European Journal of Applied Physiology
https://doi.org/10.1007/s00421-026-06491-4

ORIGINAL ARTICLE

Neural, contractile, and muscular determinants associated with lower strength in recreationally active older men: a cross-sectional study

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Received: 26 June 2026 / Accepted: 15 October 2026
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Abstract

Skeletal muscle strength declines with age. However, the relative contributions of various neuromuscular factors to this decline appear to not be fully understood. Thus, we compared relative attenuations in key neural and muscular strength determinants with age, in vivo and in vitro, recruiting 22 old (75 ± 1 years) and 18 young (25 ± 1 years) recreationally active men. Leg extension maximal voluntary contraction (MVC), early (0-100 ms) and late (0-200 ms) rate of force development (RFD), were reduced by −39%, −55%, and −49%, respectively, in the old (all $p < 0.001$); however, estimated thigh cross-sectional area (CSA) was not different between groups. In contrast, efferent neural drive assessed by evoked recordings (m.soleus normalized V-waves; V/M_{sup}), largely dependent on corticospinal factors, revealed a −72% age-related reduction ($p < 0.001$). A −51% reduction was also observed in peripheral motoneuron excitability, measured as H_{max}/M_{max} at 10% MVC ($p < 0.001$). The neural age-related decline was confirmed applying peak electromyography (EMG; −14%), early (−31%), and late (−30%; all $p < 0.05$) rate of EMG rise, during leg extension MVC. While voluntary activation was unaltered with age, quadriceps contractility was reduced (resting twitch: peak force, −41%; RFD, −43%; delay, +39%; time-to-relaxation, +56%; all $p < 0.01$). Biopsies from m.vastus lateralis showed an age-related decrease in proportion, −16% (type II: 43% (old) vs. 51% (young); $p < 0.001$), but not size, of fast twitch fibers. Concomitantly, slow twitch fibers were 12% larger in old vs. young ($p < 0.05$). Collectively, these findings showed that age-related reductions in muscle strength were accompanied by pronounced alterations in neural function alongside changes in muscle phenotype, despite preserved thigh CSA.

Keywords Dynapenia · Sarcopenia · Aging · Ageing · Muscle strength · Neuromuscular · Efferent neural drive · Muscle fibers

Introduction

Maximal muscle strength typically declines with 1–2% per year from the fifth decade of life, and this accelerates to ~3% per year from ~70 years of age (Delmonico et al. 2009). Muscle strength is determined by the integrated capacities of every single step from the brain to muscle fibers to produce force, as well as through returning afferent feedback. Historically, the decline in muscle strength has predominantly been attributed to a loss of muscle mass, or sarcopenia (Clegg et al. 2013). However, maximal muscle strength is lost at a rate 2–5 times higher than muscle mass (Bruce et al. 1997), pointing towards factors other than muscle mass to be largely responsible for the age-related decline (Clark et al. 2013; Goodpaster et al. 2006; Reid et al. 2014). Importantly, there remains uncertainty of what the relative contribution of key factors along the neuromuscular pathway to the age-related decline in muscle strength may be. Particularly since various neuromuscular measures typically are obtained from different populations, in different investigations. A more complete understanding of neural and muscular relative contributions could have fundamental importance when aiming to design tailored interventions to counteract the age-related decrease in muscle strength.

Total muscle cross-sectional area (CSA) has an estimated annual loss of 0.3–0.5% from the 5th decade (Borkan et

Communicated by William J. Kraemer.

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Published online: 31 October 2026

al. 1983; Wroblewski et al. 2011), advancing to ~ 0.5–1% from the 7th decade of life (Frontera et al. 2000; Mitchell et al. 2012). Although the loss of muscle mass may be considered relatively modest, myofiber alterations, in particular atrophy of the fast-twitch type II fibers, may be more pronounced (Lexell 1993, 1995; Nilwik et al. 2013). Specifically, whereas the size of slow-twitch type I fibers may largely remain unaltered with aging (Mcphee et al. 2018), in contrast type II fiber size may be reduced by ~30% in old vs. young males, yielding a fiber type II proportion decline from 56 to 46% (Nilwik et al. 2013). In return, fiber type I proportion increases reciprocally, impacting the capacity of the muscle to produce force. However, of importance, these alterations in muscle morphology may be greatly influenced by the nervous system. As the aging muscle undergoes a series of denervation-reinnervation cycles (Piasecki et al. 2016; Sonjak et al. 2019), with a potential loss of the largest axons, a remodeling of motor units may occur and result in a muscle bed with more muscle fiber type I-like properties (Hepple and Rice 2016; Toien et al. 2023a).

Interestingly, the loss of muscle strength with age appears to be mirrored in the muscle's ability to produce force rapidly (rate of force development; RFD) (Olmos et al. 2019). RFD is strongly associated with the function of the nervous system, warranting a closer look into efferent and afferent neural signals between motor cortex and muscle. Factors such as lower corticospinal excitability, α-motoneuron loss affecting activation and firing frequency, reduced synaptic efficacy and nerve conduction velocity, may all limit the nervous system's ability to produce strong, rapid muscle contractions (Manini et al. 2013; Sale and Semmler 2005; Aagaard et al. 2010). Indeed, a number of studies suggest that attenuated efferent neural drive, i.e., the integrated ability to modulate α-motoneuron recruitment and firing frequency, may play a major role of muscle strength decline with age (Toien et al. 2023b; Unhjem et al. 2016; Aagaard et al. 2002b). In fact, descending efferent neural drive during maximal muscle contractions, assessed with V-wave measurements in the soleus muscle, is shown to be ~70% lower in recreationally active older men compared to young (Unhjem et al. 2016). Similar findings have been revealed when using surface electromyography (EMG) to quantify the nervous system-generated activation signal for maximal voluntary muscle actions, e.g., peak EMG is shown to be ~40–50% lower in older men during MVC when compared to young (Deschenes et al. 2008; Marshall et al. 2020). Notably, rate of EMG rise (RER), particularly in the functionally important early phase (0–100 ms) of contractions, may also be greatly impacted, with 50–70% lower values reported (Jenkins et al. 2014; Toien et al. 2022; Varesco et al. 2019), albeit substantial variance is associated with this methodology and normalization procedures (De Luca 1997). Although it is difficult to determine which steps in the neuromuscular pathway contribute to the attenuation in efferent neural drive with advancing age, it is likely, at least in part, due to corticospinal factors since peripheral reflex arc-induced α-motoneuron excitability may appear to exhibit more modest age-related decline (Scaglioni et al. 2003).

Although there are indications that neural factors exhibit larger relative age-related differences than muscular factors, a comparison from different populations is challenging because of aspects such as training status, age, sex, as well as many other demographic factors. Thus, a proportionate comparison of the differences in key neural and muscular strength factors in the same individuals may expand the current body of evidence. Acknowledging the clinical importance of lower extremity muscle strength to longevity, mechanistic insight into how alterations are manifested may not only be important in itself but also help to reveal which factors should be targeted in medical interventions. Therefore, this cross-sectional study aimed to compare the age-related differences in neural and muscular strength factors in recreationally active old men with young counterparts. Specifically, we hypothesized that: (1) lower extremity force generating capacity would be lower in the older men compared to young, and (2) accompanied by the largest relative differences (%) in neural (efferent neural drive [V/M_{sup}], motoneuron excitability [H_{max}/M_{max}, H_{sup}/M_{sup}], voluntary activation, EMG amplitude, and RER) rather than muscular (muscle CSA, type II fiber proportion and area, quadriceps resting twitch) factors.

Methods

Participants

A total of 40, 22 old (>65 yrs) and 18 young (<35 yrs) healthy, recreationally active men were recruited for this cross-sectional study (Table 1). Older participants were recruited through senior society clubs and young controls from a university. Activity levels for all participants were confirmed using the international physical activity questionnaire (IPAQ), with both old and young participants reporting physical activity to be unsystematic and typically done 2–4 times per week, in various forms, i.e. skiing, hiking. No strength training was reported in the included participants. Participants were excluded if they had reported any cardiovascular, neurological, musculoskeletal, or pulmonary diseases. The study was approved by the regional ethics committee (REC number: 2018/1207) and all measures conducted in accordance with the declaration of Helsinki.

Table 1 Participant characteristics

	Young (n = 18)	Old (n = 22)
Age (years)	25 ± 1	75 ± 1**
Height (cm)	180 ± 1	179 ± 2
Body mass (kg)	77.9 ± 2.1	82.0 ± 2.0
Weekly physical activity level (MET min week^{-1})	2838 ± 1381	2945 ± 1754

Data presented as mean ± standard deviation

**$p < 0.01$ compared to young

Participants provided written informed consent before data was collected.

Study outline

In this cross-sectional study, participants completed all tests on two separate occasions. Detailed instructions, familiarization, and warm-up procedures were provided before tests were carried out. On the first occasion, anthropometry was measured before progressing to evoked reflex recordings (H-reflex and V-wave) during isometric plantar flexion. Afterwards, isometric leg extension muscle strength (maximal strength (MVC) and RFD) was measured along with electromyography (EMG), before resting twitch and voluntary activation procedures were conducted in the same apparatus. On a second visit to the lab, vastus lateralis muscle biopsy sampling was carried out.

Anthropometry

The height and body mass of all participants were measured in the lab before progressing to thigh muscle. Estimated muscle volume for the self-selected dominant thigh was calculated and measured using measuring tape and skinfold caliper (Holtain, Crosswell, UK). Thigh length was measured between the lateral femoral epicondyle and the greater trochanter. Circumference of the thigh was measured at three points: the midpoint, and 10 cm both proximally and distally. Skinfold thickness was recorded at three locations on the midpoint of the thigh: medial, anterior, and lateral. Measurements were taken 3 times each with an average being used for the measurement. The values from each site were then used in the following equation to estimate thigh muscle volume:

$$Thigh\ Muscle\ Vol. = \left(\frac{L}{12\pi}\right) x\left(C1^2 + C2^2 + C3^2\right) - \left(\frac{(S-0.4)}{2}\right) \times L \times \left(\frac{(C1+C2+C3)}{3}\right)$$

where L = thigh length, C = thigh circumference at each measurement point and S = mean skinfold thickness across each measurement point. This method has been validated with acceptable accuracy when compared to proton magnetic resonance imaging (Layec et al. 2014). Finally, the volume was divided by the length of the thigh to obtain cross-sectional area.

Evoked potentials

Evoked H-reflex and V-wave responses were taken from the soleus muscle during both low force and maximal voluntary contractions (MVC) (Aagaard et al. 2002a, b). First, to ensure minimal skin resistance, the skin was prepped by shaving, abrading (Nuprep, Weaver and company, Aurora, CO), and cleansing with alcohol, until conductive resistance was ≤5 kΩ. Using anatomical landmarks, muscle architecture and following SENIAM guidelines (Hermens et al. 2000), self-adhesive bipolar Ag/AgCl electrodes (Ambu, M-00-S/50, Ballerup, Denmark) with an interelectrode distance of 25 mm were placed on the soleus muscle surface. These electrodes were connected to a ME6000 Biomonitor (Bittium Biosignals, Kuopio, Finland) to collect data with a 2 kHz A/D sampling rate and a common mode rejection ratio of 110 dB.

Participants were seated in a custom built isometric plantar flexion apparatus with a load cell (620-1000M-F, Vishay Tedea Huntleigh, Israel). Stimulation was applied to the tibial nerve located in the popliteal fossa, until the location with the highest H-reflex and smallest M-wave was located. Intensity was then increased by 2–5 mA to identify the maximal H-reflex amplitude (H_{max}), during low force contraction (~10% MVC). Following this, participants were instructed to contract their plantar flexors as hard as they could, while being given the same stimulus as H_{max} to obtain H-reflex during maximal contractions (H_{sup}). Afterwards, the stimulus intensity was further increased to detect the maximal peak-to-peak amplitude (M_{max}) during ~ 10% MVC. V-waves were evoked during MVC by using 150% of the stimulation intensity used to evoke M_{max}, for 4–6 V-wave recordings. The maximum peak-to-peak amplitude of the V-wave was normalized relative to the highest M-wave amplitude (M_{sup}) recorded during MVC, expressed as V/M_{sup}. For inclusion in the analysis, the M-wave amplitude needed to be ≥ 95% of M_{sup}, and the plantar flexor force had to reach ≥ 90% of MVC. Additionally, the peak-to-peak amplitude of H_{max} was normalized to M_{max} (H_{max}/M_{max}), and both the H_{sup} and V-wave amplitudes were normalized to the maximal M-wave recorded during the V-wave trials (H_{sup}/M_{sup}, V/M_{sup}).

Muscle strength and electromyography

MVC and RFD were tested in the self-selected dominant leg, with participants seated in a custom-built, rigid isometric leg extension apparatus with their knees and hips flexed at a 90° angle, including fastenings to reduce countermovement. Following the same skin preparation procedures, bipolar Ag/AgCl electrodes were placed on the vastus lateralis muscle, using Surface EMG for Non-Invasive Assessment of Muscles (SENIAM) guidelines (Hermens et al. 2000), and connected to the ME6000 Biomonitor. To minimize movement, the participants were strapped down at the chest and thigh, ensuring that the upper body remained firmly fixed in place throughout the testing. The lower leg was secured to a lever connected to the same load cell (620-1000M-F, Vishay Tedea Huntleigh, Israel) as used during isometric plantar flexion, and a reference point was provided to minimize head movement. Participants were instructed to utilize the handles on each side of the leg extension apparatus. The force analyses were conducted using MegaWin 3.1.2.13 software (Mega Electronics Ltd., Kuopio, Finland).

To warm up, participants performed 10–15 isometric contractions by attempting leg extensions without exerting full force. After warming up, participants were instructed to attempt to extend their leg "as hard and fast as possible". The participants had 3 attempts, interspersed with 3 min of recovery, to produce the highest force possible. To determine MVC and RFD, the test with the highest peak force was used and analysed. The onset of force production was determined by manual inspection by the same investigator and set as the initial deviation from the baseline value. RFD was calculated as change in force during early (0–100 ms) and late (0–200 ms) phases, with 0 ms representing onset of force production.

The test with the highest peak force during isometric leg extension MVC was used to analyse EMG in the vastus lateralis muscle. The recorded EMG signals were amplified and filtered with a band-pass range of 8–500 Hz. Average root mean square was used to analyse the EMG signals. The onset of EMG was inspected manually and set as the deviation from baseline, irrespective of onset of force production. RER was calculated as change in EMG during early (0–100 ms) and late (0–200 ms) phases, with 0 ms being onset of EMG rise. Delay was determined by the time from EMG to force onset.

Resting twitch and voluntary activation

After muscle strength and EMG measurements, participants progressed to resting twitch (RT) procedures. Seated in the same isometric leg extension apparatus, evoked potentials were obtained from the quadriceps femoris muscle of the same leg. The same current stimulator (DS7AH, Digitimer, Welwyn Garden City, UK) and bipolar felt pad electrodes (Digitimer), as used for V-wave and H-reflex measurements, delivered a 1 ms square wave stimulus percutaneously to the femoral nerve in the groin region. Initially, the position that elicited the largest M-wave was identified, after which the stimulus intensity was progressively increased by 5–10 mA to determine M_{max}. Applying the same stimulus intensity, detection of the RT was performed within 4–6 attempts, in an unpotentiated state. The RT with the highest peak force was used for analysis. Delay, time to peak force, and time to relaxation were determined by the time from stimulus to force onset; force onset to peak; and peak force to relaxation, respectively.

Voluntary activation of the quadriceps was assessed through the interpolated twitch technique. Two–three isometric leg extension MVCs were performed, with 2–3 min rest in between. During these, a 1 ms single square wave supramaximal stimulus (150% of the current required to evoke M_{max}) was delivered to the femoral nerve once a plateau in force was observed. Approximately 3 s after MVC, another stimulus of the same intensity was applied again, to determine the RT in a potentiated state. Voluntary activation was calculated as $(1 - (T / RT)) \times 100$ and expressed as a percentage.

Muscle biopsy sampling, preparation, and analysis

Muscle biopsies were collected from the vastus lateralis approximately 10–14 days after the physical tests. Participants were instructed to refrain from alcohol and strenuous exercise 48 h prior to the biopsy. After administering a local anesthetic (1% xylocaine), a 6-mm Bergstrom needle attached to a suction syringe was used to obtain the biopsies, with samples extracted 3.5 cm deep, approximately 15 cm proximal to the knee and slightly distal to the ventral midline (Richardson et al. 2000). Tissue samples were immediately frozen in liquid nitrogen and stored at −80 °C for further processing. For immunohistochemical analysis, 8–10 µm transverse serial sections were cut using a cryostat and mounted on glass slides. To identify muscle fiber types, the sections were treated with primary antibodies BA-F8 1:50 (type I fibers), SC-71 1:250 (type IIa fibers) and 6H1 1:25 (type IIx fibers), as well as Z0097 1:1000 to visualize membrane perimeter (antibodies: DAKO). After applying these, secondary antibodies (A21240, A21140, AB_2535711, and A21206; Thermo Fisher Scientific Inc., Waltham, MA; USA) were used. Primary and secondary antibody steps were incubated for 1 h at room temperature, interspersed by wash procedures. The stained sections were mounted with mounting medium (H5501; Vector). The sample images were captured under microscope (Carl Zeiss Axio Imager

M1, Germany) and a high resolution AxioCam (Carl Zeiss, Germany) using the ×10 objective and standardized exposures between images. Fiber number and cross-sectional area were analysed using AxioVision (v. 4.6, Carl Zeiss). On average, 748 ± 58 myofibers were analysed per biopsy for fiber type distribution, and 144 ± 21 for fiber area.

Statistical analyses

Statistical analyses were performed using SPSS Statistics 29.0 (IBM, Chicago, IL, USA). Figures were made using Graphpad Prism 10 (San Diego, CA, USA). All data were assessed for normality using Shapiro–Wilk's test for normality. MVC and RFD were considered primary outcomes a priori. All other neural to muscular strength determinants were considered secondary outcomes. Independent t-tests were used to detect differences between groups. A two-tailed p value of < 0.05 was considered statistically significant and < 0.1 considered a tendency. Simple linear regression analyses were performed as exploratory to examine associations between MVC and RFD and the strength determinants. In addition, all regression analyses should be considered exploratory outcomes. A priori sample-size calculation from MVC results indicated that 20 participants per group (40 total) were required to detect an intergroup difference in primary outcome with an effect size of $d = 1.20$ with 95% power at $\alpha = 0.05$ (two-tailed) (Izquierdo et al. 1999; Ling et al. 2009; Roos et al. 1999). Data in tables and figures were presented as mean ± standard error unless otherwise stated.

Results

Testing compliance

All participants completed the full testing protocol with the exception of muscle biopsies, which were obtained from the majority of older (18) and younger (11) participants. Apart from age, there were no differences in participant characteristics or physical activity levels between groups (Table 1). A number of individuals chose not to undergo the biopsy primarily due to discomfort during the procedure. Participants who underwent muscle biopsy did not differ significantly from those who declined with respect to age, body mass, estimated thigh CSA, MVC, or RFD in either the younger or older group (all $p > 0.05$; Supplementary Table 1).

Muscle strength and cross-sectional area

Older participants demonstrated 39% lower maximal strength during the isometric leg extension MVC compared to the younger participants (mean difference = −196 N, 95% CI −258 to −133 N, Hedges' $g = 1.99$; Fig. 1A). Additionally, MVC normalized to estimated thigh CSA (−39%) and thigh volume (−42%) were lower in the older participants (Hedges' $g = 1.66$ and 1.54, respectively). However, no difference in estimated thigh CSA was observed between the younger and older participants (mean difference = −6.5 cm^2, 95% CI −18.7 to 5.6 cm^2, Hedges' $g = 0.34$; Fig. 1B). Concomitantly, RFD was 55% and 49% lower during the early and late phases, respectively, in the older participants (95% CI −1328 to −355 N s^{-1}, Hedges' $g = 1.09$; and 95% CI −1171 to −407 N s^{-1}, Hedges' $g = 1.30$; Fig. 2).

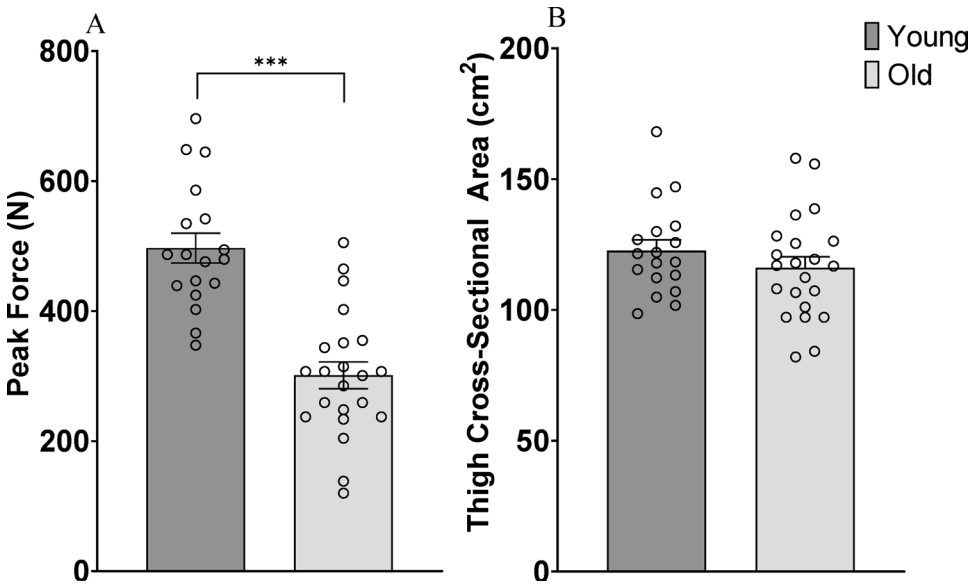


Fig. 1 **A** Peak force obtained from isometric leg extension maximum voluntary contraction, and **B** thigh cross-sectional area. Data presented as mean and SE. ***$p < 0.001$ between groups

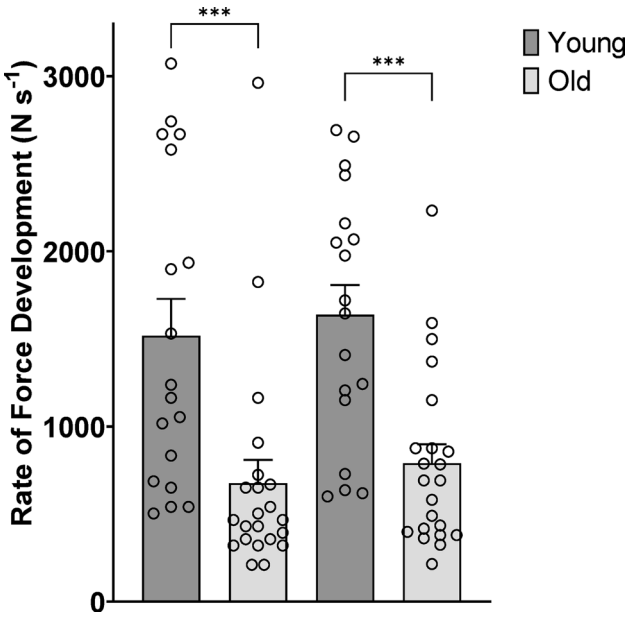


Fig. 2 Comparison of early (0–100 ms) and late (0–200 ms) rate of force development during isometric leg extension maximal voluntary contraction in young and older recreationally active subjects. Data presented as mean and SE. ***$p < 0.001$ between groups

Table 2 Efferent neural drive and α-motoneuron excitability from m. soleus during plantar flexor torque

	Young ($n = 18$)	Old ($n = 22$)
V-wave (μV)	1596 ± 910	500 ± 292***
V/M_{sup}	0.29 ± 0.15	0.08 ± 0.03***
H_{max}/M_{max}	0.36 ± 0.18	0.18 ± 0.15***
H_{sup}/M_{sup}	0.36 ± 0.26	0.14 ± 0.11***
M_{max} (μV)	6391 ± 2684	5879 ± 2642
Latency (ms)		
M-wave	6.7 ± 1.7	8.6 ± 1.0***
H-reflex	33.2 ± 2.4	41.4 ± 4.4***

Data presented as mean ± standard deviation

V maximal V-wave amplitude during maximal voluntary contraction (MVC), M_{sup} maximal M-wave amplitude during MVC, H_{max} maximal H-reflex amplitude during 10% MVC, M_{max} maximal M-wave amplitude during 10% MVC, H_{sup} maximal H-reflex amplitude during MVC

***$p < 0.001$ compared to young

Efferent neural drive and α-motoneuron excitability

The older participants demonstrated lower V-wave (95% CI −1511 to −680 μV, Hedges' $g = 1.66$) and V/Msup (95% CI −0.275 to −0.144, Hedges' $g = 2.02$) compared to the younger participants (Table 2; Fig. 3) during isometric plantar flexion. H_{max}/M_{max} (95% CI −0.284 to −0.078, Hedges' $g = 1.11$) and H_{sup}/M_{sup} (95% CI −0.343 to −0.101, Hedges' $g = 1.16$) were also lower in the older participants. Likewise, measures of conduction velocity showed longer latency in both the H-reflex (95% CI 5.87 to 10.55 ms, Hedges' $g = 2.21$) and M-wave responses (95% CI 0.96 to 2.71 ms, Hedges' $g = 1.32$).

Electromyography

Peak EMG was lower in the older participants compared to the younger participants (Hedges' $g = 0.47$; Table 3). In addition, early (0–100 ms) and late (0–200 ms) RER were lower (95% CI −643 to −37 μV s^{-1}, Hedges' $g = 0.71$; and 95% CI −494 to −49 μV s^{-1}, Hedges' $g = 0.77$, respectively), in combination with a longer delay ($p < 0.001$; 95% CI 9.1 to 41.5 ms, Hedges' $g = 0.99$) in the older participants compared to the younger participants.

Muscle contractility and voluntary activation

Isometric leg extension RT revealed lower peak force (95% CI −29.6 to −10.6 N, Hedges' $g = 1.33$) and RFD (95% CI −713 to −255 N s^{-1}, Hedges' $g = 1.33$) in the older participants compared to the younger participants. This was accompanied by longer twitch delay (95% CI 3.9 to 10.4 ms, Hedges' $g = 1.39$) and time to relaxation (95% CI 13.8 to 125.3 ms, Hedges' $g = 0.79$), but not time to peak force. Quadriceps voluntary activation also showed a tendency ($p = 0.063$) to be attenuated in the older participants (Hedges' $g = 0.53$).

Muscle fiber characteristics

As documented in Table 4, total area distribution (%) was smaller for type II fibers (95% CI −12.0 to −3.0%, Hedges' $g = 1.27$) and larger for type I fibers (95% CI 3.0 to 12.0%, Hedges' $g = 1.27$) in the older participants compared to the younger participants. Whereas the count distribution (%) of type IIa fibers was lower (Hedges' $g = 0.73$), type IIx fibers tended to be lower ($p = 0.075$; Hedges' $g = 0.61$), while type I fibers were higher (Hedges' $g = 0.91$) in the older participants. This was accompanied by a larger average type I fiber area in the older participants (Hedges' $g = 0.82$), whereas no differences were observed in average type II fiber size.

Association and regression analysis

Exploratory regression analyses revealed that several neural and contractile measures were positively associated with both MVC and both early and late RFD. Specifically, V/M_{sup} and rate of EMG rise were associated with both MVC

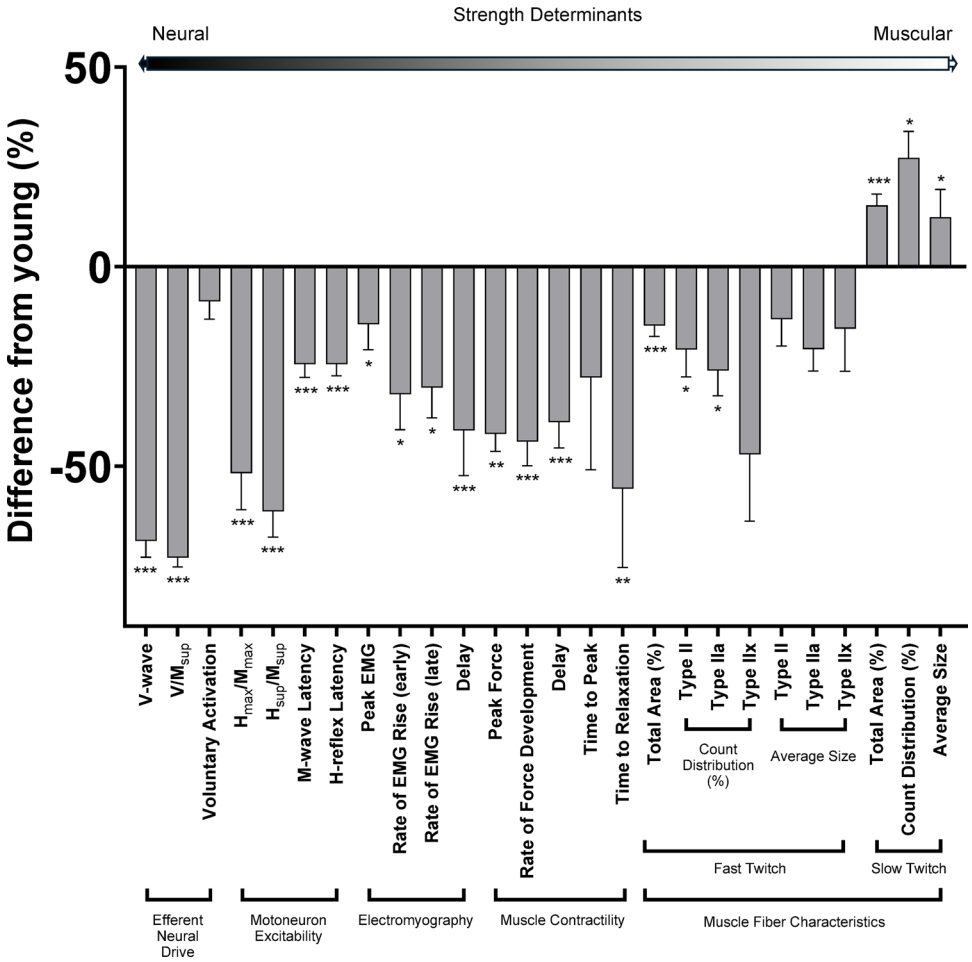


Fig. 3 Neural to muscular strength determinants in older recreationally active participants, presented as percentage difference from young. Data are mean and SE. *V* maximal V-wave amplitude during maximal voluntary contraction (MVC), *M-sup* maximal M-wave amplitude during MVC, *Hmax* maximal H-reflex amplitude during 10% MVC, *Mmax* maximal M-wave amplitude during 10% MVC, *Hsup* maximal H-reflex amplitude during MVC, *EMG* electromyography; rate of EMG rise early (0–100 ms) and late (0–200 ms) phase. EMG-delay and resting twitch (time to peak, delay, and time to relaxation) are presented as inverted values to illustrate decreased performance. *$p < 0.05$, **$p < 0.01$, and ***$p < 0.001$

(V/M_{sup}: $R^2 = 0.381$; RER: $R^2 = 0.166$–0.209, all $p < 0.05$) and early/late RFD (V/M_{sup}: $R^2 = 0.108$; RER: $R^2 = 0.161$–0.249, all $p < 0.05$). MVC was associated with H-reflex amplitudes (H_{max}/M_{max}: $R^2 = 0.149$, $p = 0.014$; H_{sup}/M_{sup}: $R^2 = 0.202$, $p = 0.004$), voluntary activation ($R^2 = 0.118$, $p = 0.030$), peak EMG, whole-muscle contractility (peak force, RFD, and delay), and type II fiber area ($R^2 = 0.230$, $p = 0.007$). Conversely, early and late RFD were associated with estimated thigh CSA ($R^2 = 0.138$–0.184, $p < 0.02$), which lacked a significant association with MVC, along with twitch force ($R^2 = 0.193$–0.225, $p \leq 0.003$), twitch RFD ($R^2 = 0.283$–0.295, $p < 0.001$), and inversely with EMG delay ($R^2 = 0.120$–0.150, $p < 0.03$). Most individual fiber number and size variables were not significantly associated with either MVC or RFD measures (Supplementary Tables 2–4). Additionally, age group remained independely associated with MVC while accounting for estimated thigh CSA, whereas CSA was not independently associated with MVC (Supplementary Table 5).

Table 3 Electromyography in m. vastus lateralis during maximal voluntary contraction, m. quadriceps voluntary activation, and contractility (resting twitch) during isometric leg extension

	Young ($n = 18$)	Old ($n = 22$)
Electromyography		
Peak EMG (μV)	467 ± 148	398 ± 139*
Rate of EMG rise (μV s^{-1})		
Early (0–100 ms)	1099 ± 478	759 ± 467*
Late (0–200 ms)	896 ± 375	625 ± 319*
Delay (ms)	59.6 ± 16.2	84.9 ± 30.5***
Voluntary activation (%)	88 ± 11	80 ± 17
Resting twitch		
Peak force (N)	51.8 ± 18.3	31.7 ± 11.2**
Rate of force development (N s^{-1})	1128 ± 389	644 ± 326***
Delay (ms)	19.8 ± 3.7	26.9 ± 5.9***
Time to peak force (ms)	47.6 ± 11.1	62.5 ± 53.1
Time to relaxation (ms)	113.7 ± 50.7	183.2 ± 108.6**

Data presented as mean ± standard deviation
EMG electromyography, *Delay* either from onset of EMG or stimulation (resting twitch) to onset of force
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ compared to young

Table 4 Muscle fiber characteristics in m. vastus lateralis

	Young (n = 11)	Old (n = 18)
Total area (%)		
Type II	51 ± 5	43 ± 6***
Type I	49 ± 5	57 ± 6***
Count distribution (%)		
Type II	51 ± 14	38 ± 14*
Type IIa	42 ± 10	33 ± 13*
Type IIx	9 ± 9	5 ± 6
Type I	49 ± 14	62 ± 14*
Average size (μm^2)		
Type II	4644 ± 1204	4033 ± 1364
Type IIa	5162 ± 1032	4095 ± 1139
Type IIx	4127 ± 1127	3691 ± 1708
Type I	4439 ± 1135	4990 ± 1345*

Data presented as mean ± standard deviation

*$p < 0.05$, ***$p < 0.001$ compared to young

Discussion

Aging leads to a decline in skeletal muscle strength, yet the relative contributions of factors along the neuromuscular pathway are still inadequately understood. This cross-sectional study aimed to examine lower extremity force generating capacity, along with a comprehensive battery of in vivo and in vitro neural and muscular measures in older and younger men in order to better understand the relative differences in these key muscle strength determinants. The main findings were: (1) Mean MVC force was 39% lower in old compared to young, accompanied by lower early and late RFD of 55% and 49%, respectively; (2) Accordingly, efferent neural drive from brain to muscle (V/M_{sup}) exhibited an age-related difference of 72%, confirmed by peak EMG (14%) and RER (~30%) differences; (3) The neural age-related difference was in part due to a 51–61% lower peripheral motoneuron excitability (H_{max}/M_{max}, H_{sup}/M_{sup}); (4) No age-related difference in estimated thigh CSA, traditionally termed sarcopenia, was observed; (5) A closer look into the muscle morphology revealed that, despite the similar estimated thigh CSA, fast twitch fiber proportion was lower in old vs. young by 16%, (43% vs. 51%), concomitantly the old had a 12% larger slow twitch fiber size; 6) Finally, while quadriceps voluntary activation only tended to be impaired in the old, muscle contractility was attenuated (resting twitch peak force, 41%; RFD, 43%; delay, +39%, time to relaxation, +56%). Collectively, in physically active individuals, the results showed that neural and muscle phenotype characteristics were among the age-related differences observed in the present study, despite no significant difference in estimated thigh CSA.

Maximal strength, rate of force development, and age

As expected, the old displayed lower MVC and early and late RFD compared to young, i.e., 0.8% and ~1% difference per year respectively, taking into account the 50-year age gap. Although muscle strength declines more rapidly after the 5th decade of life, these differences are on par with what is typically observed with advancing age (Delmonico et al. 2009). Considering the close relationship between RFD and neural factors, in particular α-motoneuron firing frequency (Christie and Kamen 2010), the somewhat larger differences observed in RFD in the older participants point towards an important role of neural mechanisms in the muscle strength loss with aging.

Muscle strength, neural deterioration, and age

Perhaps the most striking age-related difference in the current study is the substantially reduced efferent neural drive to the maximally contracting musculature. The 72% lower V/M_{sup} in older individuals in the current study is on par with what previously has been reported (Unhjem et al. 2015, 2016). The advantage of the V/M_{sup} response is that it can be measured during strong muscle contractions and MVCs and the technique has returned very consistent results, related to muscle strength measurements (Unhjem et al. 2016; Vila-Cha et al. 2012). By experimental design the V/M_{sup} response not only relies to some extent upon corticospinal factors, but is also generally regarded as sufficiently sensitive to capture potential differences in motoneuron firing frequency and/or recruitment (Aagaard et al. 2002b). This is because efferent output from brain to motoneuron axons cancel out antidromically propagating action potentials triggered by the supramaximal electrical stimulus, allowing the reflex potential to transmit via the cleared neurons to the muscle. Crucially, in particular, maximal firing frequency has previously been shown to be lower by 30–40% in old compared to young (Christie and Kamen 2010; Kamen and Knight 2004), and possibly account for a great portion of the observed age-related attenuated efferent neural drive. Indeed, in support of this, positive associations between MVC and V/M_{sup} were shown ($R^2 = 0.38$), reinforcing that efferent neural drive may be closely related to maximal force output.

Reinforcing the assumption of an age-related reduction in efferent neural drive is the impaired muscle activation in the old, expressed as reduced peak EMG (14%) and early and late RER (~30%). Indeed, declines in maximal firing

frequency would be expected to contribute to reductions in EMG and RER, as close relationships between these indices and firing frequency, particularly in the early phase of strong muscle contractions, is previously established (De Luca 1997; Aagaard et al. 2002a). However, the differences in EMG and RER appear to be of a lower relative size than efferent neural drive assessed as $V/M_{sup,}$ but still remaining positiviely associated with MVC. Similar findings of reduced EMG have been reported in previous studies (Clark et al. 2013; Reid et al. 2014), though to a lesser extent in others (Clark et al. 2010, 2011). Caution should be used when interpreting EMG findings, as inconsistent results are often yielded, possibly due to influence from background electrical activity as well as methodological variance, such as electrode placement, signal filtering and peripheral tissue properties (Clark et al. 2010; Enoka and Duchateau 2015). Nevertheless, the reduction in muscle activation in the old corroborates and extends the observation of reduced efferent neural drive with age.

While not statistically significant, the results revealed a tendency ($p = 0.06$) for voluntary activation to be lower in the old (88% vs. 80%) compared to young. Previous observations on age-related changes in voluntary activation have been conflicting (Harridge et al. 1999; Phillips et al. 1992; Scaglioni et al. 2002), which may be caused by the fact that different age groups and muscle groups have been examined. Although this observation should be interpreted with caution, a somewhat lower voluntary activation may potentially indicate lower motoneuron firing frequency and/or recruitment (Aagaard et al. 2002b).

Motoneuron excitability and age

H_{max}/M_{max} and H_{sup}/M_{sup} age-differences of 51% and 61%, respectively, in the current study suggests impaired motoneuron excitability in the old, in line with previous investigations (Kido et al. 2004; Scaglioni et al. 2003; Unhjem et al. 2016). Albeit, a direct comparison of transmission efficacy along the Ia reflex arc between different age groups may be challenging, because the H reflex predominantly manifests in type I fibers, of which the old group had a higher proportion and larger size compared to young. However, recognizing the rather large age-related differences, the current results suggest that the lower H/M ratio in old, at least in part, is due to a lower number, or proportion, of motoneurons recruited. This implies that a stronger input must be delivered to the motoneurons to generate an equal output to the muscle. Suppressed H reflex responses may be caused by a number of neural adaptive rearrangements as a consequence of senescence. Specifically, atrophy and loss of the largest afferent neurons, related to denervation and reinnervation, likely play a key role (Ochoa and Mair 1969; Rao and Krinke 1983), making it challenging to elicit a large H-reflex response without concurrently eliciting an antidromic signal. Converging excitability thresholds between afferent and efferent fibers due to a reduction in the diameter of Ia afferent fibers has also been proposed to reduce excitability (Scaglioni et al. 2003).

Notably, the 25% greater H reflex latency observed in the older men, in line with previous studies (Kido et al. 2004; Scaglioni et al. 2002), has, in the absence of alterations in M wave latency, put forward the assumption that the afferent pathway may be preferentially impaired in age-related neural degeneration. However, interestingly, a greater M wave latency of 28% was documented in the old group which, to the best of our knowledge, has only been reported in quadriceps previously (Mau-Moeller et al. 2013). Although the contribution of interneuronal activity mediating presynaptic inhibition, or oligosynaptic modulation, cannot be discounted (Burke et al. 1984), the increased H and M latencies suggests not only that conduction velocity along the reflex arc is impaired, but also an age-related systemic neural degeneration affecting both afferent and efferent pathways to contracting human muscle.

Muscle strength, muscular deterioration, and age

Importantly, in the current study, estimated thigh CSA in the older men was not different compared to the young, which contrasts with previous studies (Borkan et al. 1983; Frontera et al. 2000). Although the extent to which muscle mass decreases with aging is subject to substantial variation due to which muscles are examined, equipment utilized, and groups included (Mitchell et al. 2012), the present findings may be interpreted with caution since anthropometric estimates alone may be less sensitive to changes in regional muscle loss, intramuscular fat infiltration or alterations to morphology. Although imaging techniques may provide a direct assessment and could capture such subtle differences more accurately, the present anthropometry leg muscle volume estimation method used in the current study remains a robust and validated approach against imaging-derived measures (Layec et al. 2014). The preserved estimated thigh CSA in the present group suggest that the old participants in our study may have been more physically active than in other cohorts. Of particular importance, a great deal of care was taken in this study to include old and young subjects with similar habitual recreational physical activity levels, to separate the effect of aging on key muscle strength determinants. However, even one year of heavy strength training may not necessarily increase thigh muscle mass in older adults, even after substantial increases in lower extremity muscle strength (Gates et al. 2024).

As such, given the traditional emphasis muscle mass has had on the understanding of age-related muscle strength decline (Clegg et al. 2013), the lack of difference in estimated thigh CSA between the old and young men, even with a 50-year age gap (25 vs. 75) and in the presence of substantial muscle strength differences, is notable. This finding is consistent with the lack of a significant association between estimated thigh CSA and MVC in the present study, suggesting that the strength differences observed between the groups were not reflected in this measure of thigh size.

Although estimated thigh CSA was similar in the old and young men, delving deeper into muscle morphology, biopsies revealed that the *m. vastus lateralis* was subject to age-differences in fast twitch type II proportion (−16%). This difference was accompanied by larger slow twitch type I myofiber size (12%) and count distribution (27%) in the old, a finding in line with other studies (Lexell 1995; Nilwik et al. 2013), possibly reflecting denervation of larger type II fibers and subsequent reinnervation from motor units typically innervating type I fibers (Kelly et al. 2018). Fiber type distribution may be regarded as the result of denervation with or without subsequent reinnervation of myofibers (Hepple and Rice 2016). Loss of the largest axons may lead to atrophy, particularly of the fastest and strongest type II fibers, either through myofiber atrophy or type conversion, resulting in myofibers with more type I-like characteristics, after collateral sprouting and grouping (Toien et al. 2023a; Wu et al. 2020). Indeed, this compensatory response is likely an important reason for the observed lack of difference in estimated thigh CSA, potentially mitigating the muscle strength reductions. Of notice, physical activity which is typically performed by old is dominated by low-force contractions (Bennie et al. 2016; Humphries et al. 2018), may be insufficient for preservation of high-threshold and strong type II fibers.

Muscle strength, contractility, and age

The present study revealed that properties of quadriceps muscle contractility was impaired in the old compared to young. Muscle contractility, a result of both neural and muscle properties, was expressed as resting twitch peak force, RFD, delay, and time to relaxation, which all exhibited an age-related difference (−39 to −56%). These findings are in line with previous work (Mau-Moeller et al. 2013; Roos et al. 1999; Toien et al. 2023b). Considering that the time from force onset to peak was not different between the groups, the lower twitch RFD was primarily the result of reductions in twitch peak force. This may be of importance for force generating capacity, because twitch and voluntary RFD are shown to be related, particularly in the earliest phase (< 50 ms) (Andersen and Aagaard 2006; Folland et al. 2014). Supporting this interpretation, both twitch RFD and peak force demonstrated strong associations with MVC, indicating that intrinsic contractile properties may accompany lower voluntary force production. The impaired quadriceps contractility may not be particularly surprising given the lower proportion of fast twitch fibers found in vastus lateralis in the old. Sarcoplasmatic reticulum Ca^{2+} release per action potential, and thus force production, is greater in fast twitch than slow twitch fibers (Baylor and Hollingworth 2003; Brody 1976). Cross bridge cycling rates are also 4–9-fold higher in the former (Harridge et al. 1996), which in combination with higher Na^{+} density may yield improved ability to produce high excitation rate from high firing frequency (Schiaffino and Reggiani 2011). Although somewhat speculative, a lower efferent neural drive, in particular firing frequency, may potentially yield disproportionally smaller increases in voluntary RFD in muscles with a low proportion of fast twitch fibers (Maffiuletti et al. 2016), as observed in the old.

Experimental considerations

In the present study, the evoked potentials assessments were obtained from the plantar flexors, whereas muscle strength, EMG, muscle contractility, and biopsies were obtained from the knee extensors. As such, muscle- and task-specific differences between methodologies cannot be excluded. Furthermore, the cross-sectional design precludes causal inference regarding the relative contribution or temporal sequence of the observed neural, contractile, and morphological differences, and the regression analyses should be interpreted as associations rather than causal relationships. Given the number of secondary, exploratory analyses, these findings should be interpreted cautiously, with emphasis on the magnitude and precision of effects rather than statistical significance alone. The reduced biopsy sample size means that selection bias specifically to these outcomes cannot be excluded. Finally, the findings are limited to men and may be subject to residual cohort or selection effects, as younger and older participants, despite reporting a physical activity level that was not different, were recruited from different environments (university vs. senior sports and activity clubs). Differences in lifetime physical activity, occupational demands, medication use, or other health-associated behaviours were not assessed; thus, residual cohort and selection confounding cannot be excluded.

Practical implications

Although lower extremity muscle strength was lower in the older men, the current study did not detect a statistically significant between-group difference in estimated thigh CSA,

even though recreational physical activity was comparable between groups. Investigating a wide range of both neural and muscle components in the same individuals, the current study strengthens the assumption with which we can assume that neural and muscular factors are substantially altered and are associated with the age-related difference in lower extremity muscle strength. Consequently, these findings may help identify potential targets for future strategies aimed at attenuating age-related declines in strength, RFD and ultimately, function. Future interventions and longitudinal studies may determine the extent to which the decline of these factors can be mitigated.

Conclusions

Recreationally active older men displayed expected reductions in lower extremity muscle strength compared to young. In the current study we show how various neuromuscular components contribute to this decline. Efferent neural drive, motoneuron excitability, quadriceps EMG and twitch characteristics were all reduced, along with differences in vastus lateralis fiber phenotype, evident as a lower fast twitch muscle fiber proportion and increased slow twitch fiber size.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00421-026-06491-4.

Acknowledgements The authors have no acknowledgments to declare.

Author contributions Conception or design of work: E.W., M.F.B. Acquisition, analysis or interpretation of data for the work: E.W., M.F.B., O.K.B., T.T., R.S. Drafting the work: E.W., M.F.B., R.S. Revising the work critically for important intellectual content: O.K.B., T.T. All authors have approved the final version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work as appropriately investigated and resolved. All persons designated as authors qualify for authorship and all those who qualify for authorship are listed.

Funding Open access funding provided by Molde University College - Specialized University in Logistics. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data availability statement Data are available upon reasonable request from the corresponding author.

Declarations

Conflict of interest The authors declare no conflicts of interest.

Ethical approval The study was approved by the regional ethics committee (REC number: 2018/1207) and all measures conducted in accordance with the declaration of Helsinki.

Consent to participate Participants provided written informed consent before data was collected.

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