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RESEARCH ARTICLE

Lower calf raise efficiency in obesity is partially related to higher triceps surae MTU passive stiffness, hysteresis, and reduced relative strength

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Abstract

The objective of this study was to assess the efficiency of calf raise exercise in individuals with obesity, and to what extent this is related to the structural and mechanical properties of the triceps surae muscle-tendon-unit (MTU). In 22 obese (body mass index; BMI, 32.2 ± 1.5 kg/m^2) and 22 nonobese (BMI, 23.3 ± 1.5 kg/m^2) men we measured anthropometric parameters, gas exchange, heart rate, and rating of perceived exertion in sitting, standing, and during a self-paced 30-s calf raise exercise. Maximal voluntary isometric contraction of the plantar flexor muscles and passive resistive torque of the triceps surae MTUs were measured using an isokinetic dynamometer. B-mode ultrasound imaging of plantar flexor muscles and Achilles tendon was also performed. Individuals with obesity exhibited a greater metabolic energy cost during standing and calf raise exercise, and a lower exercise efficiency and ankle mobility ($P < 0.05$). Plantar flexor MTUs stiffness and hysteresis were greater, whereas gastrocnemius-medialis tendon strain was lower in subjects with obesity compared with controls ($P < 0.05$). There was a negative correlation between calf raise exercise net efficiency, plantar flexor MTUs hysteresis ($r = 0.38$; $P < 0.05$), and the body mass by maximal voluntary isometric torque ratio ($r = 0.41$; $P < 0.05$). These observations indicate that increased musculotendinous stiffness and hysteresis, together with a lower strength to body mass ratio, are among the factors contributing to higher metabolic energy costs, and lower exercise efficiency in individuals with obesity.

NEW & NOTEWORTHY Obesity is associated with greater triceps surae muscle-tendon stiffness, hysteresis, and a lower muscle strength to body mass ratio that, reduce calf raise efficiency, contributing to reduced exercise tolerance in individuals with obesity.

mechanobiology; mobility; musculoskeletal disease; obesity; tendons

INTRODUCTION

Beyond life-threatening comorbidities, obesity is associated with a lower muscle strength to body mass ratio, tendinopathy, higher risk of muscle and tendon injuries, and compromised locomotor function (1, 2). Structural and biomechanical alterations resulting from weight-related overloading and systemic dysmetabolic factors, such as the release of inflammatory cytokines triggered by adipokines from fat tissue, are implicated in muscle and tendon remodeling in individuals with obesity (3–5). Indeed, evidence from diet-induced obesity in mice highlights musculotendinous fibro-adipogenic structural remodeling, biomechanical properties alterations, compromised tissue repair, and reduced tolerance to tensile stress, ultimately reflected in loss of function and increased risk of injury (6, 7). Such mechano-structural musculoskeletal changes are likely to contribute to impaired physical performance, elevated injury risk, and reduced life quality, all of which have been observed in people with obesity (8).

Individuals with obesity expend more energy and are quicker fatigued when performing daily tasks like walking (9, 10). This may be not only attributable to their larger body mass and in turn, a lower muscle strength to body mass ratio, but also to a reduced capacity for elastic energy storage and reutilization (11). Muscle-tendon unit (MTU) mechanical properties play a crucial role in locomotion efficiency by storing kinetic and potential energy as elastic strain energy and returning it through elastic recoil (12, 13).

Obesity often leads to alterations in mechanical and contractile properties due to the physical interactions between excess fat and fibrotic tissue within and around muscles and tendons, and a disorganized tendinous structure (2, 6, 7, 14, 15). Tendons in individuals with obesity can exhibit increased collagen cross-linking, accumulation of advanced glycation end-products, and reduced collagen alignment, all of which contribute to structural disorganization and altered mechanical properties (4, 5). Such musculotendinous structural remodeling in obesity can increase viscosity and nonelastic stiffness (i.e., stiffness arising from pathological alterations such as fibrosis or fat infiltration, rather than functional adaptations), leading to greater resistance to lengthening and shortening, which dissipates more elastic potential energy and reduces the

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mechanical energy available for locomotion (12, 13). This is compounded by a higher body mass to muscle mass ratio, along with loss in fatigue-resistant and more efficient type I fibers, which make the muscles of individuals with obesity more prone to fatigue than nonobese muscles (16–18).

Obesity-induced intramuscular adipose tissue accumulation can negatively impact muscle quality by reducing specific force—the force generated per unit of muscle cross-sectional area—thereby impairing muscle strength and physical function (5, 14, 19). Given the critical role of the gastrocnemius in plantar flexion and locomotion, such changes in this muscle are especially detrimental. Reductions in gastrocnemius muscle strength can, in turn, lead to diminished Achilles tendon (AT) stretch and less stored strain energy, ultimately reducing movement efficiency (13). These findings suggest that changes in musculotendinous structures, alongside excess body mass, are significant factors in reducing functional capacity and movement efficiency in obesity, yet the specific effects of persistent obesity on the musculoskeletal system remain debated. In particular, there is limited understanding of how obesity-related alterations in the musculoskeletal system may affect exercise efficiency during tasks that involve repetitive muscle-tendon stretch-shortening cycles.

Therefore, the aim of this study was to assess the extent to which obesity-induced alterations in MTU mechano-structural properties contribute to changes in exercise efficiency. We hypothesized that subjects with obesity exhibit reduced efficiency in calf raises and diminished ankle mobility, which is at least partly attributable to a higher hysteresis of triceps surae MTUs during stretch-shortening cycles and higher nonelastic stiffness.

MATERIALS AND METHODS

Study Design and Participants

Forty-four 30- to 50-yr-old men voluntarily participated in this cross-sectional study. Twenty-two men had a healthy weight range [CN, body mass index (BMI): 18.5–24.9 kg/m^2] and another 22 men were classified as class I obese (OB, BMI: 30.0–34.9 kg/m^2) (Table 1) (20). All participants were sedentary and reported no engagement in regular physical activity or structured exercise training in the past 2 yr. Comprehensive information about the study's procedures, aims, and potential risks was provided, and written informed consent was obtained from each participant. The research protocol followed the Declaration of Helsinki and obtained approval from the Kaunas Regional Biomedical Research Ethics Committee (No. 2023-BE10-0001). Potential participants were excluded if they self-reported knee or ankle injuries that impeded maximal muscle contraction, a history of anterior knee pain or ankle pain, history of Achilles tendon rupture or tendinopathy, cardiovascular disorders, respiratory and neuromuscular diseases, or disclosed drug abuse. An a priori power analysis (IBM SPSS Statistics, v.21.0, IBM Corp., Armonk, NY) indicated that the present study was sufficiently powered (required sample size, $n = 15$) using $\alpha = 0.05$, $\beta = 0.80$, and an effect size = 1.09, based on previous research (5), where Achilles tendon cross-sectional area and gastrocnemius medialis tendon strain were compared between normal-weight and obese individuals.

Table 1. *Characteristics of the study population (means ± SD)*

	CN	OB	*P*	ES
Age, yr	38.6 ± 5.6	39.7 ± 4.7	0.437	0.24 (S)
Height, cm	181 ± 6	180 ± 5	0.436	0.24 (S)
Body mass, kg	77.5 ± 7.6	103.6 ± 7.9	<0.001	3.35 (V)
BF, %	11.9 ± 2.3	27.9 ± 2.9	<0.001	6.11 (V)
Lean body mass, kg	68.2 ± 5.7	74.6 ± 5.6	0.005	1.13 (M)
BMI, kg/m^2	23.3 ± 1.5	32.2 ± 1.7	<0.001	5.78 (V)
RMR, kcal/day	2650 ± 288	2709 ± 336	0.536	0.18 (S)
RMR/LM, kcal/day/kg	39.5 ± 3.5	36.2 ± 4.3	0.018	0.65 (M)
Calf$_P$, cm	37.1 ± 2.3	38.8 ± 1.8	<0.001	1.16 (M)
Calf$_M$, cm	38.5 ± 1.9	40.7 ± 1.6	<0.001	1.41 (L)
Calf$_D$ (cm)	36.1 ± 2.0	37.9 ± 1.7	<0.001	1.13 (M)
MGt resting length	18.7 ± 1.1	18.6 ± 1.3	0.441	0.04 (T)

BF, body fat; BMI, body mass index; CN, control; D, distal girth; LM, lean mass; M, medial girth; MGt, medial gastrocnemius tendon resting length; OB, obese; P, proximal girth; RMR: resting metabolic rate; T, trivial; S, small; M, moderate; L, large; V, very large.

Procedures

Participants were instructed to refrain from vigorous physical activity for 24 h before the testing session and to maintain their usual diet, avoiding large meals, energy drinks, or caffeinated beverages in the 2 h preceding the tests. Consistency was ensured by having the same group of investigators conduct all measurements at the same time of day for all participants, with the testing room maintained at a constant temperature of 22°C and a humidity level of 55%–60%. Following anamnesis and familiarization, testing commenced with collecting anthropometric data, including body mass and body fat analysis using a Tanita-305 body-fat analyzer (Tanita Corp), height (KaWe PERSON-CHECK stadiometer, Kirchner & Wilhelm GMBH + Co. KG), and dominant leg calf girths (ROLLFIX DIA Ø, Hoechstmass Balzer GMBH), following the International Society for the Advancement of Kinanthropometry (ISAK) guidelines (21).

Figure 1 illustrates the experimental procedures. After determination of anthropometric characteristics, B-mode ultrasound (Telemed, Lithuania) was used to examine the gastrocnemius lateralis (GL), gastrocnemius medialis (GM) and soleus (SOL) muscles, and the Achilles tendon (AT). Then participants performed a standard warm-up on a stationary bike followed by a passive resistive protocol and maximal voluntary isometric contractions of the plantar flexors at 0°, 10°, 20°, and 30° ankle joint angles angle using a calibrated Biodex System 4 dynamometer (Biodex Medical Systems). To record muscle activity, self-adhesive preamplified electrodes (F3010 FIAB) were attached to shaved, abraded, and cleaned skin regions over the GL and SOL muscles, following SENIAM recommendations (22), with ground electrodes fixed over the malleolus. Surface electromyographic (sEMG) signals were sampled at 1,000 Hz and synchronized with the dynamometer through a Biopac 12-bit-to-digital converter system (EL254S, Biopac Systems) and AcqKnowledge software (v.4.1, Biopac Systems). Recognizing the potential impact of subcutaneous adiposity on sEMG signal quality in individuals with obesity, we performed a pilot study to ensure adequate signal integrity without signal artifacts or apparent attenuations before data collection. Subsequently, gas exchange was determined during resting in sitting and standing positions, during a calf-raise

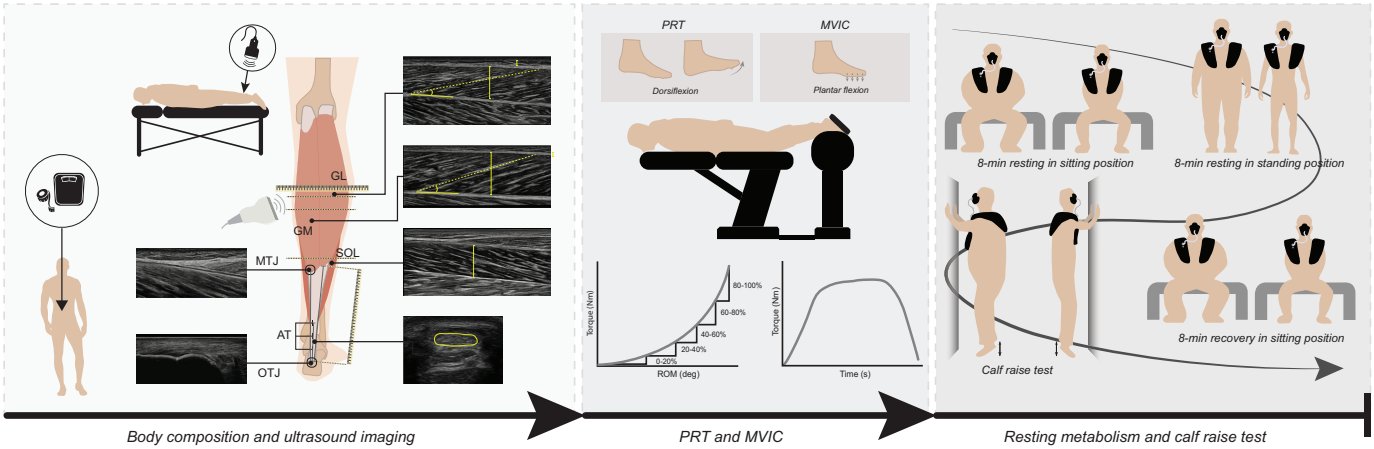

Figure 1. Representation of the study procedures. The tests commenced with anthropometric measurements and body composition. This was followed by the acquisition of static B-mode ultrasound images capturing images of the plantar flexor muscles and Achilles tendon. Subjects then participated in passive resistive torque tests (PRT) that stretched the plantar flexor muscle-tendon units, followed by a maximal voluntary isometric contraction protocol (MVIC). Subsequently, an 8-min resting metabolism assessment was conducted in a seated position and then in a standing position. Further evaluations included a 30-s calf raise test and an additional 8-min recovery interval, during which gas exchange was monitored in a seated position. AT, Achilles tendon; GL, gastrocnemius lateralis; GM, gastrocnemius medialis; MTJ, musculo-tendinous junction; OTJ, osteo-tendinous junction; ROM, range of motion; SOL, soleus.

exercise protocol and a recovery period. The exercise consisted in a 30-s calf raise test (CRT), where in addition to gas exchange, the dominant leg ankle joint displacement was tracked by recording the motion of the marked malleolus (23).

Musculoskeletal Ultrasound Imaging

Images of the GL, GM, and SOL muscles, and AT were obtained at 90° ankle joint angle using a grayscale B-mode ultrasonography 64-mm linear-array transducer (10- to 15-MHz transducer, Echoblaster 128, UAB; Telemed, Lithuania). The settings of the ultrasound system were identical for all participants and images were recorded using EchoWave II video-based software (Telemed). The probe was positioned at the midpoint of the GL, GM, and SOL muscle bellies, identified as the region of maximal muscle girth. This procedure ensured consistent placement across participants. Adjustments to the probe position were made, as needed, to obtain optimal image clarity and fascicle visualization. The fascicle length (Lf), pennation angle (PA), and muscle thickness (MT) were measured as the length of the fascicular path between the superficial and deep aponeuroses, the angle between the fascicular path and deep aponeurosis, and the shortest distance between superficial and deep aponeurosis, respectively (24). Subcutaneous adipose tissue (SAT) was measured by assessing the distance between the superficial aponeurosis and the skin over the GL and GM (25). Three ultrasound images were recorded and analyzed for each participant for each muscle, and the measured values were averaged (Fig. 1).

Images of the AT were collected, and AT resting length was measured as the linear distance between the most distal point of the gastrocnemius medialis tendon (GMT) musculotendinous junction (MTJ) and the tendon insertion to the calcaneus bone (26). Ultrasonography was used to locate the MTJ of the GM, GL, and SOL muscles, and the calcaneal osteotendinous junction. The linear distance between these points was then measured using a flexible measuring tape (ROLLFIX DIA Ø, Hoechstmass Balzer GMBH).

To measure the AT cross-sectional area (CSA), three points along the free AT length were marked at 25% (proximal), 50% (middle), and 75% (distal) of the length between the SOL-Achilles MTJ and the calcaneal insertion (27). Images were then collected in both longitudinal and transverse planes to measure the anterior-posterior tendon thickness and cross-sectional area (CSA) (28). The images were imported into ImageJ (v.1.46, US National Institutes of Health) (24) and the AT cross-sectional area (CSA_{AT}) was measured proximal, middle, and distal point (28). The CSA_{AT} was 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. Three ultrasound images were recorded and analyzed for each participant at each of the three locations, and the measured values were averaged.

Intra-rater repeatability for all measurements was assessed in five individuals who underwent three measurements per session across five separate days, each separated by a 7-day interval. Intraclass correlation coefficient (ICC) values ranged from 0.89 to 0.96 (means ± SD: 0.93 ± 0.03), and within-subject coefficient of variation (CV) ranged from 2.0% to 5.0% (means ± SD: 3.4 ± 1.2%), indicating high reliability across all variables (29).

Passive Resistive Torque

To determine the passive resistive torque (PRT), a Biodex System 4 isokinetic dynamometer was used to measure torque during passive stretch, capturing ankle joint angle and angular velocity. Participants lay prone with their trunk, lower back, and thigh secured by adjustable lap belts, with legs fully extended. The dominant foot was firmly strapped to a footplate attached to the dynamometer's lever arm, ensuring alignment of the dynamometer's input axis with the ankle joint's axis of rotation (i.e., the malleolus). In this configuration, and with the participant fully relaxed, torque data were collected using two distinct protocols to capture

comprehensive information about muscle-tendon behavior. First, a fixed range of motion (ROM) protocol was performed, where the ankle was passively flexed to a predefined range of motion (30° dorsiflexion) at a speed of 4°/s (30). In the second protocol, referred to as the full ROM test, the dynamometer passively flexed the ankle from 10° plantar flexion to the participant's maximal dorsiflexion (participants' self-reported point of pain) at the same speed. Both protocols were repeated three times, with a 1-min rest between sets. For the full ROM protocol, the maximal ankle ROM and peak passive torque were recorded, whereas during the fixed ROM test, GMT displacement (GMT_D) was measured by tracking the GM muscle-tendon junction using B-mode ultrasound videos (31). GMT strain was calculated as: $\frac{L-L_0}{L_0} \times 100\%$, where L_0 is the AT resting length and L is the instantaneous length (32). Triceps surae MTU passive stiffness was calculated using the fixed ROM test data and by dividing the change in MTU passive torque by the change in ankle joint angle, expressed as:

$$\text{Stiffness} = \frac{\Delta\text{Torque}}{\Delta\text{ROM}}$$

The slope of the torque-angle curve from the ascending part of the loop was evaluated across distinct ranges of the ROM: 0%–20%, 20%–40%, 40%–60%, 60%–80%, and 80%–100% using a least-squares regression method within these discrete time points. To account for gravity, torque data were adjusted using a customized Excel spreadsheet for calculations. Gravitational effects were corrected by estimating the foot weight as the torque produced in a fixed plantar-flexed position, where only the weight of the foot contributed to the torque, after normalization for the weight of the dynamometer tool. Stiffness was normalized to the sum of the muscle thicknesses of the SOL, GM, GL, and the CSA_{AT} (i.e., the estimated triceps surae MTU size, expressed in arbitrary units), to express stiffness relative to the size of the contributing tissues. The area within the passive torque—joint angle loop, as percentage of the area beneath the curve during the ascending phase, was calculated as hysteresis (HYS) of the plantar flexor muscle-tendon units, representing the energy dissipation within the plantar-flexor system:

$$\text{HYS} = \frac{\text{Area between the loading and unloading curve}}{\text{Area underneath the loading curve}} \times 100\%.$$

PRT measurements were deemed valid only if sEMG activity was <5% of the maximum sEMG value (sEMGmax) recorded during the maximal voluntary contraction test (33).

Intra-rater repeatability for all measurements was assessed in five individuals who underwent three measurements per session across five separate days, each separated by a 7-day interval. ICC values ranged from 0.87 to 0.94 (means ± SD: 0.90 ± 0.04), and within-subject CV ranged from 4.1% to 6.2% (means ± SD: 5.3 ± 1.4%), indicating high reliability across all variables (29).

Maximal Voluntary Isometric Contractions

After the passive-resistive torque measurements, participants performed maximal voluntary isometric contractions. Participants were signaled by a supervisor to contract as fast and forcefully as possible, receiving verbal encouragement during the test (12). The subjects executed three maximal contractions at ankle joint angles of 0°, 10°, 20°, and 30° dorsiflexion, where 0° represents the neutral position with the ankle at 90°. All measurements were conducted using a calibrated Biodex System 4 dynamometer (Biodex Medical Systems). Each contraction lasted 2 s, with 2-min recovery between each contraction. The maximal voluntary torque (MVT) represented the peak isometric torque (Nm) during the contraction. The rate of torque development (RTD) was calculated as peak slope (Nm/s) in early (RTD_E) (0–100 ms) and late (RTD_L) (0–300 ms) phases of force development (34). The onset of a contraction was defined as the moment that plantar flexors' torque surpassed 2.5% of the baseline-to-peak torque difference, ensuring a reproducible criterion while minimizing baseline noise (34). The sampling frequency was 1,000 Hz. The collected torque signals were filtered using a fourth-order low-pass Butterworth filter (cut-off frequency: 10 Hz) to minimize high-frequency noise while preserving the mechanical signal characteristics, with baseline noise consistently below 1% of MVT. All values were normalized to the sum of the muscle thickness of the SOL, GM, and GL, and by body mass raised to the power of $\frac{2}{3}$ (35). In addition, the RTD by maximal voluntary isometric torque and the body mass by maximal voluntary isometric torque ratio ($BM \times MVT^{-1}$) were calculated (36). The MVT and RTD data obtained from the best of the three repetitions of the voluntary isometric contractions at each angle were analyzed. The sEMG signal of the GL and SOL muscles were recorded and the sEMGmax value quantified by calculating the root mean squared over a 0.05-s period around the peak torque achieved during the maximal voluntary contractions.

Intra-rater repeatability for all measurements was assessed in five individuals who underwent three measurements per session across five separate days, each separated by a 7-day interval. ICC values ranged from 0.89 to 0.97 (means ± SD: 0.93 ± 0.05), and within-subject CV ranged from 2.2% to 6.1% (means ± SD: 4.1 ± 2.4%), indicating high reliability across all variables (29).

Calf-Raise Test

To determine energy expenditure during resting, standing, exercise, and recovery, gas exchange was monitored using a stationary spiroergometric device (MetaLyzer 3B, Cortex Biophysik) synchronized with a chest heart rate (HR) belt sensor (Polar H10, Polar Electro). Gas exchange data were collected breath-by-breath for 8 min under two conditions: sitting at rest and standing at rest, followed by a 30-s CRT and an 8-min sitting recovery period. The first min in each condition was excluded to allow for steady-state gas exchange to be reached. The integral of gas exchange over the subsequent minutes was calculated and used as representative of the metabolic response during that condition. For the 30-s CRT, gas exchange data from the entire test duration were included, along with a 3-min excess postexercise oxygen consumption (EPOC) interval recorded during the sitting recovery period (37).

For the CRT, participants were instructed to perform as many calf-raises as possible at a self-determined pace in 30 s (23). These test conditions were chosen to reflect functional tasks performed at an individual's natural pace, as previously validated in a similar protocol developed for elderly

participants (38). During the test the subjects were instructed to reach the maximal ankle ROM with, previously marked on the wall, with both limbs and touching the heels to the ground at each repetition. The participants were barefoot, heels on the ground, knees extended, using their fingers touching a wall for balance. A reflective marker was placed on the malleolus, and a video was recorded at 88 frames/s using a high-speed camera (acA1300-75gc, Basler AG, Ahrensburg, Germany) in a sagittal position to track (Kinovea v.0.9.5, Charmant, J., & contributors) the vertical displacement of the ankle, count the calf raise repetitions, and to analyze the different phases of the calf raise exercise (i.e., isometric, concentric, and eccentric). The concentric phase was identified as the upward movement of the ankle, the eccentric phase as the downward movement, and the isometric phase as the period when the ankle was stationary between movements. Rate of perceived exertion (RPE) was assessed using the BORG-CR10 scale immediately after the exercise (39).

Data were analyzed separately for each stage (i.e., sitting rest, standing rest, exercise, recovery). Gas exchange data were integrated and HR data was averaged during the different intervals for further calculations. Energy expenditure (EE) was calculated from the measures of $\dot{V}CO_2$ and $\dot{V}O_2$ (in L/min) and analyzed using the formula of Brouwer (40): $\left[(3.869 \times \dot{V}O_2) + (1.195 \times \dot{V}CO_2)\right] \times (4.186/60) \times 1,000$. The metabolic cost of exercise (MCE) was computed by subtracting the standing resting EE from the exercising EE and total work (W) calculated using body mass, gravity force, and CRT total vertical displacement: $W = M \times g \times \Sigma h$ (37). CRT net efficiency (NE) was calculated using the formula: $\frac{\text{Total work}}{\text{MCE}} \times 100\%$.

Intra-rater repeatability for all measurements was assessed in five individuals who underwent three measurements per session across five separate days, each separated by a 7-day interval. ICC values ranged from 0.85 to 0.91 (means ± SD: 0.90 ± 0.02), and within-subject CV ranged from 5.0% to 6.8% (means ± SD: 5.1 ± 1.3%), indicating high reliability across all variables.

Statistical Analysis

Statistical analysis was conducted using IBM SPSS Statistics (v.21.0; IBM Corp., Armonk, NY), and graphs were created using GraphPad Prism (v.7.0; GraphPad Software, San Diego, CA). Descriptive statistics (means ± SD) were calculated for each variable, and the normality of the sample distribution was assessed using the Shapiro–Wilk test. Independent *t* tests were carried out to determine if there were significant differences in the mean values of all tested variables among the two groups (CN vs. OB). Effect size (ES) was determined based on Cohen's guidelines (41), with the standardized mean difference (*d*) for the pairwise comparisons interpreted as trivial (T), <0.20; small (S), 0.20 to 0.59; moderate (M), 0.60 to 1.19; large (L), 1.20 to 1.99; and very large (V), ≥2.00 (42). In addition, a repeated-measures ANOVA was used to assess differences between resting, standing, exercise, and recovery metabolism. The between-factor was group (CN vs. OB) and the within-factor condition (sitting vs. standing vs. exercising vs. recovering), with EE, HR, and respiratory exchange ratio (RER) as the comparison variables. A Tukey-corrected post hoc test was performed to assess statistical significance of differences between mean values of the four different conditions if a main effect of condition, or a group × condition interaction was found. The standardized effect (η^2) being small (S) for $\eta^2 > 0.1$, medium (M) for $\eta^2 > 0.25$, and large (L) for $\eta^2 > 0.4$ (41). Pearson correlation analysis and stepwise linear regression ($n = 44$) were used to explore potential relationships between NE (dependent variable) and musculotendinous mechanical properties indicators (independent variables). Stepwise regression was conducted using a forward selection method, with variables entered if $P < 0.05$ and removed if $P \geq 0.10$. The stopping rule for model selection was based on the Akaike Information Criterion (AIC) to avoid overfitting. The critical level of statistical significance was set at an α level of 0.05.

RESULTS

OB were characterized by greater body mass, body fat, and BMI than CN ($P < 0.001$) (Table 1). OB showed also greater calf girths than CN ($P < 0.001$) (Table 1).

Plantar Flexors Musculotendinous Structures

OB showed greater SOL muscle thickness ($P < 0.001$, ES: 2.15, L; Fig. 2*A*). There were, however, no significant differences in GL and GM MT between OB and CN (Fig. 2*A*, Supplemental Table S1). The AT cross-sectional area was larger in the OB than CN ($P < 0.001$, ES: 1.71, L; Fig. 3*B*, Supplemental Table S1). Calf muscles SAT was greater in OB than CN ($P < 0.001$, ES: 3.38, V) (Fig. 2*C*, Supplemental Table S1). There were no significant differences between OB and CN in the muscle architecture of GM and GL muscles (data not shown). OB showed greater cumulative muscle thickness (CN: 44.75 ± 3.04 mm; OB: 47.4 ± 4.1 mm; $P = 0.01$, ES: 0.74) and triceps surae MTU size (CN: 100 ± 5 au; OB: 109 ± 5 au; $P < 0.001$, ES: 1.61).

Plantar Flexors Passive Mechanical Properties

A second-order polynomial function best fitted the triceps surae MTU passive torque-angle curves ($R^2 = 0.998 \pm 0.001$). OB were characterized by greater triceps surae MTU stiffness at the different intervals of the passive torque-angle curve ($P < 0.001$, ES: 1.44, L; Fig. 3*B*). Normalization for triceps surae MTU dimensions did not alter the observed differences in passive stiffness between CN and OB. OB plantar flexor muscle-tendon units showed greater HYS than CN ($P < 0.001$, ES: 2.58, V; Fig. 3*C*).

OB showed lower GMT_D ($P < 0.001$, ES: 1.63, L), GMT strain ($P < 0.001$, ES: 2.34, V), and passive ROM ($P < 0.001$, ES: 2.01, V) of the plantar flexors, and greater peak passive torque (PPT) than CN ($P < 0.001$, ES: 1.64, L) (Fig. 4).

Plantar Flexors Contractile Properties

No significant differences were found in the plantar flexor MVT and RTD at different muscle lengths between OB and CN (Supplemental Table S2). However, CN shower greater MVT ($P < 0.05$) when normalized for muscle dimension (Table 2) or body mass ($P < 0.001$, ES: 1.17, L). RTD normalized to MVT did not differ significantly between OB and CN.

Calf Raises Efficiency and Physiological Response to Resting, Standing, and Exercising Conditions

The repeated-measures ANOVA revealed significant interactions (group × condition) for EE ($P < 0.001$, η^2:

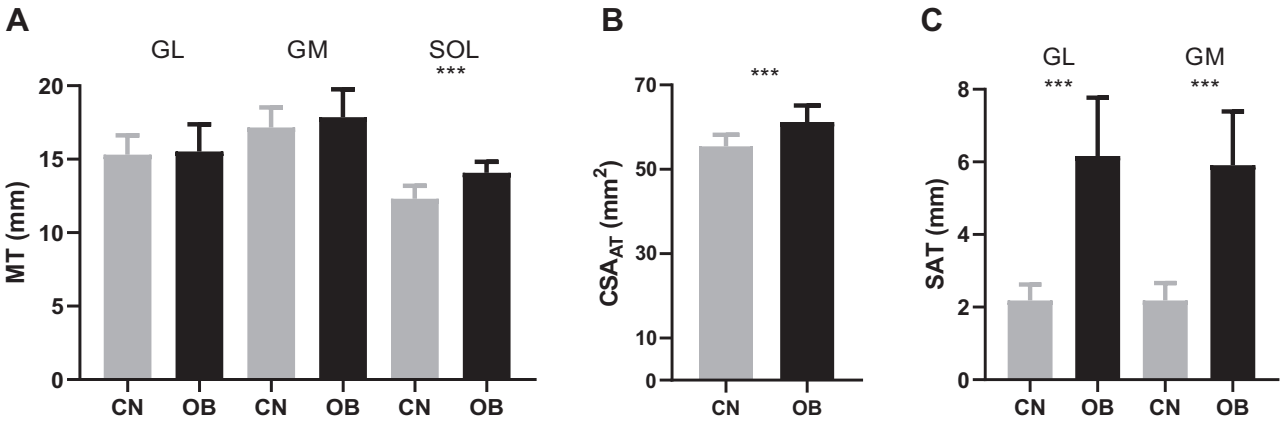

Figure 2. Bar graphs representing the differences in muscle thickness (MT) (*A*), tendon cross-sectional area (CSA_{AT}) (*B*), and calf subcutaneous adipose tissue thickness (SAT) (*C*). AT, Achilles tendon; CN, control group; GL, gastrocnemius lateralis; GM, gastrocnemius medialis; OB, obese group; SAT, subcutaneous adipose tissue; SOL, soleus. ***$P < 0.001$.

0.10, S) and RER ($P < 0.001$, η^2: 0.12, S) (Fig. 5). More in detail, OB showed similar EE and RER but higher HR ($P < 0.001$, ES: 1.66, L) than CN while resting in a sitting position. EE and HR were greater in OB than CN ($P < 0.024$, ES: 0.62, M) with no significant differences in RER while standing.

During the calf-raise test OB showed greater HR ($P < 0.001$, ES: 1.61, L), MCE, NE, and RPE than CN ($P < 0.001$, ES: 1.08, M), and the total work was greater in OB than CN ($P < 0.01$, ES: 1.14, M). MCE was greater in OB also when expressed per kilogram of body mass ($P < 0.033$, ES: 1.01, M). Furthermore, CN completed more calf raise test repetitions and were characterized by a greater total vertical displacement (VDtot) than OB ($P < 0.015$, ES: 0.68, M) (Fig. 6). OB showed greater time spent in concentric, eccentric, and isometric phases per calf raise ($P < 0.001$, ES: 1.44, L) and a greater drop in vertical displacement from the start to the end of the test ($P < 0.001$, ES: 2.02, V) than CN (Fig. 7). Yet, OB showed greater HR and RER during the recovery period, sitting at rest, than CN ($P < 0.003$, ES: 0.96, M) (Fig. 5).

There was a negative correlation between calf raise velocity and BM $\times$ MVT^{-1} ($R = -0.60$, $P < 0.001$, 95% confidence interval (CI): -0.76 to -0.37) (Fig. 8).

Relationships between NE, HYS, and BM $\times$ MVT^{-1}

The calf raise velocity was negatively related to the BM·MVT^{-1} (Fig. 8) indicating that the larger the ratio of BM to MVT the slower the calf raise. This is to be expected from the force-velocity relationship and given that the data of the CN and OB follow the same curve, this indicates that the slower calf raise is explicable by a lower MVT:BM ratio rather than a slower contraction velocity of the muscles of the OB compared with the CN. Furthermore, there was a negative relationship between NE and BM·MVT^{-1} ($R = -0.41$, $P = 0.005$, 95% CI: -0.63 to -0.13).

A negative correlation was found between NE and HYS ($R = -0.38$, $P = 0.018$, 95% CI: -0.62 to -0.09) and a positive correlation between HYS and BM·MVT^{-1} ($R = 0.58$, $P < 0.001$, 95% CI: 0.35–0.75) indicating a higher hysteresis was associated with a lower relative strength. Triceps surae MTU hysteresis accounted for 14.4% (ES: M), whereas the BM $\times$ MVT^{-1} ratio accounted for 16.8% (ES: L) of the NE (Fig. 9).

Stepwise regression analysis revealed that while the BM $\times$ MVT^{-1} ratio was the primary determinant of NE, triceps surae MTU hysteresis modestly contributed as a secondary

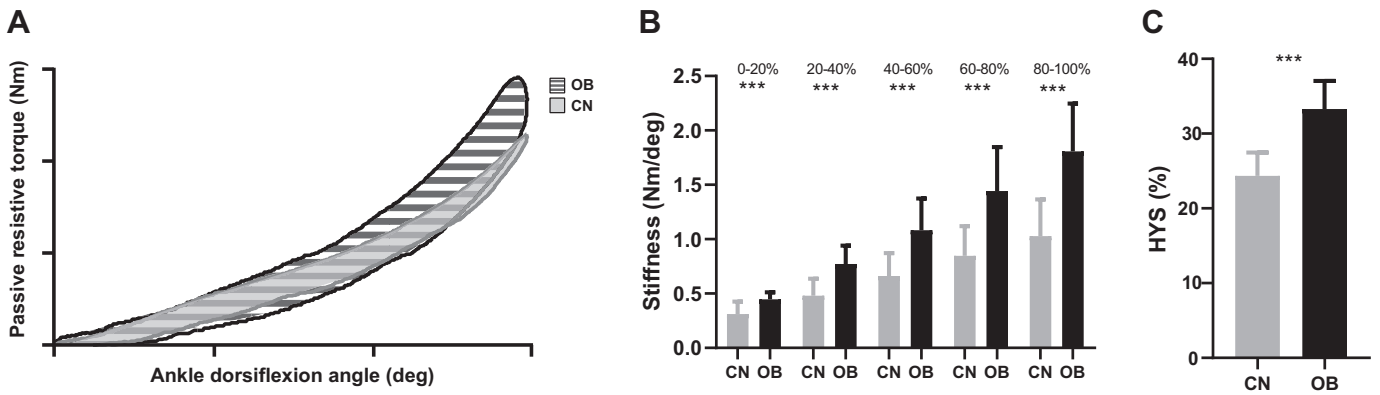

Figure 3. Representation of the raw ankle muscle tendon units passive resistive torque–ankle dorsiflexion angle curves during fixed range of motion (ROM) protocol (*A*) and bar graphs representing the differences in passive stiffness (*B*) and hysteresis (HYS) (*C*) between the control (CN) and obese (OB) group. ***$P < 0.001$.

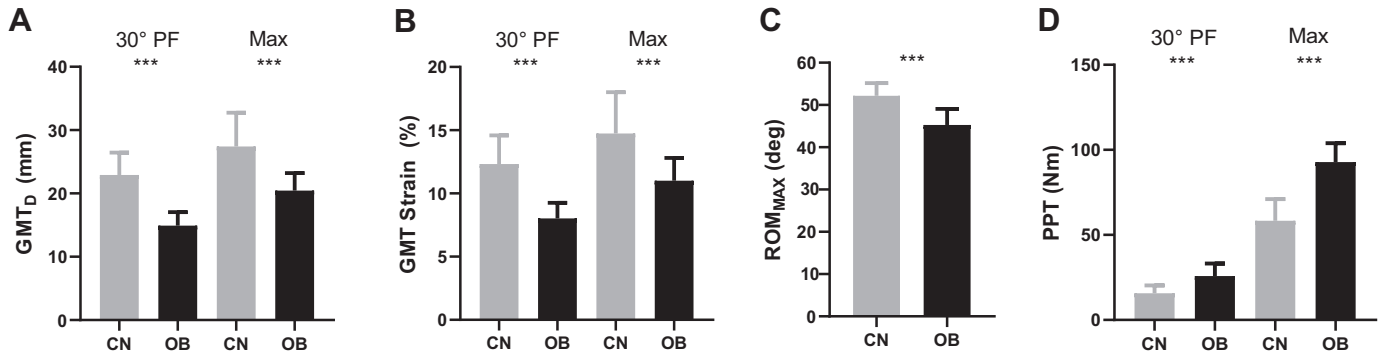

Figure 4. Bar graphs representing the differences in gastrocnemius medialis tendon displacement (GMT_D) (*A*), gastrocnemius medialis tendon (GMT) strain (*B*), plantar flexor muscle-tendon unit range of motion (ROM) (*C*), and peak passive torque (PPT) (*D*), in the fixed ROM test (30° DF) and maximal ROM test (Max). CN, control group; GL, gastrocnemius lateralis; GM, gastrocnemius medialis; Max: maximal range of motion trial; MGT_D, gastrocnemius medialis tendon displacement; OB, obese group; PPT, peak passive torque; SOL, soleus; 30 DF, till 30° dorsiflexion. ***$P < 0.001$.

predictor, with both factors together explaining 22.7% of the variance in NE ($P < 0.05$).

DISCUSSION

The primary observation of our study is that inefficiency in calf raises observed in individuals with obesity is associated with a higher body mass-to-maximal voluntary isometric torque ratio and greater triceps surae MTU hysteresis. Specifically, these factors were estimated to account for 16.8% and 14.4% of the variability in calf raise efficiency, respectively.

The bigger AT and SOL muscle did not translate into greater strength and was in fact less when adjusted for body mass and muscle thickness. This imbalance may also contribute to calf raise inefficiency. In addition, we observed that individuals with obesity exhibited greater triceps surae MTU stiffness, along with lower GMT displacement and strain, which could be associated with restricted ankle ROM. However, this relationship is likely bidirectional: reduced joint ROM may itself result in less tendon elongation and strain, underscoring the complex interplay between these factors.

Table 2. *Plantar flexors' contractile properties (MVT and RTD) between CN and OB participants (means ± SD)*

	CN	OB	*P*	ES
0°				
MVT, Nm	141 ± 29	128 ± 38	0.215	0.381 (S)
nMVT, Nm/mm	3.14 ± 0.59	2.69 ± 0.77	0.018	0.652 (L)
$_E$RTD, Nm/s	724 ± 157	601 ± 124	0.078	1.073 (M)
n_ERTD, Nm/s/mm	16.2 ± 3.3	12.3 ± 2.3	<0.001	1.353 (V)
$_L$RTD, Nm/s	520 ± 114	469 ± 116	0.153	0.439 (S)
n_LRTD, Nm/s/mm	11.6 ± 2.4	9.8 ± 2.0	0.006	0.798 (L)
10°				
MVT, Nm	169 ± 39	158 ± 49	0.384	0.256 (S)
nMVT, Nm/mm	3.78 ± 0.80	3.31 ± 0.98	0.047	0.515 (M)
$_E$RTD, Nm/s	807 ± 175	730 ± 183	0.098	0.426 (S)
n_ERTD, Nm/s/mm	17.9 ± 3.4	13.2 ± 3.4	<0.001	1.381 (V)
$_L$RTD, Nm/s	565 ± 130	504 ± 157	0.169	0.422 (S)
n_LRTD, Nm/s/ mm	12.6 ± 2.6	10.5 ± 2.8	0.009	0.745 (L)
20°				
MVT, Nm	197 ± 49	184 ± 60	0.444	0.233 (S)
nMVT, Nm/mm	4.39 ± 1.02	3.86 ± 1.16	0.060	0.478 (M)
$_E$RTD, Nm/s	830 ± 160	755 ± 206	0.095	0.411 (S)
n_ERTD, Nm/s/mm	18.5 ± 3.1	13.7 ± 3.9	<0.001	1.336 (V)
$_L$RTD, Nm/s	577 ± 123	531 ± 182	0.336	0.293 (S)
n_LRTD, Nm/s/mm	12.8 ± 2.4	11.1 ± 3.4	0.029	0.589 (M)
30°				
MVT, Nm	229 ± 67	213 ± 74	0.449	0.231 (S)
nMVT, Nm/mm	5.09 ± 1.39	4.44 ± 1.38	0.064	0.468 (M)
$_E$RTD, Nm/s	883 ± 198	773 ± 201	0.097	0.548 (S)
n_ERTD, Nm/s/mm	19.7 ± 3.9	14.1 ± 3.8	<0.001	1.442 (V)
$_L$RTD, Nm/s	605 ± 139	548 ± 183	0.251	0.352 (S)
n_LRTD, Nm/s/mm	13.5 ± 2.8	11.4 ± 3.3	0.017	0.661 (L)

CN, control; MVT, maximal voluntary torque; OB, obese; PT, peak torque; RTD, rate of torque development; n, normalized to muscle thickness; E, early; L, late; T, trivial; S, small; M, moderate; L, large; V, very large.

Triceps Surae MTU Stiffness

In our study, individuals with obesity had a greater passive triceps surae MTU stiffness than age-matched controls. The greater stiffness observed in OB may be driven by structural differences within the MTU. Specifically, we found that CSA_{AT} was on average 10% larger in OB, with cumulative muscle thickness and triceps surae MTU size being 6% and 9% larger, respectively. Furthermore, SAT surrounding GL and GM was 2.8 and 2.7 times thicker in OB. Greater muscle SAT is a common feature of obesity, especially in the gastrocnemius muscles (19). Ectopic fat not only surrounds soft tissues but also accumulates between and within muscles and tendons in obesity, alongside an aberrant accumulation of fibrotic tissue (43, 44). Given the higher viscosity of adipose tissue relative to skeletal muscle (45) and its potential to alter skeletal muscle material properties (15), the heightened MTU stiffness may be attributable to structural modifications in muscles and tendons, such as fat infiltration (46), abnormal accumulation of cross-linking by e.g., advanced glycation end products (2, 47), and/or interactions between adipose tissue and musculotendinous structures (14, 15, 45) that all have been associated with obesity.

As seen in the present study, previous research has suggested that obesity is associated with greater axial stiffness of the triceps surae MTU (48), greater (~12%) AT thickness (49), CSA_{AT}, and stiffness (5). Paradoxically, increased tendinous and MTU stiffness has been positively associated with physical performance, particularly in athlete populations, attributed to faster and more efficient muscle-to-bone force

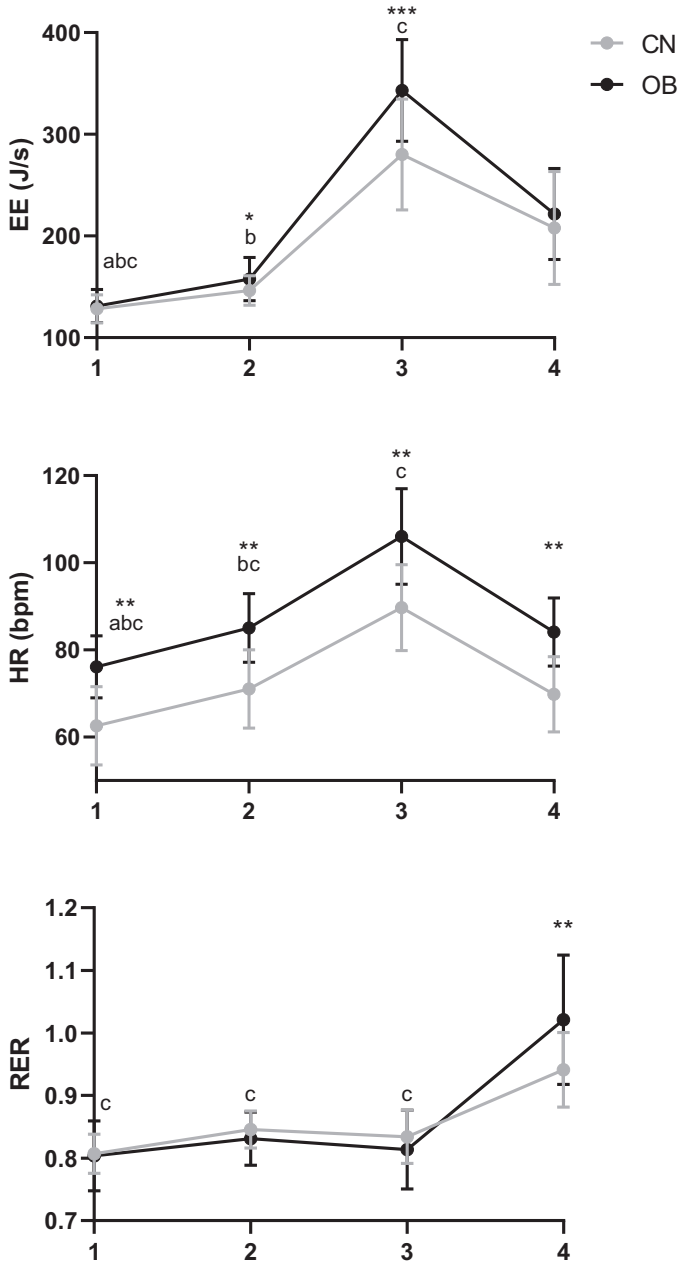

Figure 5. Variations in energy expenditure (EE), heart rate (HR), and respiratory exchange ratio (RER) during different test phases (1: sitting; 2: standing; 3: calf raise test; 4: recovery) and between obese (OB: black line and symbol) and control (CN: gray line and symbol) groups. $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$, for CN versus OB; $^{a}P < 0.05$: vs. standing (2); $^{b}P < 0.05$: vs. calf raise test (3); $^{c}P < 0.05$: vs. recovery (4).

transfer (29, 50, 51). Consequently, in the healthy population, an elevated musculotendinous stiffness might be viewed as a beneficial adaptation to mechanical loading (52). In athletes, however, this increased stiffness is not accompanied by an increased body mass, and perhaps that is why a similar adaptation does not confer a performance benefit for the individuals with obesity.

Furthermore, obesity is also linked to an elevated risk of tendinopathy (53). This increased risk may be a consequence of altered acute tendinous responses to exercises, where fluid redistribution within the tendon may be compromised (49) and result in intrinsic alterations of the material properties of the MTU, as an increase in stiffness in individuals with obesity. Here, we observed that during passive stretching, the maximal AT strain at 30° flexion was significantly lower in individuals with obesity compared with those who were not obese. This finding suggests that obesity may be associated with proportionally greater stiffness in the tendon than in the muscle. Consequently, when passively stretching the MTU, the muscle may be required to elongate further to compensate for the stiffer tendon. Similarly, during (isometric) MTU contraction at a given force level, the stiffer tendon elongates less, potentially reducing heel elevation and contributing to the observed lower ankle joint ROM during calf raises in individuals with obesity. These findings provide insight into the potential mechanical constraints affecting joint mobility and calf raise performance in obesity, highlighting the complex interplay between tendon stiffness and muscle behavior. However, it remains unclear whether similar compensatory mechanisms occur under dynamic conditions, and further studies are warranted to explore the interplay between tendon and muscle stiffness, elongation, and neural control during functional movements such as calf raises.

Triceps Surae MTU Hysteresis

In our research, the viscosity of the triceps surae MTU—measured as hysteresis—was significantly greater in OB as reflected by the 33% loss of elastic strain energy during a stretch-shortening cycle in OB compared with just 24% in CN. This is similar to the hysteresis of the AT reported for nonobese people ranging from 18% (54) to 20% (55) during isometric contractions and up to 26% during one-legged hopping (56). While acknowledging the complexity of MTU system behavior, the increased loss of elastic energy may be attributed to frictional interactions, with obesity-associated alterations in muscles and tendons potentially contributing to greater energy dissipation during MTU stretch-shortening cycles (15, 45). Since low hysteresis of the MTU is beneficial for efficient locomotion as it allows for most of the stored elastic energy to be reused during propulsion it is no surprise that we found a negative relationship between hysteresis and the net efficiency of calf raises.

Therefore, the results of the passive triceps surae MTU stretch-destretching test indicate that *1*) the triceps surae MTU is stiffer in individuals with obesity, possibly due to obesity-related structural and material properties alterations. *2*) During stretching, the obese triceps surae MTU stores more mechanical energy than in normal-weight men. However, it is conceivable that the presence of increased connective tissue, including adipose and fibrotic tissue within and around the muscle and tendon, may lead to increased viscosity of the MTU. As a result, during a stretch-shortening cycle a greater amount of elastic energy is lost as heat. Thus, despite a larger energy input into the triceps surae MTU in OB, calf raising was less efficient in OB compared with CN. Notably, individuals with obesity also exhibited longer pauses between calf raise "stretch-shortening" cycles, which could further influence efficiency, as longer pauses between tendon stretching and relaxation can potentially reduce the amount of stored mechanical energy that can be used.

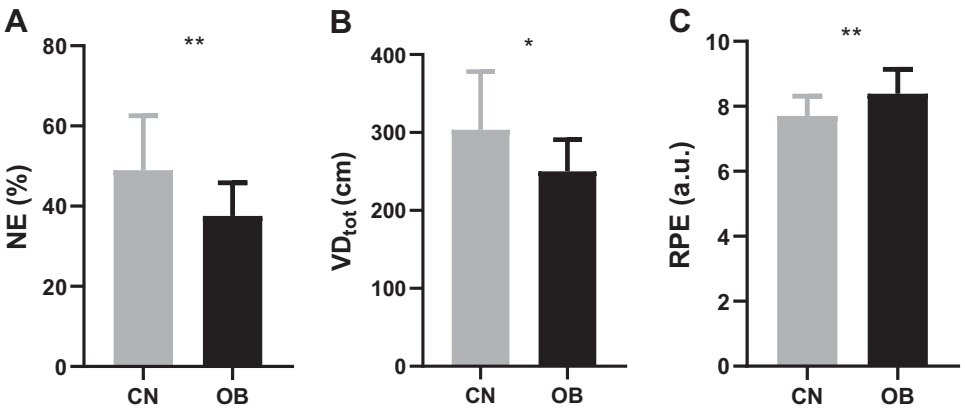

Figure 6. Bar graphs representing the differences between control (CN) and obese (OB) in calf raise test: net efficiency (NE) (*A*); total vertical displacement (VD_{tot}) (*B*); and rate of perceived exertion (RPE) (C). $*P < 0.05$; $**P < 0.01$.

Muscle Strength

Here we found that not only the MVT relative to body mass was less in OB than CN, but also the MVT relative to muscle thickness, suggesting a lower muscle quality. This is in line with previous observations of a lower force-generating capacity per unit of muscle tissue, associated with accumulation of noncontractile material, in OB (15). These alterations likely impair force transmission and contribute to diminished muscle efficiency. Despite these differences in strength measures, the relationship between calf raise velocity and the BM:MVT ratio was similar in the OB and CN group, indicating that—as seen with loading and unloading in older and younger people (36)—obesity is not associated with slowing of the muscle. In other words, the shortening velocity of a maximally activated muscle at a specific fraction of the maximal force-generating capacity appears to be similar in obese and nonobese individuals. Further evidence for similar contractile properties is the lack of significant differences in the rate of torque development when normalized to the maximal torque-generating capacity of the muscle.

Nevertheless, as a consequence of the higher BM:MVT ratio, the OB plantar flexors had to operate on the right side of the calf raise $V - BM \times MVT^{-1}$ curve resulting in a slower calf raise. This slower calf rise velocity suggests that individuals with obesity operate at a less efficient part of the force-velocity curve (36, 57, 58), which likely contributes to reduced movement efficiency, as supported by the observed relationship between net energy efficiency of calf raises and the $BM \times MVT^{-1}$ ratio (57, 58). This inefficiency is further compounded by the greater mechanical work required to overcome the increased body mass during each repetition.

During a calf raise, as plantar flexion begins, the muscle fibers generate force to overcome both body mass and friction, moving the ankle against the inertial load. When the

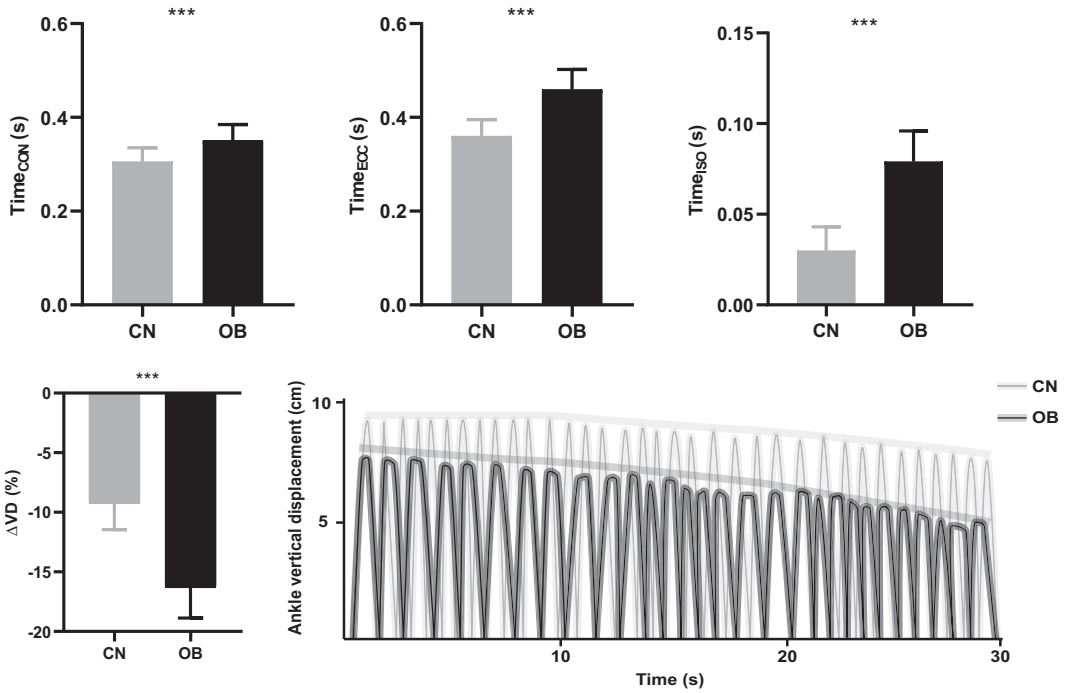

Figure 7. Differences in time spent in concentric ($Time_{CON}$), eccentric ($Time_{ECC}$), and isometric ($Time_{ISO}$) phases and the delta (first to last repetition) of ankle vertical displacement (ΔVD), during the calf raise test between obese (OB) and control (CN) groups. $***P < 0.001$, for CN vs. OB.

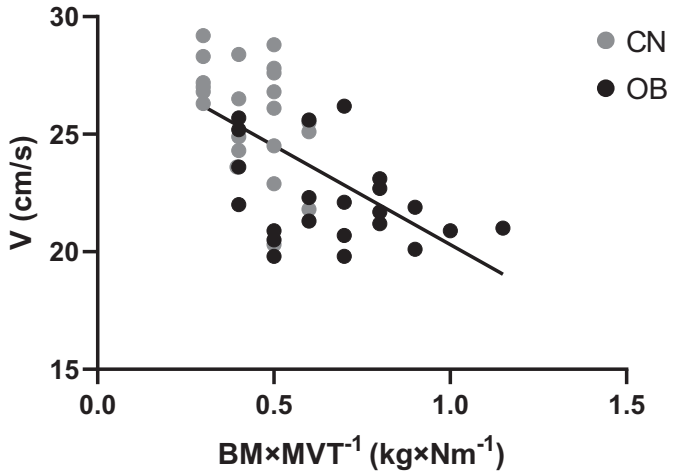

Figure 8. Calf raises velocity (*V*) as a function of body mass divided by maximal voluntary isometric torque (BM × MVT^{-1}) of pooled data from control (CN, black dots) and obese (OB, gray dots) groups.

muscle fibers actively contract to perform positive work, their mechanical energy output is used to overcome friction and to increase the kinetic and gravitational potential energies of the whole body (12). In individuals with obesity, increased viscosity of the muscle and tendon, as discussed earlier, likely causes greater friction, dissipating more mechanical energy as heat and requiring the muscle to exert more force to overcome this friction.

We can infer muscle inefficiencies in OB through indirect measures. For instance, previous research has found a negative correlation between musculotendinous hysteresis and greater muscle activity with exercise efficiency (59, 60), likely leading to earlier fatigue onset. Such an earlier onset of muscle fatigue may be further acerbated by the fact that, as discussed earlier, the muscles of people with obesity work at a less efficient part of the force-velocity curve (36, 57, 58). Indeed, we did observe an earlier onset of fatigue, reflected by a steeper drop in vertical displacement during the calf raise test, and higher perceived exertion in individuals with obesity than controls.

Limitations

Overall, this study indicates that obesity is accompanied by a higher cost of energy for basic daily functions, like standing or stair climbing. We illustrated the role of greater passive stiffness and hysteresis in the often-observed lower joint mobility and movement inefficiency in individuals with obesity. Future studies could enhance our understanding by investigating muscle activity in greater detail, including antagonist muscles sEMG recordings and incorporating sEMG data together with more comprehensive kinetic and kinematic analyses during active movements (60). Although the CRT used in this study has been validated and effectively reflects exercise capacity and plantar dorsi-flexor function, the lack of standardized tempo during the CRT should be considered when interpreting our results. When analyzing Achilles tendon elongation during passive stretching, we did not account for its natural curvature, which may introduce minor inaccuracies (61). However, given that the movement was limited to plantar dorsiflexion on a fixed dynamometer chair, the impact of this limitation is likely minimal. Furthermore, the displacement of the calcaneal osteo-tendinous junction (OTJ) was not explicitly controlled for, potentially introducing an error that falls within the measurement error of our measurements. This is in addition to the inherent measurement error associated with evaluating dynamic MTJ displacement (26). We acknowledge that the triceps surae MTU is a complex system, and the approach used in this study may have oversimplified its behavior. Specifically, some discrepancy may exist between passive and dynamic assessments of soft tissue material properties. Furthermore, we focused solely on GMT behavior; a more comprehensive evaluation, including gastrocnemius lateralis and soleus MTJ behavior, may reveal further insights (32). Employing advanced imaging techniques, such as MRI, to examine the structural properties of muscles and tendons could provide a more detailed characterization of the obese musculotendinous phenotype (62). This approach could also offer valuable insights into the relationship between intramuscular fat accumulation, joint mobility, and contractile performance.

Conclusions

Our study indicates that individuals with obesity had a lower energy efficiency in performing repeated calf raises. This lower efficiency may be partially explained by *1*) a higher body mass-to-muscle strength ratio, which

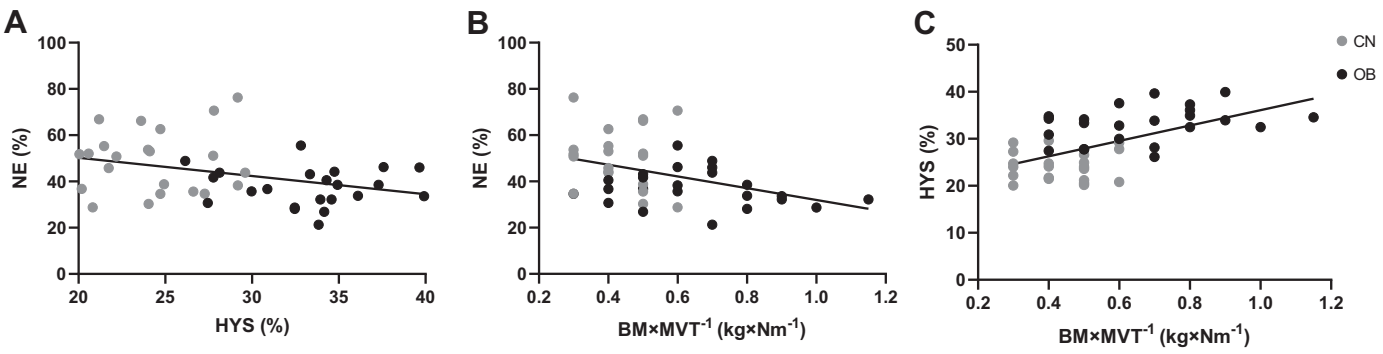

Figure 9. Scatter plots representing correlation between calf-raise test net efficiency (NE) and muscle-tendon-units' hysteresis (HYS) from the plantar flexors stretch-destretch cycle (*A*); NE and body mass divided by maximal voluntary isometric torque (BM × MVT^{-1}) (*B*); and HYS and (BM × MVT^{-1}) (*C*), of pooled data from control (CN, black dots) and obese (OB, gray dots) groups.

results in the muscle operating at a less efficient part of the force-velocity relationship, and *2*) greater hysteresis of the triceps surae MTU, potentially limiting the recovery of stored elastic energy. These findings highlight the potential role of musculotendinous properties, including higher stiffness and hysteresis, in limited joint mobility and the higher metabolic energy cost of movement in obesity.

GLOSSARY

ANOVA	Analysis of variance
AT	Achilles tendon
BMI	Body mass index
CN	Individual with normal weight
CRT	Calf raise test
CSA	Cross sectional area
D	Displacement
MCE	Metabolic cost of exercise
EE	Energy expenditure
EPOC	Excess post-exercise oxygen consumption
GL	Gastrocnemius lateralis
GM	Gastrocnemius medialis
HR	Heart rate
MT	Muscle thickness
MTU	Muscle tendon unit
MTJ	Musculo-tendinous junction
MVT	Maximal voluntary torque
NE	Net efficiency
OB	Individuals with obesity
OTJ	Osteo-tendinous junction
PRT	Passive resistive torque
RER	Respiratory exchange ratio
ROM	Range of motion
RPE	Rate of perceived exertion
RTD	Rate of torque development
SOL	Soleus

DATA AVAILABILITY

The data that support the findings of this study are available upon request from the corresponding author.

SUPPLEMENTAL MATERIAL

Supplemental Table S1: https://doi.org/10.17605/OSF.IO/4KFXG.

Supplemental Table S2: https://doi.org/10.17605/OSF.IO/4KFXG.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

L.C. and D.S. conceived and designed research; L.C., C.R.T., and D.S. performed experiments; L.C., H.D., and D.S. analyzed data; L.C., H.D., S.K., and D.S. interpreted results of experiments; L.C. prepared figures; L.C., H.D., and D.S. drafted manuscript; L.C., H.D., S.K., and D.S. edited and revised manuscript; L.C., H.D., C.R.T., S.K., and D.S. approved final version of manuscript.

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