In vivo physiological cross-sectional area and specific force are reduced in the gastrocnemius of elderly men

Christopher I. Morse, Jeanette M. Thom, Neil D. Reeves, Karen M. Birch, and Marco V. Narici. In vivo physiological cross-sectional area and specific force are reduced in the gastrocnemius of elderly men. J Appl Physiol 99: 1050–1055, 2005. First published May 19, 2005; doi:10.1152/japplphysiol.01186.2004.—Sarcopenia and muscle weakness are well-known consequences of aging. The aim of the present study was to ascertain whether a decrease in fascicle force (Ff) could be accounted for entirely by muscle atrophy. In vivo physiological cross-sectional area (PCSA) and specific force (Ff/PCSA) of the lateral head of the gastrocnemius (GL) muscle were assessed in a group of elderly men [EM, aged 73.8 yr (SD 3.5), height 173.4 cm (SD 4.4), weight 78.4 kg (SD 8.3); means (SD)] and for comparison in a group of young men [YM, aged 25.3 yr (SD 4.4), height 176.4 cm (SD 7.7), weight 79.1 kg (SD 11.9)]. GL muscle volume (Vol) and Achilles tendon moment arm length were evaluated using magnetic resonance imaging. Pennation angle and fiber fascicle length (Lf) were measured using B-mode ultrasonography during isometric maximum voluntary contraction of the plantar flexors. PCSA was estimated as Vol/Lf. GL Ff was calculated by dividing Achilles tendon force by the cosine of θ, during the interpolation of a supramaximal doublet, and accounting for antagonist activation level (assessed using EMG). Achilles tendon moment arm length, and the relative PCSA of the GL within the plantar flexor group. Voluntary activation of the plantar flexors was lower in the EM than in the YM (86 vs. 98%, respectively, P < 0.05). Compared with the YM, plantar flexor maximal voluntary contraction torque and Ff of the EM were lower by 47 and 40%, respectively (P < 0.01). Both Vol and PCSA were smaller in the EM by 28% (P < 0.01) and 16% (P < 0.05), respectively. Also, penannation angle was 12% smaller in the EM, whereas there was no significant difference in Lf between the YM and EM. After accounting for differences in agonists and antagonists activation, the Ff/PCSA of the EM was 30% lower than that of the YM (P < 0.01). These findings demonstrate that the loss of muscle strength with aging may be explained not only by a reduction in voluntary drive to the muscle, but mostly by a decrease in intrinsic muscle force. This phenomenon may possibly be due to a reduction in single-fiber specific tension. Aging; muscle architecture

AGING IS KNOWN TO RESULT in a decrease in muscle mass that has a direct impact on the ability to produce force (4, 22). However, it is apparent that muscle strength declines at a greater rate than muscle mass with aging (16). To quantify the relative contributions of atrophy and additional intrinsic factors to the loss of strength with aging, muscle strength [usually reported as joint torque during a maximum voluntary contraction (MVC)], is normalized to muscle mass [e.g., anatomical cross-sectional area (ACSA, the cross-sectional area of the muscle at right angles to its longitudinal axis)]. As a result of the disparity between the decline in muscle mass and muscle strength, the MVC-to-ACSA ratio (MVC/ACSA) is reported to be lower in the elderly than in the young (5, 8, 43, 47). This reduction in MVC/ACSA of elderly skeletal muscle has been proposed to be due to a number of neural and morphological changes associated with aging. For instance, an increase in the coactivation of antagonist muscles and a decrease in agonist muscle activation are both known to contribute to reductions in MVC/ACSA of elderly muscle (27, 43). Also, the inclusion of denervated muscle fibers in the assessment of ACSA has been shown to contribute to a reduced muscle force-to-ACSA ratio in elderly rats (42). In addition, a decrease in single-fiber specific tension (maximal in vitro fiber tension-to-fiber cross-sectional area ratio) may also contribute to the decline in MVC/ACSA in old age (11, 23). However, evidence to the contrary suggests that reduced physical activity, not aging per se, can account for reductions in fiber-specific tension (41). This is consistent with evidence at the whole muscle level demonstrating that, if physical activity levels are matched between the young and elderly, there is no apparent deficit in MVC/ACSA in the elderly (18). Although ACSA is a major determinant of MVC torque (1, 35), the scaling of joint torque to muscle ACSA presents a number of limitations, especially when comparing groups with contrasting architectural features, the result of which is an overestimation of contractile area in those muscles with a smaller pennation angle (θ) (31, 35). It is likely, therefore (in addition to intermuscle variance), that this contributes to the evidence that MVC/ACSA is not reduced in the tibialis anterior of the elderly compared with the young (18), particularly because this is a bipennate muscle. To this end, muscle volume (Vol) gives a better indication of the entire contractile material and fiber area than ACSA and is therefore a better determinant of joint torque (12), particularly when comparing heterogeneous groups such as the young and elderly (34). Although normalization of strength to Vol (MVC/Vol) gives a more accurate estimate of specific force compared with MVC/ACSA (13), force per unit of muscle area should strictly be based on physiological cross-sectional area (PCSA, the area of the muscle at right angles to the longitudinal axis of the fibers), while taking into account the activation of agonist and antagonist muscles (29), muscle architecture, and moment arm...
length, ultimately providing an in vivo estimate of fascicle force (Ff) as opposed to MVC torque (29). Therefore, although we have previously demonstrated that a reduction in agonist muscle activation contributes to a reduction in MVC/Vol in the elderly (34), normalizing Ff to PCSA allows a direct comparison of the intrinsic force-producing capacity of skeletal muscle in vivo, between the young and elderly, without the confounding variables of neural drive, muscle architecture, or moment arm length as is commonly included in comparisons of MVC/Vol or MVC/ACSA.

Currently, in vivo specific force (Ff/PCSA) measurements comparing the young and elderly are scanty, and, where available, specific force and PCSA were estimated by combining fiber length and pennation based from cadaveric data, with in vivo volume measurements (20). Muscle architecture, however, has been shown to be altered in the elderly (37), and, furthermore, the fixing of cadaveric muscles is known to result in muscle shrinkage. Hence the use of architectural data obtained from cadavers may lead to errors in the estimation of PCSA, particularly when comparing two heterogeneous groups (29). Therefore, the aim of the present study was to ascertain whether the loss of muscle strength with aging may be due to a decrease in muscle force per cross-sectional area once differences in muscle architecture between young and elderly individuals, and in muscle activation, are taken into account. To this purpose, specific force of a human locomotor muscle [the gastrocnemius lateralis (GL)] was assessed from evaluations of muscle fascicles’ force and PCSA performed in vivo in a group of young and elderly individuals.

MATERIALS AND METHODS

Subjects. Nineteen elderly men [EM, aged 73.83 yr (SD 3.5), height 173.4 cm (SD 4.4), mass 78.4 kg (SD 8.3), means (SD)] and 12 young men [YM, aged 25.3 yr (SD 4.4), height 176.4 cm (SD 7.7), mass 79.1 kg (SD 11.9)] volunteered to participate in this study. All subjects were medically screened by a general practitioner aware of the purpose and testing procedures of this investigation. The exclusion criteria adopted in this study are listed in Table 1.

Consent to participate in this experimental study was obtained from the general practitioner for each elderly participant after a review of his medical history and an assessment of his health status at the time of the investigation. All elderly participants were community dwelling and lived independently. Both young and elderly individuals were all recreationally active but not participating in a structured training regime. All procedures were approved by the Ethics Committee of Manchester Metropolitan University, and prior informed consent was obtained from each subject. Previous analysis of the elderly individuals revealed their physical activity levels to be matched on all levels except for high-intensity activity, in which they were 30% less active than their young counterparts (34).

Familiarization. Participants were familiarized to all proceedings in a separate session before data collection; in addition, the elderly volunteers had previously taken part in a study performed in this laboratory that involved procedures similar to the present (31, 32, 34). Familiarization sessions consisted of repeated MVCs at the joint angle required for force measurements (both plantar flexion and dorsiflexion); further to this, supramaximal doublets were applied over three to four MVCs. Those participants who were unable to tolerate the doublets were excluded from the testing session.

Plantar flexor strength. Isometric plantar flexion (PF) MVC torque was recorded with the subjects lying prone, the left foot secured to the foot adapter of an isokinetic dynamometer (Cybex Norm, Cybex International, New York, NY). Straps were used about the hip to prevent forward displacement of the body during maximal plantar flexions. Subjects were positioned with the knee at full extension and the lateral malleolus aligned with the axis of rotation identified on the dynamometer. Before MVC, the participants performed three submaximal isokinetic plantar flexion and dorsiflexion contractions as a warm up. Two isometric maximal voluntary plantar flexion contractions (MVC) were performed at an ankle joint angle of −20° (the foot in dorsiflexion), 2 min separated each MVC attempt. The foot was placed at −20° as this has previously been shown to be the optimum fascicle length (Lf) within the physiological range of the gastrocnemius muscle (25). During each MVC, visual feedback was given by using an online graphical display, and constant verbal encouragement was provided by the investigator.

The sum of the torque generated during the strongest MVC and that of the twitch-doublet was considered at the true maximal MVC and used for the calculation of specific tension (as described by Refs. 13, 29). The value of torque calculated with this method probably represents a peak MVC and not the true maximal MVC as would have been obtained by adding a tetanus rather than doublet to the voluntary torque. However, considering that the delivery of a tetanic current is extremely painful and that the population studied included older individuals, we opted to use a doublet instead. The same considerations have been made in previous studies assessing activation capacity and specific force in the elderly (20, 38). Although the aim of the present investigation was to attain a PF torque as close to maximum without the confounding variable of reduced activation, rather than to comment on activation differences between the young and elderly person, we have previously presented data on muscle activation capacity (34) and the possible influence of tendon compliance on the assessment of activation in the elderly (32).

Two maximal isometric dorsiflexion contractions were performed after the PF MVC to obtain maximal dorsiflexion electromyographic (EMG) data for calculation of antagonist coactivation in the tibialis anterior. PF and dorsiflexion MVC attempts were held for ~4 s, a time amply sufficient to reach a torque plateau.

Twitch interpolation and PF peak torque. To obtain an estimation of specific force without the confounding variable of reduced agonist muscle activation, supramaximal doublets were superimposed over the second PF MVC. The doublets (pulse width = 50 μs, interpulse interval = 10 ms) were applied percutaneously (DS7 Digitimer Stimulator, Digitimer, Welwyn Garden City, UK) by using rubber stimulation pads (76 mm × 127 mm, and 38 mm × 89 mm, Versastim, Conmed, NY). The anode was placed distal to the popliteal crease and the cathode over the distal myotendinous junction of the soleus. The supramaximal doublet amplitude was determined before superimposition by administering twitches of progressively increasing current intensity. Twitches were administered with the subjects in a relaxed state, starting from 100 mA and increasing by 50- to 100-mA increments until no further increase in twitch torque was observed with a further 50-mA increase in stimulation amplitude.

Table 1. Exclusion criteria

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<td>Acute febrile or systemic disease within the previous 2 years</td>
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The PF isometric torque (peak torque) used in the estimation of specific force was determined as PF MVC torque + superimposed doublet torque, as described previously (29).

Coactivation. EMG activity of the tibialis anterior was recorded while maximal isometric plantar flexion and dorsiflexion contractions were performed using two Ag-AgCl percutaneous unipolar electrodes 10 mm in diameter (Neuroline, Medicotest, Rugmarken, Denmark). Electrodes were set in a bipolar configuration with a 20-mm inter-electrode distance at one-third of muscle length. Muscle boundaries were identified by ultrasonography to reduce the influence of cross talk, and the electrodes were placed along the mid sagittal plane of the muscle. A reference electrode was placed on the head of the fibula. Before placement of the electrodes, the skin was shaved to remove hair and the recording sites were abraded lightly with abrasive gel and cleaned with alcohol swabs to reduce interelectrode impedance below 5 kΩ. The raw EMG activity was acquired with a sampling frequency of 2,000 Hz and processed with a multichannel analog-digital converter (Biopac Systems, Santa Barbara, CA). The raw EMG signal was filtered with low and high band-pass filters set at 500 and 10 Hz, respectively, and amplified with a gain of 2,000. The level of coactivation of the tibialis anterior was assessed by using the root mean square of the raw EMG signal, which was integrated over 1 s about the peak MVC torque during PF; this was then expressed as the percentage EMG activity recorded from the tibialis anterior during maximal dorsiflexion (20, 26, 27). The torque produced by the dorsiflexors during plantar flexion was estimated by assuming a linear relation between torque and EMG activity, which has previously been reported from the tibialis anterior muscle (30). To obtain an estimate of plantar flexion torque, dorsiflexion torque during plantar flexion was added to the net plantar flexion torque.

Muscle volume. Serial axial plane magnetic resonance imaging (MRI) scans were acquired along the length of the GL by using a fixed 0.2-T MRI scanner (E-Scan, ESAOTE Biomedica, Genova, Italy). The first scan was centered at the lower edge of the patella and was sufficient to include the origin of the GL. Subsequent scans were carried out in contiguous 7-cm distal sections until the myotendinous junction of the GL had been reached (Fig. 1). Scans were obtained with a T1-weighted three-dimensional isotropic profile using the following scanning parameters: time to echo, 16 ms; repetition time, 38 ms; field of view, 180 mm × 180 mm; matrix, 256 × 192. Subjects were supine for 15 min before the scan to allow fluid shifts to occur (3). In total, four scans were performed along the length of the GL consisting of seven contiguous axial slices (slice thickness = 8 mm and gap = 2 mm). To ensure that each section was reconstructed accurately, reference markers were placed along the leg to coincide with each 7-cm section. These markers were placed on the skin, along the tibia at a distance relative to the proximal end of the patella (identified using ultrasound with the subject in a supine position). The ACSA of the GL muscle (excluding visible intramuscular fat and connective tissue. Fig. 2) was measured from each scan using imaging software (NIH image version 1.61/ppc, National Institutes of Health, Bethesda, MD), and multiplied by the slice thickness (inclusive of gap) to give an estimate of Vol. The accuracy of this technique has been reported previously (21, 35).

Muscle architecture. Muscle architecture was recorded from participants during performance of PF MVC and is presented in the results from the time point of the superimposed doublet. Therefore, as with the measurement of MVC, participants were prone, with the foot secured in −20° dorsiflexion. Fiber Lf and θ were measured using B-Mode, real time ultrasonography (HD-3000, ATL, Bothell). Images were obtained along the mid sagittal plane of the GL, at the mid distance between the proximal and distal tendon insertion identified by ultrasound (7.5-MHz linear-array probe, Fig. 3). The head of the probe was held perpendicular to the dermal surface to provide an image including both superficial and deep aponeuroses and a number of clearly visible fasciculi that could be followed between the aponeuroses. In total, at least six fasciculi were analyzed per participant. To improve acoustic coupling, water-soluble transmission gel was placed over the scan head. Ultrasound scans were recorded on VHS at 25 Hz, digitized on an Apple Macintosh G4 computer, and analyzed offline with digitizing software. The θ was measured as the angle of fascicular insertion into the deep aponeurosis (44). Lf was defined as the length of the fascicle between the deep and superficial aponeuroses (36). PCSA (cm²) was calculated as the ratio of GL Vol (cm³) to Lf (cm). To ensure that measurements of Lf and θ were taken at the time point of superimposed stimulation, the VHS and torque recordings were synchronized. Accuracy of the ultrasound method for determining muscle architectural features has previously been validated by comparison with direct anatomical measurement on cadavers (36). One limitation of the ultrasound technique is that no information can be obtained outside of the two-dimensional orientation in which the ultrasound probe is placed. Therefore, care was taken to ensure that the orientation of the ultrasound probe coincided as much as possible with that of the muscle fascicle orientation. In fact, when the probe is aligned along the same sagittal axis of the fascicles, the entire length of the fascicles in the central portion of the image can be tracked from the deep to the superficial aponeuroses.

Moment arm length. In vivo MRI was performed to determine moment arm length of the Achilles tendon ankle at rest. A series of sagittal plane scans was taken of the left ankle with the foot secured at a right angle to the tibia (0°), at 10° dorsiflexion, and at 20° plantar flexion. Three scans were required to determine the center of rotation of the talocrural joint as described previously (39). Scans were obtained by using T1-weighted three-dimensional isotropic profile with the same scanning parameters that were described above to determine Vol. The ankle was secured with an adjustable wooden foot mount and Velcro straps, and the subjects were stabilized in a right lateral position. The moment arm length was calculated as the per-
pendicular distance from the center of rotation of the talocrural joint to the Achilles tendon action line.

**Achilles tendon force.** Tendon force was calculated by dividing PF peak torque by the Achilles tendon moment arm length (Fig. 3).

**GL-fascicle force and specific force.** To estimate the contribution of the GL to Achilles tendon force, the relative PCSA of the GL within the triceps surae (TS) was calculated. The method of estimating the relative contribution of each constituent muscle to the total tendon force component from relative PCSA of each constituent of a muscle group has been previously reported (35). By using the same procedure, it was possible to estimate the GL component of Achilles tendon force, from the ratio of GL PCSA to the total PCSA of the TS multiplied by the total tendon force of the PF group. We have previously determined that the GL constitutes 11% of the triceps surae PCSA (31), and the TS group was estimated to represent 91% of the total PF PCSA in the elderly (45); hence this seems to justify the assumption that the TS PCSA represents the main PCSA of the PFs. Furthermore, it was also demonstrated that there was no difference in the contribution of each muscle to the triceps surae PCSA as a whole between the young and elderly. It seems reasonable, therefore, to assume that the technique of force allocation based on the relative PCSA of a muscle to the muscle group as a whole, which has been used previously in the quadriceps group (35), is sound. Once the GL component of the Achilles tendon force was calculated, this was divided by cos θ to determine the Ff (Fig. 3).

Specific force of the GL muscle was calculated by dividing GL Ff by GL PCSA. The present method of assessing specific force in vivo has been derived from techniques previously utilized in the soleus, tibialis anterior, and vastus lateralis muscles (13, 29, 35, 38). A Kruskal-Wallis nonparametric ANOVA was performed. Differences were considered significant at an alpha level of $P < 0.05$. To determine reliability, muscle architectural characteristics were measured on two occasions, separated by at least 1 day, in nine elderly subjects, by the same investigator. Intraclass correlation coefficients (1,2) were high and significant for all measurement techniques. Lf = 0.90, muscle Vol = 0.94, and PCSA = 0.87. The θ reliability was lower (0.80); however, the mean difference between the 2 test days was minimal (0.27°, $R^2 = 0.92$).

**RESULTS**

**Strength, agonist activation, and antagonist coactivation.** PF MVC torque was 47% lower in EM compared with YM ($P < 0.01$, Table 2). Peak PF torque (MVC + elicited torque) was 40% lower in the EM compared with the YM ($P < 0.01$); accordingly, the ratio of voluntary to elicited torque was significantly lower in the EM compared with the YM [86% (SD 7) and 98% (SD 2), respectively, $P < 0.01$]. Antagonist muscle coactivation was not significantly different between the YM and EM [13.5% (SD 5.9) and 9.6% (SD 5.0), respectively]. No difference was found in Achilles tendon moment arm length between the young and elderly (Table 2). When agonist and antagonist muscle activity was accounted for, both Achilles tendon force and GL Ff were lower in the EM by 37 and 38%, respectively ($P < 0.01$, Table 2).

**GL muscle size and architecture.** GL Vol and ACSA were 28 and 17% smaller in EM than in YM (Table 2, $P < 0.001$). PCSA was found to be 16% smaller in EM ($P < 0.05$, Table 2). Compared with YM, θ was smaller in the EM by 12% ($P < 0.05$), and although Lf was 9% smaller in EM, the difference was not significant ($P = 0.23$, Table 2). GL-specific force (Ff/PCSA) was 30% smaller in the EM compared with the YM ($P < 0.01$, Table 2). For comparative purposes, MVC/ACSA and ACSA/Vol were found to be 18.6 and 24.9% smaller in the elderly men, respectively (Table 2).

**Reliability.** To determine repeatability, muscle architectural characteristics were measured on two occasions, separated by at least 1 day, in nine elderly subjects, by the same investigator. Intraclass correlation coefficients (1,2) were high and significant for all measurement techniques. Lf = 0.90, muscle Vol = 0.94, and PCSA = 0.87. The θ reliability was lower (0.80); however, the mean difference between the 2 test days was minimal (0.27°, $R^2 = 0.92$).

**DISCUSSION**

The main finding of the present investigation was that even when differences inactivation of agonist and antagonist muscles were accounted for, the specific force of the GL muscle in the elderly men was lower than in the young men. This finding suggests that additional factors, other than a reduced PCSA and a lower agonist activation, contribute to the reduction in fascicle force in the elderly.

![Fig. 3. Schematic representation of the lower leg showing joint torque (A), Achilles tendon force (B), and fascicle force (C) and incorporating a sagittal plane sonograph of the gastrocnemius lateralis, to determine fascicle length and pennation angle during plantarflexion MVC.](image-url)
In line with most previous reports, muscle mass was found to be smaller in the elderly men than the young men. Depending on the scaling factor used to quantify muscle geometric dimensions (ACSA, PCSA, or Vol), the difference between the young and elderly ranged from 25% when using Vol to 17% when using PCSA or ACSA. Interestingly, our observations of a greater loss in muscle PCSA than in Lf suggests that, at least for the GL, the difference in Vol between the young and the elderly is mainly due to a decrease in sarcomeres in parallel (evidenced by the reduction in PCSA) rather than in series. This is because any significant change in Lf should reflect a loss of sarcomeres in series (46). The functional implications of these alleged changes in parallel and in series sarcomeres are exemplified by the observation that the loss of strength, more than of shortening velocity, contributes to the decline in muscle power in old age (10).

In the present study, a 17% smaller PCSA was not fully accounted for by a 38% smaller fascicle force in the elderly men; hence specific force (Ff/PCSA) was less in the elderly men. Previously reduced agonist activation and increased antagonist coactivation have been shown to result in lower MVC/Vol in the elderly (27, 34). In agreement with the present investigation, we have previously reported PF activation to be reduced in elderly men, with no difference in coactivation of the antagonist muscles (32, 34). Because of the apparent decline in agonist muscle activation of the present elderly participants, it was necessary to account for both agonist and antagonist muscle activity in the calculation of specific force. Indeed this technique has been previously utilized in both young and elderly individuals to estimate specific force (29, 38). Therefore, in the present study, the calculation of GL-specific force accounted for agonist and antagonist muscle activity, and as such the lower specific force in these elderly men may be regarded as independent of neural factors. Although we have previously shown that the decline in specific force (MVC/Vol) in old age was accounted for by a deficit in activation (34), the present findings show that, in addition to the activation deficit, muscular factors (presumably a decrease in single-fiber specific tension) play a significant role in the observed deterioration in Ff/PCSA. In accordance with our previous results (34), when strength is normalized with Vol the present elderly men are 25% weaker than their young counterparts, which can be partly accounted for by a 12% difference in agonist muscle activation.

A number of possible mechanisms have been identified that may contribute to the observed reduction in whole muscle specific force in the present elderly men. A reduction in single-fiber specific tension and a selective atrophy of fast-twitch fibers are likely to have the greatest influence on the reductions in specific force reported in the present study. Single-fiber specific tension from elderly humans has been shown to be reduced (7, 23, 25, 40) and to contribute in part to the reductions in whole muscle MVC/ACSA in the elderly (11). These reductions in fiber-specific tension can be accounted for both by changes in the number of active cross bridges as well as by a reduction in excitation-contraction coupling. Indeed, recently D’Antona et al. (7) has shown that fiber-specific tension is a linear function of myofibrillar protein density, which suggests that in both aging and disease the decrease in fiber-specific force is due to a decrease in the number of cross bridges rather than a decrease in force per cross bridge. When specific tension is measured in elderly fibers with an intact sarcolemma membrane, a deficit in tension may result from uncoupling of the excitation-contraction process and a decrease in sarcoplasmic calcium sensitivity (9, 15); both factors may contribute to whole muscle-specific force deficits in the elderly.

Although single-fiber specific tension appears to be reduced in the elderly, evidence to the contrary suggests that, when physical activity of both the young and elderly is matched, there is no apparent difference in fiber-specific tension of the elderly (41). In the present study, even though we purposefully recruited nonsedentary and independently dwelling elderly individuals, their physical activity level was 30% lower compared with their younger counterparts (34). Therefore, the reduction in PCSA in the present elderly can be considered to be both the result of an obligatory loss in muscle mass as a result of the aging process and also the result of reduced physical activity, which is known to contribute to reductions in single-fiber specific tension (7) and muscle mass (2). It is reasonable to speculate that the reductions in specific force and PCSA observed in the present study are partly reversible with increased physical activity. Indeed, resistance training has been shown to result in significant increases in the specific force of the vastus lateralis muscle of elderly individuals (38).

Although the architectural characteristics of a muscle appear to have a greater influence on the force-producing capacity than fiber type (6), a selective atrophy of fast-twitch muscle associated with an obligatory denervation with aging (24) would result in a reduction of type II fibers, which are known to have a higher specific tension than type I fibers (14, 41). This selective atrophy has previously been proposed as a possible mechanism to account for reductions in whole muscle-specific force when there is no apparent reduction in single-fiber specific tension (41).

In addition to a decrease in fiber-specific tension and altered fiber type, it is likely that additional factors such as denervated muscle fibers (17, 42) and an increased intramuscular fat and connective tissue content (19) are likely to contribute to the observed decrease in specific force in the elderly.

When compared with previous literature, the specific force values yielded by the GL of young men in the present investigation are comparable to values obtained in other ankle muscles. As a matter of fact, in the soleus and tibialis anterior, specific force has been reported to be 15 N/cm (29), compared with 13.1 N/cm in the present young men. Furthermore, the GL fascicular forces obtained in young men in the present investigation are in close agreement with those previously reported (416.2 N·m in the present study and 393 N·m obtained by Maganaris (28)).

In conclusion, the present study has demonstrated that, with aging, GL muscle force is reduced to a greater extent than GL PCSA, and hence specific force is reduced even when activation of the agonist and antagonist muscle are accounted for. Therefore, at least in the GL of elderly men, the decline in specific force is likely to be accounted for by changes intrinsic to the muscle.

GRANTS

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REFERENCES


