In vivo specific tension of human skeletal muscle

CONSTANTINOS N. MAGANARIS,1 VASILIOS BALTZOPoulos,1 D. BALL,1 AND ANTHONY J. SARGEANT1,2

1Active Life Span and Neuromuscular Biology Research Groups, Manchester Metropolitan University, Alsager ST7 2HL, United Kingdom; and 2Institute for Fundamental and Clinical Human Movement Sciences, Faculty of Human Movement Sciences, Vrije Universiteit, 1081 BT Amsterdam, The Netherlands

Received 16 August 2000; accepted in final form 2 October 2000

Maganaris, Constantinos N., Vasilios Baltzopoulos, D. Ball, and Anthony J. Sargeant. In vivo specific tension of human skeletal muscle. J Appl Physiol 90: 865–872, 2001.—In this study, we estimated the specific tensions of soleus (Sol) and tibialis anterior (TA) muscles in six men. Joint moments were measured during maximum voluntary contraction (MVC) and during electrical stimulation. Moment arm lengths and muscle volumes were measured using magnetic resonance imaging, and pennation angles and fascicular lengths were measured using ultrasonography. Tendon and muscle forces were modeled. Two approaches were followed to estimate specific tension. First, muscle moments during electrical stimulation and moment arm lengths, fascicular lengths, and pennation angles during MVC were used (data set A). Then, MVC moments, moment arm lengths at rest, and cadaveric fascicular lengths and pennation angles were used (data set B). The use of data set B yielded the unrealistic specific tension estimates of 104 kN/m² in Sol and 655 kN/m² in TA. The use of data set A, however, yielded values of 150 and 155 kN/m² in Sol and TA, respectively, which agree with in vitro results from fiber type I-predominant muscles. In fact, both Sol and TA are such muscles. Our study demonstrates the feasibility of accurate in vivo estimates of human muscle intrinsic strength.

The former can be estimated from the muscle joint moment when the moment arm length and pennation angle of the muscle are known, and the latter can be estimated by dividing the muscle volume by the muscle fiber length. Some authors have incorporated such calculations in their analyses (see Table 1). The validity of the resultant specific tensions, however, is questionable because net resultant joint moments and resting-state/cadaveric musculoskeletal geometry data have been used. The following three factors need to be considered in analyses of in vivo maximum isometric forces and joint moments. 1) Antagonist muscles may cocontract during voluntary contractions, and this results in a lower net measured joint moment compared with the joint moment of the agonists tested (22, 28). Therefore, muscle-specific as opposed to net resultant joint moments should be measured. 2) Moment arm lengths and pennation angles at rest or from cadaveric studies do not represent measurements during contraction (26, 27, 29–31). Therefore, contraction-specific musculoskeletal geometry data as opposed to resting-state/cadaveric data should be used in muscle-tendon force calculations. 3) Cadaveric muscles may shrink during fixing (9); therefore, cadaver-based estimations of PCSA may not represent actual in vivo dimensions. In vivo PCSA estimates should thus be used in the calculation of specific tension.

In the present study, we have estimated and compared the specific tensions of human soleus (Sol) and tibialis anterior (TA) muscles 1) with the use of muscle-specific joint moments and in vivo contraction-specific musculoskeletal geometry measurements and 2) by following the traditional approach, i.e., with the use of net resultant joint moments and resting-state/cadaveric musculoskeletal geometry data. We hypothesized that, in contrast to the latter approach, the former approach would yield estimates consistent with experimental results from isolated muscle.

METHODS

Six healthy men [average (mean ± SD) age, height, and body mass: 28 ± 4 yr, 175 ± 8 cm, and 75 ± 7 kg, respec-

http://www.jap.org 8750-7587/01 $5.00 Copyright © 2001 the American Physiological Society

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
Table 1. Summarized reports in the literature on in vivo specific tension of the ankle plantar flexors

<table>
<thead>
<tr>
<th>Ref. No.</th>
<th>Force Measurements</th>
<th>Cross-Sectional Area Estimations</th>
<th>Specific Tension, kN/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>External force</td>
<td>ASCA from anthropometry</td>
<td>329</td>
</tr>
<tr>
<td>11</td>
<td>External moment →</td>
<td>PCSA from MRI and cadavers</td>
<td>108</td>
</tr>
<tr>
<td>13</td>
<td>External force →</td>
<td>PCSA from cadavers</td>
<td>383</td>
</tr>
<tr>
<td>14</td>
<td>External force →</td>
<td>ACSA from cadavers</td>
<td>628</td>
</tr>
<tr>
<td>33</td>
<td>External force →</td>
<td>PCSA from MRI and in vivo measures</td>
<td>97*</td>
</tr>
<tr>
<td>36</td>
<td>External force →</td>
<td>ACSA from cadavers</td>
<td>549</td>
</tr>
<tr>
<td>42</td>
<td>External force →</td>
<td>ACSA from cadavers</td>
<td>412</td>
</tr>
</tbody>
</table>

ACSA, anatomical cross-sectional area (cross-sectional area of the whole muscle); PCSA, physiological cross-sectional area (sum of cross-sectional areas of all fibers); MRI, magnetic resonance imaging. *Gastrocnemius medialis muscle.

tively), from whom informed consent had been obtained, volunteered to participate in this study. All were physically active, and none had any history of musculoskeletal injury or any orthopedic abnormality in the lower extremities. The study was approved by the local ethics committee.

The following eight steps were taken to calculate specific tension: step 1, measurement of joint moment; step 2, measurement of moment arm length; step 3, calculation of tendon force from steps 1 and 2; step 4, measurement of fascicular length and pennation angle; step 5, calculation of muscle force from steps 3 and 4; step 6, measurement of muscle volume; step 7, calculation of PCSA from steps 4 and 6; and step 8, calculation of specific tension from steps 5 and 7. All measurements were taken on the right leg.

Step 1: Measurement of Joint Moment

Subjects were securely fixed in the prone position on an isokinetic dynamometer (Lido Active, Loredan Biomedical, Davis, CA), with the knee of the tested leg flexed at 60° (Fig. 1). The pivot point of the lever arm of the dynamometer was aligned with the malleoli, and the ankle joint was fixed at the neutral anatomic position (the foot at right angles to the tibia), which corresponds to near-optimal lengths in both Sol and TA muscles (6). No passive forces were detected by the dynamometer load cell in the plantar flexion and dorsiflexion directions at the position studied (see also Ref. 38). Three isometric plantar flexion maximum voluntary contractions (MVCs) and three dorsiflexion MVCs were performed 2 min apart, after having compensated for the effect of gravity by performing dynamic weighing of the limb. The highest plantar flexion and dorsiflexion MVC angles corresponding to 15° of dorsiflexion, neutral ankle position, and 15° of plantar flexion. Scanning was performed using a 1.5-T, 64-Hz magnet (G.E. Signa Advantage, Milwaukee, WI) and a fast-GRASS sequence with 15-ms repetition time, 6.7-ms echo time, 24-cm field of view, 1 excitation, 256 × 128 matrix, 5-mm slice thickness, and 2-s scanning time. The ankle joint complex was represented by the tibiotalar joint, and the orientation of Achilles tendon was considered representative of that covered in tissue that was soaked in water. For Sol stimulation, the electrodes were bandaged over the gastrocnemius muscle and the distal part of Sol. For TA stimulation, the electrodes were bandaged over the proximal and distal halves of TA. Axial-plane sonographs were taken to locate the stimulating electrodes within the borders of each muscle studied. Stimuli were delivered by a custom-built high-voltage stimulator and controlled by a purpose-developed computer software. Two contractions 2 min apart were elicited in each muscle, and the highest moment values obtained were used for further analyses. Surface electromyogram (EMG) signals from TA and peroneus tertius during Sol stimulation and from Sol and peroneus tertius during TA stimulation indicated negligible current spread. When the recording electrodes were placed closer to stimulating muscle, the detected EMG signal was more prominent. Ultrasound scanning of electrically active regions in nonstimulated muscles revealed, however, no fascicular shortening, thus indicating the presence of cross-talk rather than coactivation. Furthermore, at the adopted knee position of 60°, the biarticular gastrocnemius is slack (yet active during Sol stimulation), thus transmitting only a minor force to the calcaneus during plantar flexion contraction (12, 15, 16). Therefore, the ankle moments recorded during stimulation in our experiment were considered to have negligible contributions from muscles other than Sol and TA.

Ankle moment data collection was repeated after 3 days. There was no difference (P > 0.05, Student’s paired t-test) in the measurements between tests; therefore, they were averaged for each subject.

Step 2: Measurement of Moment Arm Length

Moment arm length measurements were performed from in vivo magnetic resonance images (MRIs) at rest and during MVC as described elsewhere (26, 29). In brief, sagittal-plane MRIs of the foot were taken at rest and during plantar flexion and dorsiflexion MVCs at ankle angles corresponding to 15° of dorsiflexion, neutral ankle position, and 15° of plantar flexion. Ankle moment data collection was repeated after 3 days.
of Sol tendon. In the MRI at the neutral ankle position, the Achilles and TA tendon action lines were identified, and, from the MRIs at 15° of dorsiflexion and 15° of plantar flexion, the center of rotation in the tibiotalar joint at the neutral ankle position was calculated using the Reuleaux method (35). The perpendicular distance from the center of rotation to the tendon action line was the moment arm length (Fig. 2).

Step 3: Calculation of Tendon Force

Tendon force was calculated by dividing joint moment by the moment arm length (Fig. 3).

Step 4: Measurement of Fascicular Length and Pennation Angle

Details about the muscle architectural measurements in the study have been described elsewhere (27, 30). Two-dimensional muscle geometry characteristics were estimated from morphometrics of sagittal-plane sonographs taken with a 7.5-MHz linear-array B-mode probe (Esaote Biomedica AU3 Partner, Florence, Italy). Sonographs were taken during MVC from the central region along the muscle midsagittal axis, where it has been reported that architectural characteristics are representative of those along and across the muscle (27, 30). The fascicular length between origin and insertion was digitized, taking into account any observable curvature along the fascicular echo. The muscle pennation angle was measured as the angle between the fascicular echo at its insertion in the aponeurosis and the aponeurotic echo (Fig. 4). For the bipennate TA, the fascicular length and pennation angle of each unipennate half were measured separately, and average values across the two halves were used for further analyses (Fig. 4).

Step 5: Calculation of Muscle Force

Muscle force was calculated by dividing tendon force by the cosine of pennation angle (Fig. 5).

Step 6: Measurement of Muscle Volume

Muscle volume was estimated from a series of continuous axial-plane MRIs along the lower leg (Fig. 6) as described by Fukunaga et al. (10). Scanning was performed using the magnet described above and a fast-spin echo sequence with 500-ms repetition time, 18-ms effective echo time, 1 excitation, 256 × 128 matrix, 48-cm field of view, 10-mm slice thickness, 0-mm interslice gap, and 37-s scanning time. All MRIs were taken at rest in the supine position with the knee of the scanned leg fully extended. In each slice, the anatomical cross-sectional areas (ACSAs) of Sol and TA were digi-
tized, and the respective muscle volumes were calculated as the product of the sum of the ACSAs in all slices times the MRI slice thickness.

All image analyses were performed by the same investigator using a computerized sonic digitizer (TDS, Blackburn, UK). Average values across three measurements were used for further analyses.

**Step 7: Calculation of PCSA**

Estimations of PCSA were obtained from the ratio of muscle volume to fascicular length (2, 9, 13). Sol was treated as a unipennate muscle (2, 10), and TA was treated as two separate equidimensional unipennate parts, each one occupying half the whole muscle volume. The whole TA PCSA was obtained by adding the PCSAs of the two unipennate halves (see also Ref. 33).

**Step 8: Specific Tension Estimation**

Specific tension was estimated and compared from the ratio of muscle force to PCSA using two data sets. First, muscle moments during electrical stimulation and in vivo moment arm lengths, fascicular lengths, and pennation angles during MVC were taken into account (data set A). After this traditional approach, muscle stress was calculated using net MVC joint moments, moment arm lengths at rest, and cadaveric fascicular lengths and pennation angles (data set...
In this data set, cadaveric pennation angles reported by Wickiewicz et al. (43) were included. Fascicular lengths were estimated with the assumption that the muscle fascicle length-to-muscle length ratio is constant for a given muscle across different cadavers (9–11, 21, 43). Ratio values reported by Wickiewicz et al. (43) were used for these calculations. Muscle length was measured in vivo as the distance between the most proximal and most distal axial-plane MRIs in which the muscle was visible (10, 21, 31).

Statistics

Values are presented as means ± SD. Differences in specific tension estimates were tested using a two × two (muscle × data set) ANOVA. Statistical difference was set at a level of \( P < 0.05 \).

RESULTS

For any of the parameters examined, the values included in data set A were different from those in data set B (Table 2).

Measured Parameters

The Sol and TA moments were smaller by \( -15 \) Nm (12%) and \( 22 \) Nm (50%), respectively, compared with the net MVC ankle plantar flexion and dorsiflexion moments. The Sol and TA moment arm lengths during MVC were larger by \( -1.1 \) cm (22%) and \( 1.5 \) cm (44%), respectively, compared with rest. The Sol fascicle length during MVC was larger by \( -1.1 \) cm (58%) compared with cadaveric values. The TA fascicle length during MVC was smaller by \( 2.7 \) cm (36%) compared with cadaveric values. Pennation angles during MVC were larger compared with cadaveric values, by \( -17^\circ \) (68%) in Sol and \( 14^\circ \) (280%) in TA.

Calculated Parameters

The above differences between resting-state and contracting-state musculoskeletal geometry measurements and muscle and net MVC joint moments yielded different estimates of tendon force, muscle force, PCSA, and specific tension using data set A and data set B.

The use of values from data set B resulted in overestimated Achilles and TA tendon forces by \( \sim 687 \) N (39%) and \( 866 \) N (189%), respectively, compared with calculations using values from data set A. The Sol and TA muscle forces were larger by \( \sim 318 \) N (13%) and \( 850 \) N (175%), respectively, when values from data set B were used rather than those from data set A. Compared with data set A, the use of data set B resulted in an overestimation by \( \sim 46 \) kN/m\(^2\) (31%, \( P < 0.01 \)) in Sol specific tension and an overestimation by \( \sim 503 \) kN/m\(^2\) (325%) in TA specific tension. No difference (\( \sim 5 \) kN/m\(^2\), 3%, \( P > 0.05 \)) was found between the Sol and TA specific tension estimates using data set A. On the contrary, the use of data set B yielded a sixfold difference (\( \sim 554 \) kN/m\(^2\), \( P < 0.01 \)) in specific tension between the two muscles.

Sensitivity Analysis

Although previous studies have indicated that most of the measurements involved in this study are reproducible and observer independent (10, 26, 27, 29–31), it is important to quantify the impact of possible size measurement errors on the specific tension estimation with our protocol (data set A). The sensitivity of specific tension estimate was examined assuming a 10% error in the measurements of joint moment, moment arm length, pennation angle, fiber length, and muscle volume. The effect of each parameter on the resultant specific tension value was examined separately. The results of this analysis are summarized in Table 3 and indicate that a 10% overestimation or underestimation in any one of the above parameters would yield a maximum error of 10% in the estimated

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Sol</th>
<th>TA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint moment, Nm</td>
<td>( 107 ± 7 )</td>
<td>( 22.5 ± 2 )</td>
</tr>
<tr>
<td>Moment arm length, cm</td>
<td>( 6 ± 0.3 )</td>
<td>( 4.9 ± 0.4 )</td>
</tr>
<tr>
<td>Tendon force, N</td>
<td>( 1,783 ± 118 )</td>
<td>( 458 ± 36 )</td>
</tr>
<tr>
<td>Fascicular length, cm</td>
<td>( 3 ± 0.3 )</td>
<td>( 4.8 ± 0.4 )</td>
</tr>
<tr>
<td>Pennation angle, degree</td>
<td>( 42 ± 3 )</td>
<td>( 19 ± 2 )</td>
</tr>
<tr>
<td>Muscle force, N</td>
<td>( 2,427 ± 170 )</td>
<td>( 487 ± 40 )</td>
</tr>
<tr>
<td>Muscle length, cm</td>
<td>( 31.5 ± 1.5 )</td>
<td>( 30 ± 1.5 )</td>
</tr>
<tr>
<td>Muscle volume, cm(^3)</td>
<td>( 497.8 ± 22 )</td>
<td>( 152.7 ± 9 )</td>
</tr>
<tr>
<td>PCSA, cm(^2)</td>
<td>( 161.7 ± 13 )</td>
<td>( 264 ± 13 )</td>
</tr>
<tr>
<td>Specific tension, kN/m(^2)</td>
<td>( 150 ± 12 )</td>
<td>( 31.5 ± 2 )</td>
</tr>
</tbody>
</table>

Values are means ± SD (n = 6). In data set A, muscle moments during electrical stimulation and moment arm lengths, fascicular lengths, and pennation angles during maximum voluntary contraction (MVC) were included. In data set B, MVC moments, moment arm lengths at rest, and cadaveric fascicular lengths and pennation angles were included. M, measured; C, calculated; SH, superficial half of tibialis anterior (TA); DH, deep half of TA. *Values reported in Ref. 43. †Values obtained assuming that the muscle fascicle length-to-muscle length ratio is 0.06 in soleus (Sol) and 0.25 in TA (Ref. 43). ‡P < 0.01 compared with data set A; §P < 0.01 between TA and Sol in the same data set.
specific tension. Such error levels would not alter the biological meaning of the results obtained.

DISCUSSION

In the present study, the following three factors were considered for estimating specific tension from in vivo measurements.

Antagonistic Coactivation During MVC

Antagonistic coactivation results in a negative moment relative to the agonistic moment, and thus net MVC joint moments should be smaller than those generated by all the agonist muscles (22, 28). In the present study, however, lower muscle moments were generated during stimulation compared with the net MVC ankle moments (see Table 2). This result is not surprising, since we aimed at eliminating the moment contribution of nontested agonists. Sol is one of the eight ankle plantar flexors (Sol, gastrocnemius, plantaris, tibialis posterior, flexor hallucis longus, flexor digitorum longus, peroneus brevis, and peroneus longus), and TA is one of the four ankle dorsiflexors (TA, extensor hallucis longus, extensor digitorum longus, and peroneus tertius) in the human body. The Sol PCSA accounts for ~75% of the sum of PCSAs of all active plantar flexors (the gastrocnemius is considered to have negligible contribution due to its slack at the knee position studied here; see Refs. 12, 15, 16), and the TA PCSA accounts for ~40% of the sum of the PCSAs of all dorsiflexors (9, 10, 43). Considering the whole plantar flexor and dorsiflexor groups as single muscles (11, 28), according to the Sol and TA moments in our study, full plantar flexor activation (without dorsiflexor coactivation) would generate an ankle moment of ~143 Nm, and full dorsiflexor activation (without plantar flexor coactivation) would generate an ankle moment of ~56 Nm. The net ankle plantar flexion and dorsiflexion MVC moments in our study are smaller by ~15 and 20%, respectively, compared with the above theoretical values. These percent differences represent the reduction in agonistic (all plantar flexors or all dorsiflexors) joint moment due to antagonistic coactivation and lie within the range of such values reported in other studies (22, 28). The effect of antagonistic coactivation on agonistic joint moment has been traditionally neglected when estimating tendon forces (4, 10, 21, 31, 33), most probably because of difficulties in quantifying the antagonistic coactivation joint moment. Optimization methodologies or interpolation of EMG data from antagonist muscles could give an estimation of the antagonistic joint moment (22, 28). This joint moment would then have to be added to the net moment output to give an estimation of the joint moment generated by the agonists only. However, antagonistic coactivation may also impair, through reciprocal inhibition, full neural agonistic activation (41), indicating that even corrected joint moment values for antagonistic coactivation could still be erroneous.

The percutaneous stimulation protocol followed in the study was assumed to activate, selectively and fully, the studied muscles, and the moments recorded were considered to represent the true muscle moment-generating capacity. Some evidence of selective muscle activation was obtained in our experiments from EMG and ultrasound recordings of nonstimulated muscles. The possibility of partial muscle activation, however, should be considered carefully. A plateau in maximal joint moment in response to a voltage increase by only 25 V (15–20%) cannot ensure maximal muscle activation. Furthermore, it must be remembered that, for given voltage and stimuli frequency, the force generated on muscle electrical stimulation is a function of the number of motoneuron branches activated (18, 23). In muscles in which motor points are located away from the skin, the current field of percutaneous stimulation might result in only partial motoneuron activation. In the case of a superficial muscle such as TA, this is probably not an important factor. Sol, however, is proximally covered by gastrocnemius. Clearly, failure to activate all motoneurons, due to either pain intolerance or muscle/motor point anatomic location, is a limitation when seeking maximal contractile forces in stimulated human muscles. Thus our moment measurements may underestimate the actual moment-generating potential of TA and Sol. T2-weighed MRI offers, however, the possibility for measurements of the inactive muscle portion size during stimulation, from which appropriate corrections in the calculations of muscle volume, PCSA, and specific tension could be made (1).

Changes in Musculoskeletal Geometry From Rest to MVC

Changes in moment arm length. Cadaveric and resting-state moment arm length data have been traditionally used for calculating tendon forces from joint moments (4, 11, 21, 31). Moment arms at rest may be substantially shorter than those during MVC as a result of 1) muscle thickening (26, 27), 2) stretch effects in retinaculum systems surrounding the tendon (29), and 3) displacements of the center of rotation in the joint (26, 27, 29). Clearly, this contraction effect on
moment arm lengths should be taken into account when reducing joint moments to tendon forces.

Changes in pennation angle. The contractile muscle force component transmitted to the tendon depends on the angle between the fibers and the tendon, i.e., the muscle pennation angle. The pennation angle is larger during contraction than during rest (27, 30, 31). Again, this contraction effect should be taken into account in the vectorial analysis of muscle-tendon forces, but it has often been neglected and pennation angles from embalmed muscles have been used (11, 21).

In the present study, pennation angles were considered to be the angles between the fascicles and the aponeuroses. These angles would only result in a realistic vectorial analysis of forces if the aponeuroses lay on the action line of the muscle-tendon unit. We could not identify any angulation between the aponeuroses and tendons studied using ultrasonography; therefore, we represented the muscle by the geometric model shown in Fig. 5. This model type has often been employed for muscle-tendon force estimations (6, 11, 21, 27, 31). Some researchers, however, have used muscle models in which the aponeuroses lie at an angle relative to the tendon (e.g., Ref. 17). For a given tendon force, an angulation between the aponeuroses and the tendon would result in a smaller muscle force compared with the force calculated from the muscle model used in our study. An angulation of the aponeuroses with respect to the muscle-tendon unit action line needs further investigation and appropriate modeling. It must be remembered, however, that all oversimplified planar muscle models may be unrealistic (11, 25).

In the present study, both pennation angles and moment arm lengths were estimated from two-dimensional scan morphometrics. Furthermore, musculoskeletal geometry during MVC was considered to be representative of that during maximal tetanic stimulation. We have observed no differences in pennation angle and fiber length between MVC and maximal muscle tetanic stimulation (C. N. Maganaris and J. P. Paul, unpublished observations). Any differences in moment arm length between MVC and maximal muscle tetanic stimulation cannot be quantified at present.

In Vivo Estimations of PCSA

Cadaver-based PCSA estimates have often been taken into account when calculating muscle-specific tension (11, 21). The assumptions made are that 1) the muscle fascicle length-to-muscle length ratio of a given muscle is constant across cadavers and 2) muscle fascicles do not undergo shrinkage during fixation. Indeed, there is evidence to suggest that the first assumption may be valid; Wickiewicz et al. (43) and Friederich and Brand (9) reported a consistency in the muscle fascicle length-to-muscle length ratio in several muscles of the human lower extremity across different cadavers. However, the second assumption is unrealistic. A maceration-induced muscle bundle shrinkage by up to 20% has been reported (9). Such sizable shrinkage levels raise serious doubts for the representativeness of cadaver-based PCSA data with respect to values under in vivo conditions.

Neglecting the above three factors reduced the Sol specific tension by 31% ($P < 0.01$) and increased the TA specific tension by 325% ($P < 0.01$), thus indicating that enormous errors may have been made in estimations of specific tension using the traditionally followed approach (e.g., Refs. 11 and 21).

The use of data set A yielded specific tensions of 150 and 155 kN/m², respectively, in Sol and TA. These values are smaller than the average specific tension of 225 kN/m² obtained from whole animal muscle preparations (5, 34), but they are in good agreement with results from experiments on fiber type I-predominant muscles (specific tensions between 152 and 157 kN/m²; Refs. 34, 37, and 44). In fact, both Sol and TA are fiber type I-predominant muscles, with type II fibers accounting for 20–30% of the total fiber number in each muscle (19). The consistency between these results indicates that specific tension may be fiber-type specific. Although some experiments have challenged this view (8, 39), most of the reports in the literature from single fiber, motor unit, and whole in situ muscle experiments indicate that type II fibers are intrinsically stronger by 40–50% compared with type I fibers (3, 20, 24, 34, 37, 40, 44). Studies on human skinned and freeze-dried muscle fibers have reported that the specific tensions of type I and type II fibers are ~120 and 180 kN/m², respectively (8, 24, 40). The specific tension value of 120 kN/m² in type I single fibers is smaller by ~20% compared with Sol, TA, and other fiber type I-predominant muscles (34, 37, 44). A similar level of difference exists between the specific tension value of 180 kN/m² in type II single fibers and the specific tension of fiber type II-predominant muscles (34, 44). These differences may be accounted for by 1) extra volume occupied by noncontractile material (collagen, water, and capillaries) in in vivo and in situ muscles compared with in vitro single fibers, 2) the force contribution of the residual type fibers in the entire muscle, and 3) imperfect force transmission from fibers to tendons (11).

In conclusion, we showed that accurate estimates of human skeletal muscle intrinsic strength can be derived from measurements under in vivo conditions.

REFERENCES


