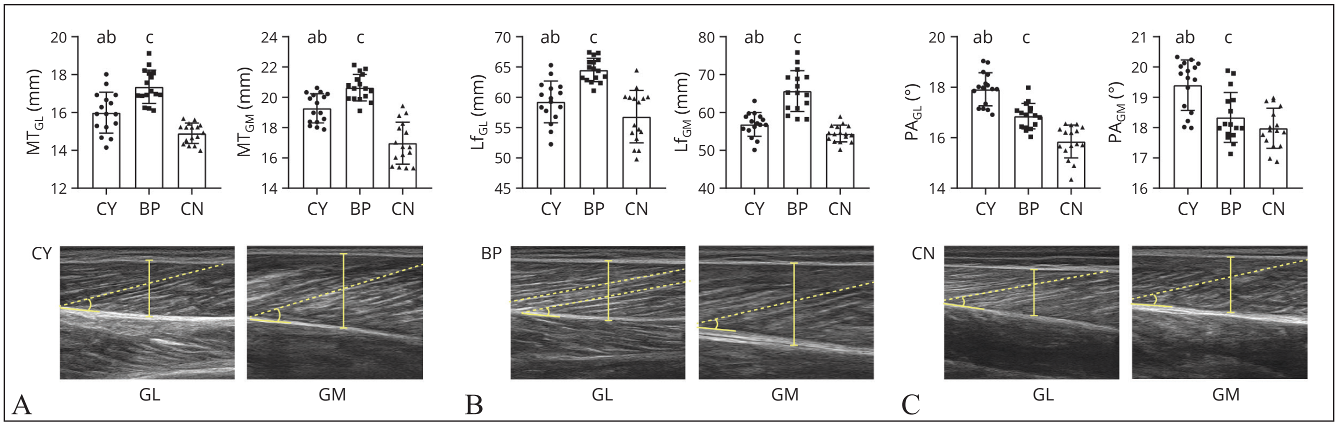

Figure 4.—A-C) Differences in GL and GM MT$_{VL}$, Lf$_{VL}$ and PA$_{VL}$ between the three groups.
MT$_{VL}$: VL muscle thickness; Lf$_{VL}$: fascicle length; PA$_{VL}$: pennation angle; GL: gastrocnemius lateralis; GM: gastrocnemius medialis; CY: cyclists; BP: basketball players; CN: control.
^{a}P<0.05 CY *vs*. BP; ^{b}P<0.05 CY *vs*. CN; ^{c}P<0.05 BP *vs*. CN.

tecture: MT$_{GL}$ (F=22.414; P<0.001; η^2=0.499; L), MT$_{GM}$ (F=23.866; P<0.001; η^2=0.515; L), Lf$_{GL}$ (F=13.009; P<0.001; η^2=0.366; M), Lf$_{GM}$ (F=17.282; P<0.001; η^2=0.434; L), PA$_{GL}$ (F=18.218; P<0.001; η^2=0.447; L), PA$_{GM}$ (F=5.340; *p*=0.008; η^2=0.192; S). Specifically, BP and CY demonstrated higher values than CN (P<0.001 *d*=1.55; large). BP had greater MT and Lf than CY (P<0.01; *d*=1.13; large), whereas for PA, CY exhibited higher values than BP (P<0.01; *d*=0.97; moderate) (Figure 4). Significant between-group differences were observed for CSA$_{PT}$ (F=14.392; P<0.001; η^2=0.390; M) and CSA$_{AT}$ (F=97.930; P<0.001; η^2=0.813; L). More in detail, BP and CY exhibited greater values than CN (P<0.01; *d*=1.09; large) with BP showing greater values than CY (P<0.01; *d*=0.85; moderate) (Figure 5). Significant between-group differences were observed for MT$_{DIA}$ at the three different respiratory stages analysed: functional residual capacity position (MT$_{DIAee}$: F=24.160; P<0.001; η^2=0.518; L), at end of tidal breaths (MT$_{DIAei}$: F=24.547; P<0.001; η^2=0.522; L) and of the maximal inspiratory efforts (MT$_{DIAeimax}$: F=24.547; P<0.001; η^2=0.505; L). Specifically, BP exhibited greater MT$_{DIA}$ at all stages compared to CY (P<0.01; *d*=0.69; moderate) and CN (P<0.001; *d*=2.07; very large), with CY showing higher values than CN (P<0.01; *d*=1.34; large) (Figure 6). The normalization of MT, Lf, and CSA for anthropometric measurements did not reveal any additional differences or alter any trends, thus confirming the results obtained with the absolute values.

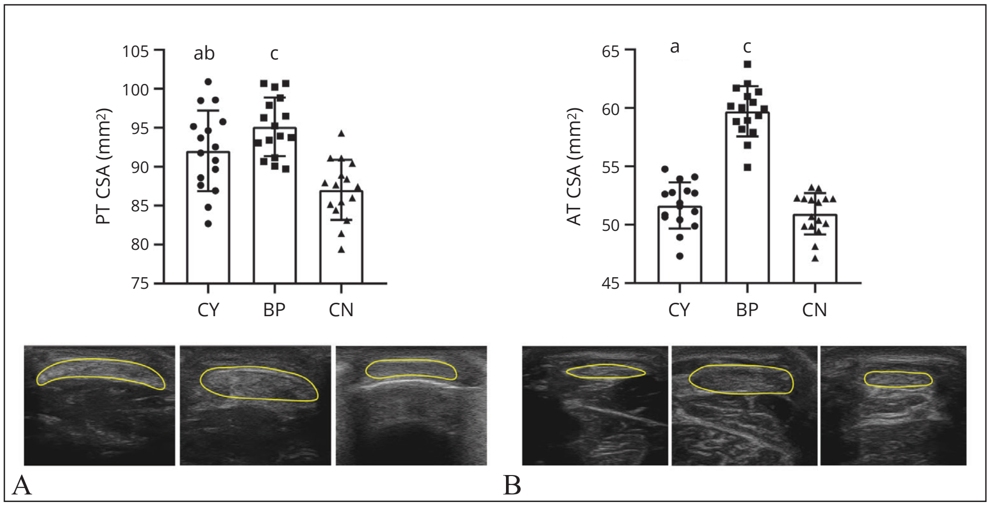

Figure 5.—A, B) Differences in PT and AT CSA between the three groups.
CSA: cross-sectional area; CY: cyclists; BP: basketball players; CN: control; PT: patellar tendon; AT: Achilles tendon.
[a]$P<0.05$ CY *vs*. BP; [b]$P<0.05$ CY *vs*. CN; [c]$P<0.05$ BP *vs*. CN.

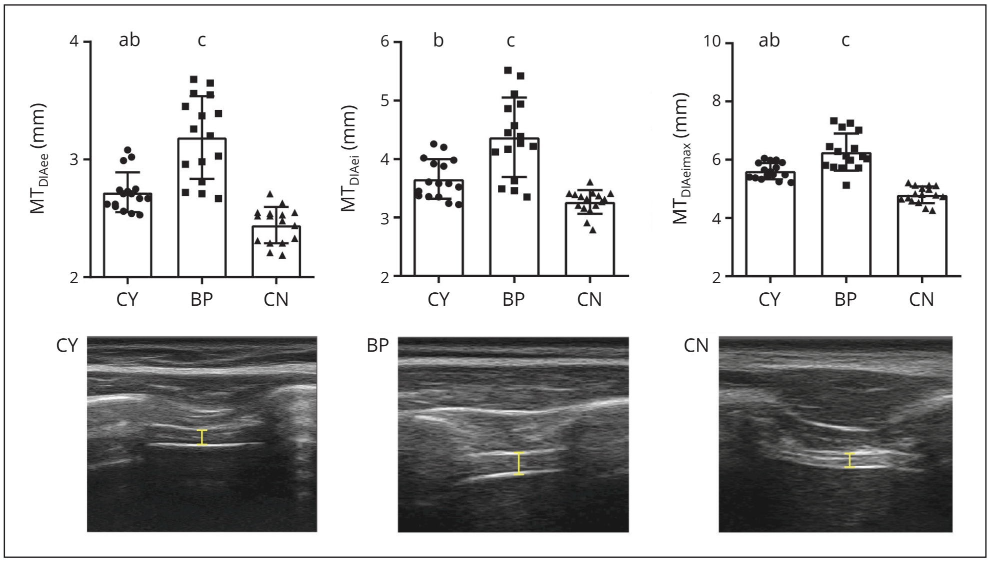

Figure 6.—Differences in diaphragm thickness between groups at three different respiratory stages: functional residual capacity position (MT_{DIAee}), at end of tidal breaths (MT_{DIAei}) and of the maximal inspiratory efforts ($MT_{DIAeimax}$).
CY: cyclists; BP: basketball players; CN: control.
[a]$P<0.05$ CY *vs*. BP; [b]$P<0.05$ CY *vs*. CN; [c]$P<0.05$ BP *vs*. CN.

Isometric strength and passive mechanical properties

Significant between-group differences emerged for knee extensors' isometric torque production capacity and passive stiffness ($P<0.05$) (Table II). Specifically, CY and BP exhibited greater values for isometric peak torque and rate of torque development than CN, both for absolute and normalized values ($P<0.01$) (Table II). Furthermore, isometric rate of torque development, particularly in the late phases of contraction, was higher in BP than CY ($P<0.05$). On the contrary, CY showed higher knee extensors' passive stiffness than BP and CN ($P<0.01$). Significant between-group differences emerged for plantar flexors' isometric torque production capacity and passive stiffness ($P<0.05$) (Table III). Specifically, CY and BP exhibited greater values for isometric peak torque and rate of torque development than CN, both for absolute and normalized values ($P<0.01$) (Table III). Furthermore, isometric rate of torque development was higher in BP than CY ($P<0.05$). BP showed higher plantar flexors' passive stiffness than CY and CN ($P<0.01$).

Respiratory function

Significant between-group differences emerged for part of the variables characterizing the respiratory function test ($P<0.05$) (Table IV). Specifically, CY and BP exhibited

TABLE II.—*Differences in knee extensors isometric contractile capacity and passive stiffness.*

Variables	CY	BP	CN	*F*	P	η^2
$\mathrm{KEPT}_{100^{\circ}}$ (Nm)	326±58^{b}	344±86^{c}	251±49	8.614	<0.001	0.277 (M)
$\mathrm{nKEPT}_{100^{\circ}}$ (Nm/kg$^{2/3}$)	6.0±0.9^{b}	5.9±1.1^{c}	4.7±1.1	7.763	0.001	0.257 (M)
$\mathrm{KEPT}_{90^{\circ}}$ (Nm)	365±72^{b}	367±93^{c}	282±61	6.333	0.004	0.220 (S)
$\mathrm{nKEPT}_{90^{\circ}}$ (Nm/kg$^{2/3}$)	6.8±1.1^{b}	6.3±1.1^{c}	5.3±1.5	5.588	0.007	0.199 (S)
$\mathrm{KEPT}_{80^{\circ}}$ (Nm)	354±85^{b}	336±88^{c}	274±68	4.353	0.019	0.162 (S)
$\mathrm{nKEPT}_{80^{\circ}}$ (Nm/kg$^{2/3}$)	6.6±1.4^{b}	5.7±1.1^{c}	5.2±1.8	3.519	0.038	0.135 (S)
$\mathrm{KERTD}_{E100^{\circ}}$ (Nm/s)	2022±247^{b}	2224±445^{c}	1562±214	17.587	<0.001	0.439 (L)
$\mathrm{nKERTD}_{E100^{\circ}}$ (Nm/s/kg$^{2/3}$)	37.7±3.2^{b}	38.5±6.3^{c}	29.4±6.2	13.693	<0.001	0.378 (M)
$\mathrm{KERTD}_{L100^{\circ}}$ (Nm/s)	1299±190^{ab}	1647±349^{c}	1002±154	27.421	<0.001	0.549 (L)
$\mathrm{nKERTD}_{L100}{}^{\circ}$ (Nm/s/kg$^{2/3}$)	24.2±2.9^{b}	28.5±5.0^{c}	18.8±4.2	7.630	0.001	0.253 (M)
$\mathrm{KERTD}_{E90^{\circ}}$ (Nm/s)	2170±351^{b}	2255±351^{c}	1675±286	10.902	<0.001	0.326 (M)
$\mathrm{nKERTD}_{E90^{\circ}}$ (Nm/s/kg$^{2/3}$)	40.4±4.9^{b}	39.1±6.7^{c}	31.6±8.4	5.705	0.006	0.202 (S)
$\mathrm{KERTD}_{L90^{\circ}}$ (Nm/s)	1393±206^{ab}	1664±369^{c}	1075±169	20.06	<0.001	0.471 (L)
$\mathrm{nKERTD}_{L90^{\circ}}$ (Nm/s/kg$^{2/3}$)	25.9±2.8^{b}	28.7±4.8^{c}	20.3±4.8	21.585	<0.001	0.490 (L)
$\mathrm{KERTD}_{E80^{\circ}}$ (Nm/s)	2138±425^{b}	2202±501^{c}	1650±336	18.179	0.001	0.263 (M)
$\mathrm{nKERTD}_{E80^{\circ}}$ (Nm/s/kg$^{2/3}$)	39.7±6.2^{b}	38.0±6.6^{c}	31.2±9.4	15.509	<0.001	0.423 (L)
$\mathrm{KERTD}_{L80^{\circ}}$ (Nm/s)	1348±223^{ab}	1625±378^{c}	1040±179	10.054	<0.001	0.447 (L)
$\mathrm{nKERTD}_{L80^{\circ}}$ (Nm/s/kg$^{2/3}$)	25.1±3.2^{b}	28.0±4.6^{c}	19.6±5.3	14.322	<0.001	0.389 (M)
KEstiffness (Nm/deg)	0.80±0.10^{ab}	0.58±0.06^{c}	0.49±0.17	74.926	<0.001	0.769 (L)
nKEstiffness (Nm/deg/kg$^{2/3}$)	0.015±0.001^{ab}	0.009±0.001	0.008±0.001	66.322	<0.001	0.747 (L)

CY: cyclists; BP: basketball players; CN: control; KE: knee extensors; PT: peak torque; RTD: rate of torque development; E: early; L: late; N.: normalized.
^{a}P<0.05 CY *vs*. BP; ^{b}P<0.05 CY *vs*. CN; ^{c}P<0.05 BP *vs*. CN.

TABLE III.—*Differences in plantar flexors isometric contractile capacity and passive stiffness.*

Variables	CY	BP	CN	*F*	P	η^2
$\mathrm{PFPT}_{0^{\circ}}$ (Nm)	142±34^{b}	163±39^{c}	109±27	10.054	<0.001	0.309 (M)
$\mathrm{nPFPT}_{0^{\circ}}$ (Nm/kg$^{2/3}$)	2.6±0.6^{b}	2.8±0.6^{c}	2.1±0.6	6.614	0.003	0.227 (S)
$\mathrm{PFPT}_{10^{\circ}}$ (Nm)	172±43^{b}	191±47^{c}	133±54	7.740	0.001	0.256 (M)
$\mathrm{nPFPT}_{10^{\circ}}$ (Nm/kg$^{2/3}$)	3.2±0.8^{b}	3.3±0.6^{c}	2.5±0.8	4.846	0.012	0.177 (S)
$\mathrm{PFPT}_{20^{\circ}}$ (Nm)	213±56^{b}	229±33^{c}	164±46	5.617	0.007	0.200 (S)
$\mathrm{nPFPT}_{20^{\circ}}$ (Nm/kg$^{2/3}$)	3.9±1.0^{b}	3.9±0.8^{c}	2.5±0.8	3.721	0.032	0.142 (S)
$\mathrm{PFRTD}_{E0^{\circ}}$ (Nm/s)	694±144^{ab}	981±232^{c}	536±114	27.741	<0.001	0.552 (L)
$\mathrm{nPFRTD}_{E0^{\circ}}$ (Nm/s/kg$^{2/3}$)	12.9±2.3^{ab}	16.9±3.2^{c}	10.1±2.7	24.548	<0.001	0.522 (L)
$\mathrm{PFRTD}_{L0^{\circ}}$ (Nm/s)	445±116^{ab}	773±217^{c}	343±90	35.111	<0.001	0.609 (L)
$\mathrm{nPFRTD}_{L0^{\circ}}$ (Nm/s/kg$^{2/3}$)	13.7±2.6^{ab}	17.2±3.3^{c}	10.7±2.8	19.399	<0.001	0.463 (L)
$\mathrm{PFRTD}_{E10^{\circ}}$ (Nm/s)	738±154^{ab}	998±241^{c}	570±123	22.879	<0.001	0.504 (L)
$\mathrm{Npfrtd}_{E10^{\circ}}$ (Nm/s/kg$^{2/3}$)	14.7±3.1^{ab}	17.8±3.3^{c}	11.5±3.2	15.468	<0.001	0.407 (L)
$\mathrm{PFRTD}_{L10^{\circ}}$ (Nm/s)	466±123^{ab}	805±27^{c}	360±96	35.400	<0.001	0.611(L)
$\mathrm{nPFRTD}_{L10^{\circ}}$ (Nm/s/kg$^{2/3}$)	8.3±3.0^{ab}	13.3±3.0^{c}	6.4±1.8	35.851	<0.001	0.614 (L)
$\mathrm{PFRTD}_{E20^{\circ}}$ (Nm/s)	789±178^{ab}	1033±58^{c}	610±142	18.769	<0.001	0.455 (L)
$\mathrm{nPFRTD}_{E20^{\circ}}$ (Nm/s/kg$^{2/3}$)	8.7±2.2^{ab}	13.8±2.9^{c}	6.7±1.9	36.705	<0.001	0.620 (L)
$\mathrm{PFRTD}_{L20^{\circ}}$ (Nm/s)	504±120^{ab}	830±224^{c}	389±94	33.878	<0.001	0.601 (L)
$\mathrm{nPFRTD}_{L20^{\circ}}$ (Nm/s/kg$^{2/3}$)	9.4±2.1^{ab}	14.3±3.0^{c}	7.3±1.9	35.165	<0.001	0.610 (L)
PFstiffness (Nm/deg)	1.56±0.32^{ab}	2.18±0.40^{c}	1.11±0.22	53.549	<0.001	0.704 (L)
nPFstiffness (Nm/deg/kg$^{2/3}$)	0.029±0.006^{ab}	0.039±0.004^{c}	0.020±0.004	55.565	<0.001	0.712 (L)

CY: cyclists; BP: basketball players; CN: control; PF: plantar flexors; PT: peak torque; RTD: rate of torque development; E: early; L: late; N.: normalized.
^{a}P<0.05 CY *vs*. BP; ^{b}P<0.05 CY *vs*. CN; ^{c}P<0.05 BP *vs*. CN.

greater values for FVC, FEV1, PIF than CN (P<0.05), without differences between the two athlete groups (Table IV). BP showed greater MIP, MEP, RIPD, and REPD than CY and CN (P<0.05), significance of differences that, however, disappeared once data were normalized for subjects' body mass (Table IV).

Fatigue response

Significant between-group differences emerged for VO_{2peak} values, peak power output and ETE completion time (P<0.001), with CY showing greater values than BP and CN (P<0.001), and BP greater than CN (P<0.001) (Supple-

TABLE IV.—*Differences in respiratory function between the three groups.*

Variables	CY	BP	CN	*F*	P	η^2
FVC (L)	6.7±1.0^{b}	6.5±0.9^{c}	5.6±0.9	4.703	0.014	0.173 (S)
FEV1 (L)	5.4±0.8^{b}	5.3±0.7^{c}	4.7±0.8	4.203	0.021	0.157 (S)
FEV1/FVC (%)	82.4±2.1	82.5±2.2	82.6±2.8	1.324	0.276	0.056 (S)
FIVC (L)	5.0±0.4	5.0±0.3	4.6±0.8	2.964	0.062	0.116 (S)
PIF (L/s)	7.2±1.1^{b}	7.6±1.1^{c}	6.6±1.1	3.229	0.049	0.126 (S)
MIP (cmH20)	149.0±14.8^{ab}	157.3±19.9^{c}	139.3±8.5	5.881	0.005	0.207 (S)
nMIP (cmH20/kg$^{2/3}$)	2.8±0.3	2.8±0.6	2.6±0.3	0.795	0.458	0.034 (S)
RIPD (cmH20/s)	1441.7±261.4^{ab}	1749.7±221.5^{c}	1344.7±199.3	17.132	<0.001	0.432 (L)
nRIPD (cmH20/s/kg$^{2/3}$)	27.3±5.5^{b}	31.7±6.9^{c}	25.0±3.2	6.351	0.004	0.220 (S)
MEP (cmH20)	187.5±25.7^{ab}	208.6±15.5^{c}	175.1±18.3	11.18	<0.001	0.332 (L)
nMEP (cmH20/kg$^{2/3}$)	3.6±0.5^{b}	3.7±0.5^{c}	3.2±0.4	3.147	0.044	0.122 (S)
REPD (cmH20/s)	2226.7±260.6^{ab}	2530.5±199.1^{c}	2088.9±252.6	14.293	<0.001	0.388 (L)
nREPD(cmH20/s/kg$^{2/3}$)	42.8±6.9^{b}	45.4±6.5^{c}	39.0±5.2	3.022	0.046	0.118 (S)

CY: cyclists; BP: basketball players; CN: control; FVC: forced vital capacity; FEV1: forced expiratory volume in the first second; FIVC: forced inspiratory vital capacity; PIF: peak inspiratory flow; MIP: maximal inspiratory pressure; MEP: maximal expiratory pressure; RIPD: rate of inspiratory pressure development; REPD: rate of expiratory pressure development; N.: normalized.
^{a}P<0.05 CY *vs*. BP; ^{b}P<0.05 CY *vs*. CN; ^{c}P<0.05 BP *vs*. CN.

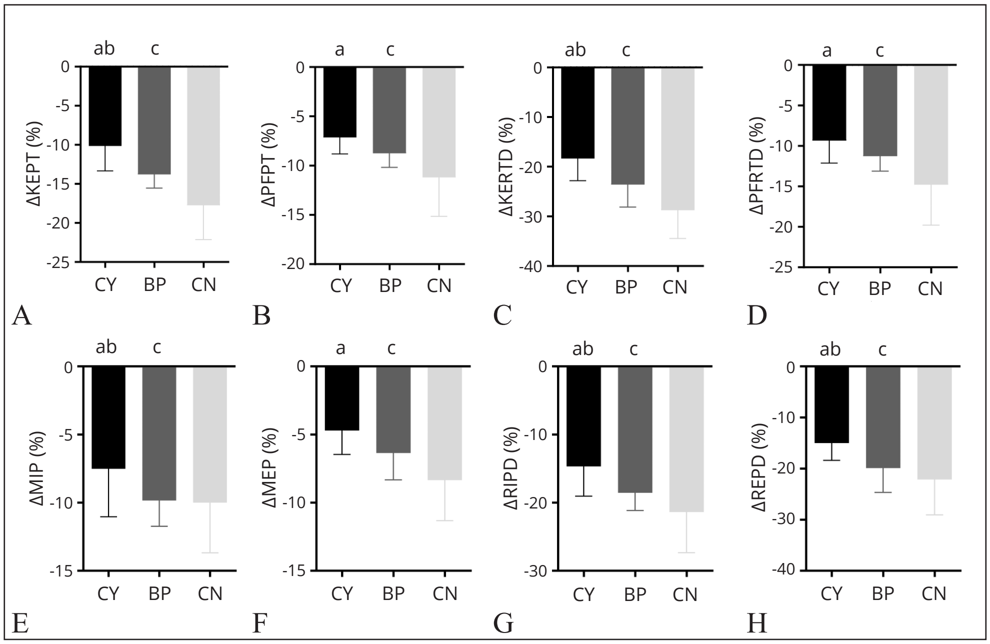

Figure 7.—Differences in pre- to post-Δ values (%) between the three groups: A-D) isometric contractile capacity; and E-H) respiratory muscles strength indicators.
CY: cyclists; BP: basketball players; CN: control; KE: knee extensors; PF: plantar flexors; PT: peak torque; RTD: rate of torque development; MIP: maximal inspiratory pressure; MEP: maximal expiratory pressure; RIPD: rate of inspiratory pressure development; REPD: rate of expiratory pressure development; N.: normalized.
^{a}P<0.05 CY *vs*. BP; ^{b}P<0.05 CY *vs*. CN; ^{c}P<0.05 BP *vs*. CN.

mentary Digital Material 2: Supplementary Table II). Significant time (P<0.001) and time × group effects (P<0.05) emerged for the knee extensors and plantar flexors' isometric peak torque and rate of torque development. Similarly, significant time (P<0.001) and time x group effects (P<0.05) emerged for the respiratory muscles' strength markers. More in detail, CN experienced greater reduction in isometric contractile capacity and respiratory muscles strength induced by fatigue compared to the athlete groups. Furthermore, BP were characterized by a higher fatigue effect compared to CY (P<0.05) (Figure 7).

Discussion

The study highlights how chronic exposure to specific exercise training (*i.e.*, high intensity intervals *vs*. endurance), such as cycling and basketball, induces distinct adaptive signatures not only in the lower limb musculotendinous structures, directly involved in the movements, but also in structures such as the respiratory muscles. BP displayed larger muscles and tendons, longer fascicles but lower PA, and quicker muscle contractions, albeit with lower fatigue resistance than CY.

Musculotendinous structural adaptive signatures

The findings reveal that muscles of CY generally exhibited greater PA, while BP had longer Lf. Conversely, BP showed thicker DIA and plantar flexor muscles compared to CY. Overall, athletes, at all anatomical landmarks examined, displayed larger muscles than CN, either through increased PA or increased Lf. Moreover, BP had larger tendons than CY, particularly the AT, with athletes exhibiting greater values than CN. Considering these findings and aligning with previous hypotheses,[41] these differences may delineate the impact of distinct type of chronic training, based on whether cycling or basketball serves as the primary form of exercise. Thus, these alterations could be attributed to elements and specificity of the exercise training, including the intensity of the mechanical stimulus, presence or absence of impact forces acting on the MTUs, and the lengths at which muscles operate in the two distinct disciplines. The observation that BP's muscles may undergo more eccentric work while CY may predominantly engage in concentric contractions further contributes to these differences.[4, 42] In this context, a muscle's architecture is believed to mirror its *in-vivo* function, encompassing exercise-specific operating lengths and the characteristics of the applied load.[4, 42, 43] Concentric loading is suggested to induce a greater increase in PA compared to eccentric contractions, possibly resulting in hypertrophy-induced parallel addition of sarcomeres rather than an increase in Lf observed with eccentric loading.[42] From a functional standpoint, BP may benefit from increased Lf, enhancing velocity during identical tendon excursions and potentially improving sprinting and jumping performances. By contrast, in CY, a greater PA, indicative of the packing of more sarcomeres in parallel, is linked to greater force output during contractions against high loads. The use of higher loads (gear combinations) and the biomechanics of pedaling in cyclists may therefore account for the different muscle architecture observed in comparison with BP. These concepts are consistent with earlier findings, demonstrating that CY exhibit greater PA and lower fascicle length Lf compared to high-intensity athletes engaged in rapid movements characterized by stretch-shortening cycles and ground impact forces acting on the MTUs.[3, 4] The observed differences in PT and, more significantly, in AT dimensions, where BP displayed greater values than CY, further contribute to delineating this picture. The larger tendons observed in BP compared to CY may be attributed to the specific demands of basketball training, which often involves explosive movements, rapid changes in direction, and frequent jumps, all of which impose substantial stresses on the tendons.[5, 44, 45] These mechanical stimuli likely contribute to tendon hypertrophy as an adaptive response to the repetitive and intense loading during basketball activities, where adaptation depends not only on the extent of tendon damage incurred during exercise but also on the speed at which the tendon repairs itself. The degree of damage appears to be closely related to the amount of stretching imposed on the tendon structures and consequently, being constantly exposed to such stimulus may induce quicker and successful adaptations, manifesting in bigger and stiffer tendons.[46] Moreover, the finding that both CY and BP exhibited larger tendons than CN emphasizes the positive adaptations induced by exercise in various forms, not only in muscles but also in tendinous structures. This suggests that chronic exposure to different forms of exercise leads to specific structural adaptations within the MTU to meet the unique demands imposed by each type of activity. The exercise-specificity effect observed in this study extended beyond the primary muscles involved in the activity. Specifically, the DIA, recognized as the main respiratory muscle, exhibited greater thickness in BP compared to CY. This highlights how distinct exercise modalities not only impact the primary musculotendinous structures but also have discernible effects on accessory muscles involved in related physiological function, such as respiration. These findings align with previous studies that have investigated the structural and dimensional characteristics of respiratory muscles in both non-specifically trained individuals and athletes.[9-15] Moreover, they contribute to the broader understanding of the impact of exercise training, having respiratory muscles as not the main targeted muscle, on the structure and function of respiratory muscles.[11, 47, 48] Exercises involving the trunk, emphasizing maximal strength or explosive movements like yoga, strength training, or powerlifting, have demonstrated improvements in respiratory muscle function.[11, 48, 49] Engagement of trunk muscles in strength-based and explosive exercises leads to increased intra-abdominal and diaphragmatic pressure, inducing muscle fatigue and potentially providing sustained adaptive stimuli to the diaphragm.[50] These findings suggest that respiratory muscles are highly responsive to intra-thoracic pressure fluctuations during various exercise training modalities, including resistance, high-intensity, and endurance regimens. The recurrent exposure to high intra-abdominal pressures during explosive or strength exercises such as jumping, sprinting, and weightlifting, as experienced by BP, may offer a more potent stimulus compared to the effects of static endurance training (CY), providing a possible explanation for our results.

Musculotendinous functional adaptive signatures

Regarding muscle contractility, BP and CY did not exhibit differences in terms of maximal isometric strength. However, BP demonstrated greater explosive isometric strength than CY. Athletes, in general, displayed higher maximal and explosive strength compared to CN. Furthermore, CY had greater stiffness in the knee extensors' MTU than BP, while BP had greater stiffness in the plantar flexors' MTU, with athletes showing higher stiffness than CN. Differences in the contractility of primary muscles engaged in different sport disciplines and, consequently, distinct exercise training have been documented.[3, 4] In this context, it appears that the specificity of the task being performed dictates the structural adaptations of musculotendinous structures and, consequently, their functional characteristics. Indeed, from a functional standpoint, the architecture and the stiffness of the MTU is thought to influence the time course of force production by affecting the efficiency of force transmission from the muscle to the bone, and therefore its contractile characteristics.[43, 51] A stiffer MTU would theoretically transfer force to the bone more effectively, resulting in a shorter time to achieve a specific force level and a more efficient movement.[51] This suggests a potential role of chronic exposure to specific exercises with different muscle mechanics in influencing MTU passive stiffness. On one hand, activities like jumping, sprinting, and weightlifting, encountered by BP, potentially present a more robust stimulus compared to the impact of static endurance training (CY). This distinction could elucidate our findings, particularly concerning the plantar flexors MTUs. In the case of BP, these muscles serve as primary effectors in supporting dynamic tasks, whereas in CY, their role mainly revolves around stabilizing the ankle joint to facilitate proper force transmission to the pedals. Therefore, the differences in isometric contractile function and MTU passive stiffness could be attributed to the specific characteristics of the exercise training being performed. No significant differences were observed between CY and BP in terms of spirometry, but BP exhibited higher maximal and explosive strength in respiratory muscles compared to CY. Respiratory function indicators were superior in athletes compared to CN. Previous studies have suggested that exercise specificity plays a role also in establishing differences in respiratory function among athletes from different sport disciplines, with BP and high-intensity athletes generally showing greater pulmonary capacity than endurance athletes like CY.[13, 14] In this context, our results align with the observations emerged from lower limb muscles, both in terms of musculotendinous structural and functional features. BP exhibited significantly higher RIPD and REPD, indicating faster respiratory muscles than CY. This parallels the observations in lower limb muscles, where the RTD was also higher in BP than CY, aligning with the characteristics of their respective activities (*i.e.*, high intensity intervals, explosive jumps and sprints, resistance training *vs.* prolonged intervals and endurance prevalent training).

Fatigue response

Considering the impact of training specificity on musculotendinous structure and function, an additional research question was posed: does the adaptive response extend beyond maximal and explosive strength markers to include fatigue resistance? Given that BP exhibited greater explosive strength, and, in some measures, maximal strength compared to CY, the anticipation was that, on the opposite, CY would excel in fatigue resistance, both for lower limb and respiratory muscles. As expected, athletes demonstrated superior fatigue resistance compared to CN, with CY exhibiting greater endurance and lower levels of fatigue in both lower limb and respiratory muscles compared to BP. Prior research has highlighted a connection between fatigue in lower limb muscles and respiratory muscles during whole-body exercises.[8, 9, 18] Fatigue in one muscle group can potentially impact the other, suggesting an interdependence between them.

Limitations of the study

This study highlights how chronic exercise training influences not only primary musculotendinous structures but also accessory ones, such as the respiratory muscles. These findings suggest potential applications for cross-discipline training to target specific adaptations that may be difficult to achieve within a single discipline, or to address gaps in the physical performance profiles of athletes from specific sports. Moreover, during rehabilitation after an injury, monitoring the effects of inactivity or detraining on both the injury site and supporting structures, such as the respiratory muscles, may reveal "hidden" consequences. Addressing these factors promptly could facilitate and expedite the recovery process. Additionally, our study suggests that incorporating various exercises interchangeably could not only optimize performance but also help bridge structural and functional deficits caused by injuries or periods of forced inactivity. While the present study has notable strengths, there are acknowledged limitations. Future studies involving larger sample sizes, incorporating a broader array of sports, could enhance the robustness of conclusions. Morphological data collection at rest in an extended leg position might be seen as a limitation, as it may not fully capture the dynamic nature of muscle behavior during activity. Future research could employ dynamic ultrasound analysis for a more nuanced exploration of discipline-specific differences in muscle architecture during contractions. Additionally, the fatigue protocol's focus on voluntary activation as a marker of fatigue may not fully capture peripheral fatigue. Another crucial aspect is the impact of the exercise specificity experienced by the athletes. BP might be more accustomed to generating maximal contractions due to their frequent resistance training. In contrast, CY might have an advantage in the fatigue protocol, given its resemblance to their regular training. Although ETE protocols on bike ergometers are widely used in labs to induce fatigue, future studies could explore various fatigue protocols to further investigate and expand upon our findings. Interpretation of our results should also consider the oversimplification inherent in using knee extensors and plantar flexors as surrogates for overall leg muscle fatigue in a complex motor task. These limitations provide avenues for future research to expand and refine our findings.

Conclusions

Our study underscores the significance of exercise-specific musculotendinous adaptations resulting from chronic exposure to distinct training regimens, specifically comparing basketball players and cyclists, alongside non-specifically trained individuals. Consistent with our aims, we found that basketball players exhibited larger musculotendinous structures, greater force production, and lower fatigue resistance in both lower limbs and respiratory muscles compared to cyclists. These adaptations were observed across three key areas: I) muscle and tendon dimensions and architectural changes, II) mechanical properties and strength, and III) fatigue resistance. The intrinsic differences in the exercises performed by the athletes, including high-intensity intervals in basketball *versus* endurance cycling, highlight the crucial role of discipline-specific mechanical stimuli in shaping the structural and functional adaptations of both primary and accessory musculotendinous structures.

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Conflicts of interest
The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Authors' contributions
Leonardo Cesanelli and Danguole Satkunskiene have given substantial contributions to the study conception and design; Daniele Conte and Deividas Saveikis contributed to the data acquisition, analysis, and interpretation; all authors have contributed to the manuscript draft, Daniele Conte and Danguole Satkunskiene revised it critically. All authors read and approved the final version of the manuscript.

History
Article first published online: October 28, 2024. - Manuscript accepted: September 24, 2024. - Manuscript revised: September 12, 2024. - Manuscript received: July 2, 2024.

Supplementary data
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