Aging, muscle fiber type, and contractile function in sprint-trained athletes

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1Department of Health Sciences, University of Jyväskylä, Jyväskylä, Finland; 2Department of Clinical Neurophysiology, University of Uppsala, Uppsala, Sweden; 3Department of Biology of Physical Activity, University of Jyväskylä; 4The Finnish Centre for Interdisciplinary Gerontology; 5KIHU — Research Institute for Olympic Sports, Jyväskylä, Finland

Submitted 10 March 2006; accepted in final form 9 May 2006

Korhonen, Marko T., Alexander Cristea, Markku Alén, Keijo Häkkinen, Sarianna Sipilä, Antti Mero, Jukka T. Viitasalo, Lars Larsson, and Harri Suominen. Aging, muscle fiber type, and contractile function in sprint-trained athletes. J Appl Physiol 101: 906–917, 2006. First published May 11, 2006; doi:10.1152/japplphysiol.00299.2006.—Biopsy samples were taken from the vastus lateralis of 18- to 84-yr-old male sprinters (n = 91). Fiber-type distribution, cross-sectional area, and myosin heavy chain (MHC) isoform content were identified using ATPase histochemistry and SDS-PAGE. Specific tension and maximum shortening velocity (V0) were determined in 144 single skinned fibers from younger (18–33 yr, n = 8) and older (53–77 yr, n = 9) runners. Force-time characteristics of the knee extensors were determined by using isometric contraction. The cross-sectional area of type I fibers was unchanged with age, whereas that of type II fibers was reduced (P < 0.001). With age there was an increased MHC I (P < 0.01) and reduced MHC IIx isoform content (P < 0.05) but no differences in MHC Ila. Specific tension of type I and Ila MHC fibers did not differ between younger and older subjects. V0 of fibers expressing type I MHC was lower (P < 0.05) in older than in younger subjects, but there was no difference in V0 of type Ila MHC fibers. An aging-related decline of maximal isometric force (P < 0.001) and normalized rate of force development (P < 0.05) of knee extensors was observed. Normalized rate of force development was positively associated with MHC II (P < 0.05). The sprint-trained athletes experienced the typical aging-related reduction in the size of fast fibers, a shift toward a slower MHC isoform profile, and a lower V0 of type I MHC fibers, which played a role in the decline in explosive force production. However, the muscle characteristics were preserved at a high level in the oldest runners, underlining the favorable impact of sprint exercise on aging muscle.

exercise; myosin heavy chain; single-fiber contractile properties; muscle strength

NORMAL AGING IN HUMANS is characterized by muscular atrophy and a loss of force-generating capacity. Earlier studies have related muscle atrophy to the decrease in the number and size of muscle fibers (45, 59). Furthermore, it is commonly reported that in aging muscles the reduction in individual fiber size is mainly confined to fast type II fibers, leading to a progressive decrease in the type II-to-type I fiber area ratio (41, 45, 69). Electrophoretic studies have revealed that older age is also associated with a change in the expression of myosin heavy chain isoforms (MHC) in favor of slow MHC I, which is probably a reflection of the selective atrophy of type II fibers (28, 36, 60). Myosin isoform content is the main determinant of a fiber’s contractile properties, and the aging-related shift in muscle MHC content has been found to play an important role in the decline in rapid force capacity of the lower-limb muscles (31, 36). Single-fiber studies, on the other hand, have provided evidence of an aging-related decrease in shortening velocity and specific tension of fibers expressing types I and/or Ila MHC isoforms (17, 38, 43), which could partially explain the impairment of whole-muscle contractile performance with aging.

The effect of long-term physical exercise on aging-associated changes in skeletal muscle structure and function has been investigated in endurance-trained people. These studies have generally demonstrated that continued endurance training maintains the aerobic capacity of muscles (29, 56) but does not slow down the aging-associated loss of muscle mass, atrophy of type II fibers, or decline in whole muscle force production (29, 36, 56, 71). Contrary to these findings, there is some evidence that long-term strength training provides a strong stimulus for the preservation of the structural and mechanical characteristics of skeletal muscle during aging (36, 53). One influential study supporting this view has been published by Kliigaard and coworkers (36). They found that elderly men with 12–17 yr of heavy resistance training had muscle fiber sizes, MHC composition, and muscle force characteristics similar to those of young adult control subjects.

To the best of our knowledge, no study has examined the interaction of age and long-term sprint training with the structural and functional properties of human skeletal muscle. Such a study would improve our understanding of the adaptability of the aging neuromuscular system to training characterized by explosive muscle actions. Because the muscle contractions in sprint training effectively stimulate fast motor units, it could be assumed that this type of training might counteract the age-dependent atrophy of type II muscle fibers. Additionally, long-term sprint training could provoke favorable adaptations in muscle strength characteristics, especially explosive force-production capacity. Evidence suggests that the ability to develop force rapidly may become a vital strength characteristic during aging because it relates to the capacity to carry out time-critical fast actions, such as correcting for a sudden loss of balance (67).

In the present study, we examined 91 active male sprinters across a wide age range from 18 to 84 yr to determine the influence of age and long-term explosive type of training on muscle structure and function. Specifically, the study addressed aging-related differences in fiber size and distribution, MHC isoform content, and the contractile properties of single muscle fibers expressing the type I and Ila MHC isoforms from the...
vastus lateralis muscle. The relationship between age, MHC isoform content, and force-production characteristics of the knee extensor muscles was examined as well.

**METHODS**

**Subjects.** Sixteen young adult (18–33 yr) and 75 master-aged (40–84 yr) male sprinters were recruited by means of personal letters from among the members of Finnish track and field organizations. To qualify for the study the master athletes had to have a long-term sprint training background and success in international or national championships in 100- to 400-m sprinting events. The young adult sprinters (personal records, 100 m: 10.97 ± 0.07; 200 m: 21.92 ± 0.19; 400 m: 49.54 ± 0.84 s) were selected for the study on the criterion that their age-adjusted sprint performance resembled that of the master athletes. According to the sprint test used in the study the runners were well matched for relative performance level: the 60-m sprint times were 109 ± 0.4, 110 ± 1.1, 107 ± 1.2, 109 ± 0.9, and 109 ± 1.6% of the indoor age-based world record times for 18- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 79-yr-old runners, respectively.

Training characteristics and competition performance of the subjects were studied by means of a questionnaire and personal interview (37) (Table 2). As expected, the data showed an aging-related increase in the number of years of prior sprint training and competition. The weekly training hours, training frequency, and strength training hours decreased with advancing age. The aging-related decrease in the weekly training and strength training hours occurred in a nonlinear fashion with the greatest change occurring from the youngest to the 40- to 49-year-old athletes.

All the subjects were apparently healthy as determined by reference to their detailed medical histories. Men over 55 yr were further evaluated for clinical evidence of cardiovascular diseases by a focused medical examination based on resting electrocardiograms and blood pressure measurements. Written consent was obtained from all subjects, who had been fully informed of the procedures, potential risks, and benefits associated with participation. This study was approved by the Ethics Committee of the University of Jyväskylä and confirmed to the Declaration of Helsinki.

**Anthropometry and muscle architecture.** Body height was measured with a height gauge and body mass with a balance beam scale. Total body fat percentage was assessed by use of bioelectrical impedance (Spectrum II; RJL Systems, Detroit, MI). Thigh length was measured with a ruler as the distance from the lateral condyle of the femur to the greater trochanter. Thigh circumference was measured with a tape at 50% thigh length. Muscle thickness and fascicle length were measured as the distance from the deep aponeuroses to the fascicle, which is located between the fiber fascicle and the deep aponeuroses. From the muscle sample was then transferred to a freezer at −80°C until the day of the histochemical and MHC analyses. The third piece was immediately placed in an ice-cold relaxing solution (in mmol/l: 100 KCl, 20 imidazole, 7 MgCl2, 2 EGTA, 4 ATP, pH 7.0; 4°C). Small bundles of ~25–50 fibers were dissected free from the muscle and tied to a glass microcapillary tube at ~110% resting length. The bundles were then placed in a skimming solution (relaxing solution containing glycerol; 50:50 vol/vol) at 4°C for 24 h and subsequently stored at −20°C for use within 3 wk, or treated with cryoprotectant (sucrose) solution for long-term storage at −80°C as described earlier (16). At the beginning of a set of experiments, a sucrose-treated frozen bundle was desiccated by stepwise lowering of the sucrose concentration of the relaxing solution until no sucrose remained. The bundle was thereafter stored in relaxing solution with glycerol at −20°C for a maximum of 2 wk.

**Myofibrillar ATPase histochemistry.** Serial 10-μm-thick transverse sections were cut on a cryostat (Leica CM 3000) at −24°C, mounted on glass slides, and stained for myofibrillar ATPase after acid (pH 4.37, 4.60) and alkaline (pH 10.30) preincubations (7). Six different fiber types (I, IC, IIC, IIA, IIAB, and IIB) were identified according to Staron et al. (64). However, because in each age group the type IC and IIC represented <1.0% and <0.5% of the fiber pool, respectively, values were collapsed into two groups, I and II. The pH 4.37 ATPase histochemistry was used as the main observation, and the pH 4.60 ATPase histochemistry was used for validation.

**Muscle biopsy.** Muscle samples were taken from the middle portion of the vastus lateralis of the dominant leg by using a needle biopsy technique (5) with suction. Before the biopsy, the surrounding area was cleaned with an antiseptic solution and then anesthetized with 1% lidocaine containing epinephrine. A needle (5 mm) was inserted into the muscle belly at a depth of ~1.5–2.5 cm below the surface of the skin, and, with the aid of suction, ~100–150 mg of muscle tissue were removed. Care was taken to achieve a consistent biopsy depth because of potential variation in fiber-type distribution and size from the superficial to deep vastus lateralis (58). The muscle sample was cleaned of any visible connective and adipose tissue and divided into three parts. The first part was frozen immediately in liquid nitrogen and stored at −80°C for future biochemical analysis. The second part of the sample was examined under a magnifying glass to determine the fiber orientation, mounted transversely in embedding medium on a cork disc, and frozen rapidly in isopentane cooled to −160°C in liquid nitrogen. The sample was then transferred to a freezer at −80°C until the day of the histochemical and MHC analyses. The third piece was immediately placed in an ice-cold relaxing solution (in mmol/l: 100 KCl, 20 imidazole, 7 MgCl2, 2 EGTA, 4 ATP, pH 7.0; 4°C). Small bundles of ~25–50 fibers were dissected free from the muscle and tied to a glass microcapillary tube at ~110% resting length. The bundles were then placed in a skimming solution (relaxing solution containing glycerol; 50:50 vol/vol) at 4°C for 24 h and subsequently stored at −20°C for use within 3 wk, or treated with cryoprotectant (sucrose) solution for long-term storage at −80°C as described earlier (16). At the beginning of a set of experiments, a sucrose-treated frozen bundle was desiccated by stepwise lowering of the sucrose concentration of the relaxing solution until no sucrose remained. The bundle was thereafter stored in relaxing solution with glycerol at −20°C for a maximum of 2 wk.

**Table 1. Physical characteristics of subjects by age group**

<table>
<thead>
<tr>
<th>Variable</th>
<th>18–33 yr</th>
<th>40–49 yr</th>
<th>50–59 yr</th>
<th>60–69 yr</th>
<th>70–84 yr</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>16</td>
<td>16</td>
<td>18</td>
<td>21</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Age, yr</td>
<td>24.3 ± 1.0</td>
<td>44.0 ± 0.9</td>
<td>53.9 ± 0.6</td>
<td>65.8 ± 0.6</td>
<td>75.3 ± 0.9</td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>178.0 ± 1.1d,e</td>
<td>180.5 ± 1.9a,d,e</td>
<td>175.5 ± 1.1b</td>
<td>172.7 ± 0.9b</td>
<td>171.1 ± 1.2b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>77.2 ± 1.4a</td>
<td>79.7 ± 1.9a,d,e</td>
<td>74.3 ± 1.4</td>
<td>71.2 ± 0.9b</td>
<td>69.8 ± 2.0b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>16.5 ± 0.9</td>
<td>13.3 ± 1.0</td>
<td>14.9 ± 0.9</td>
<td>13.6 ± 1.0</td>
<td>15.0 ± 1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Thigh length, cm</td>
<td>45.0 ± 0.5</td>
<td>47.1 ± 0.6</td>
<td>45.0 ± 0.4</td>
<td>45.1 ± 0.7</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Thigh circumference, cm</td>
<td>55.9 ± 0.6d,e</td>
<td>54.5 ± 0.7d,e</td>
<td>52.2 ± 0.5a,d,e</td>
<td>50.7 ± 0.5b,cb</td>
<td>48.7 ± 0.8b,cd,e</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VL thickness, cm</td>
<td>2.61 ± 0.08d,e</td>
<td>2.35 ± 0.09a</td>
<td>2.08 ± 0.1a</td>
<td>2.10 ± 0.09a</td>
<td>1.96 ± 0.08b</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VL fascicle length, cm</td>
<td>7.91 ± 0.49</td>
<td>7.90 ± 0.35</td>
<td>7.11 ± 0.41</td>
<td>7.99 ± 0.27</td>
<td>7.38 ± 0.27</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are means ± SE. N, number of subjects; VL, vastus lateralis muscle. ANOVA P values and location of significant differences are shown. a,b,c,d,e Group significantly (P < 0.05) different from the 18- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 84-yr-old groups, respectively. NS, not significant.
they were not included in the analyses. Figure 1A illustrates cross sections from a young and an old muscle stained for mATPase activity after alkaline preincubation, discriminating between slow and fast fibers. The fiber area and relative proportion of the various fiber types were analyzed from the stained cross sections by using a microscope combined with a computer-assisted image-analysis system (Tema, Scanbeam, Hadsund, Denmark) (61). Relative fiber-type distribution was calculated from an average of 508 ± 29 fibers in each biopsy sample. The measurements of fiber cross-sectional area comprised an average of 228 ± 15 type I, 162 ± 10 type IIA, 60 ± 5 type IIA, and 53 ± 6 type IIB fibers.

**Homogenate electrophoresis.** The MHC isoform content of the biopsy samples was determined by SDS-PAGE according to previously described methods (1) with slight modifications. For the analysis, 10–15 cryosections (10 μm) from each biopsy were placed into 700 μL of a lysine buffer containing 10% (vol/vol) glycerol, 5% (vol/vol) 2-mercaptoethanol, 2.3% (mass/vol) SDS in 62.5 mM Tris-HCl buffer (pH 6.8) and heated for 10 min at 60°C. A small amount of the muscle extracts (3–10 μL) was loaded into each lane of the SDS-PAGE gel system consisting of stacking gel with 3% acrylamide and separating gel with 6% acrylamide and 30% glycerol. The gels were run on an electrophoresis device (Bio-Rad Protein II xi Cell) at 4°C at a constant voltage of 70 V for 42 h. After the run, the gels were fixed for 24 h in 5% acetic acid and 50% methanol, stained with Coomassie blue, and destained in 7.5% acetic acid and 5% methanol overnight or until the background was clean. In the stained gels three distinct protein bands could be separated and identified as MHC I, IIA, or IIX isoforms according to their migration characteristics. The relative proportion of each MHC isoform in a biopsy sample was determined by using a densitometric system (Cream 1D, Kem-En-Tec aps, Copenhagen, Denmark). Examples of SDS-polyacrylamide gel and densitometric tracings illustrating separation of the MHC isoforms in samples from a younger and an older subject are shown in Fig. 1B. It has been shown previously that the slowest migration protein band in humans is analogous to the MHC IIX, not the MHC IIB isoform, in rats (54) and therefore the MHC IIX nomenclature is used in this study.

**Single-fiber contractile measurements.** On the day of an experiment, the fibers were placed for 30 min in a relaxing solution containing 0.5% Brij-58 (polyoxyethylene 20 cetyl ether; Sigma Chemical). The fibers were mounted in an experimental apparatus similar to the one described previously (20, 51), leaving an average fiber segment length of 2.02 ± 0.51 mm (mean ± SD, range 1.05–3.60 mm) exposed to the solution between connectors to a force transducer (model 403, Cambridge Technology) and a direct-current torque motor (model 300H, Cambridge Technology). The apparatus was mounted on the stage of an inverted microscope (Zeiss Axiovert-35, Carl Zeiss, Oberkochen, Germany). When the fiber was in the relaxing solution, sarcomere length (SL) was set to 2.76 ± 0.04 μm (range 2.66–2.89 μm) by adjusting the overall segment length. The segments were observed at a magnification of ×320. Prints of the fiber segments were taken with a videoprinter (P71E, Mitsubishi Electric). Sarcomere length, segment width, and the length of the segment between the connectors were measured directly from the microscope via a TV overlay with the aid of a digitizer connected to a microcomputer (Videoplan, Kontron Bildanalyes Gmbh, Munich, Germany). The final magnification with the image analysis system on the TV screen was ×1,480. Fiber SL was measured routinely in the fibers during maximal activation. Fiber depth was measured by recording the vertical displacement of the microscope nosepiece while focusing on the top and bottom surfaces of the fiber. Cross-sectional fiber area was calculated from the width and depth, assuming an elliptical circumference. Specific tension was calculated as maximum tension (P0) normalized to cross-sectional area, and was corrected for the 20% swelling that is known to occur during skinning (51).

Relaxing and activating solutions contained (in mM) 4 MgATP, 1 free Mg2+, 20 imidazole, 7 EGTA, 14.5 creatine phosphate, and sufficient KCl to adjust the ionic strength to 180. The pH was adjusted to 7.0. The free Ca2+ concentrations were 10−4−9 M (relaxing solution) and 10−4.5 M (maximum activation solution) and are expressed as pCa (−log [Ca2+]). Apparent stability constants for Ca2+ -EGTA were corrected for temperature and ionic strength (13). The computer program of Fabiato (13) was used to calculate the concentrations of each metal, ligand, and metal-ligand complex. Immediately preceding each activation, the fiber was immersed for 10–20 s in a solution with a reduced Ca2+-EGTA buffering capacity (50). This solution was identical to the relaxing solution except that EGTA was reduced to 0.5 mM, which resulted in more rapid attainment of steady tension during subsequent activation and helped to preserve the regularity of cross-striations during activation.

Maximal unloaded shortening velocity (V0) was measured by the slack-test procedure (12). Fibers were activated at pCa 4.5 and, once steady tension was reached, various amplitudes of slack (ΔL) were rapidly introduced (within 1–2 ms) at one end of the fiber. The time (Δt) required to take up the imposed slack was measured from the onset of the length step to the beginning of tension redevelopment. For each amplitude of ΔL, the fiber was reextended while relaxed to minimize nonuniformity of sarcomere length. A straight line was fitted to a plot of ΔL vs. Δt, using a least-squares regression, and the slope of the line was recorded as V0 for that fiber. P0 was calculated as the difference between the total tension in the activating solution (pCa 4.5) and the resting tension measured in the same segment while in the relaxing solution. All contractile measurements were carried out at 15°C. The contractile recordings were accepted in subsequent analyses if a V0 value was based on linear regressions including four or more data points, and data were discarded if the coefficient of reliability (r) for the fitted line was less than 0.97, if P0 changed more than 10% from first to final activation, or if SL during isometric contraction was not constant during activation.
tension development changed by more than 0.10 μm compared with SL when the fiber was relaxed (51).

After mechanical experiments, each single fiber, dissolved in sample buffer, was loaded onto a 6% SDS-PAGE gel and run at 120 V for 24 h at 10°C as described earlier (44). Gels were subsequently silver stained and MHC isoforms were determined.

Sprint performance. Eighty-six subjects participated in the sprint and strength performance tests. Sprint performance was determined by standing-start 30- and 60-m sprint trials performed on an indoor tartan running track (air temperature 19–20°C). Times for the sprint tests were measured by use of double-beam photocell gates connected to an electronic timer (starting line was 0.7 m behind the first photocell gates). The testing session was preceded by a ~30- to 45-min general warm-up such as the subjects were accustomed to (jogging, stretching) and submaximal practice runs to familiarize them with the procedures. The subjects performed two maximum-effort trials at both sprint distances with 5–7 min of rest between runs. During the sprint tests all subjects were spiked track shoes. For the 60-m sprints the test-retest r value varied from 0.93 to 0.98 and the coefficient of variation (CV) from 0.7 to 0.9% in the different age groups.

Strength measurements. Maximal bilateral isometric strength and force-time parameters of the knee extensor muscles were measured with an electromechanical dynamometer (23). In the test, the subjects were in a seated position with 107° knee and 110° hip angles (180° with an electromechanical dynamometer (23). Times for the force tests were measured by use of double-beam photocell gates connected to an electronic timer (starting line was 0.7 m behind the first photocell gates). The testing session was preceded by a ~30- to 45-min general warm-up such as the subjects were accustomed to (jogging, stretching) and submaximal practice runs to familiarize them with the procedures. The subjects performed two maximum-effort trials at both sprint distances with 5–7 min of rest between runs. During the sprint tests all subjects were spiked track shoes. For the 60-m sprints the test-retest r value varied from 0.93 to 0.98 and the coefficient of variation (CV) from 0.7 to 0.9% in the different age groups.

Dynamic explosive strength was evaluated by means of a vertical counter movement jump (61). The test was performed on a contact mat (Newtest, Oulu, Finland) connected to a digital timer (±0.001 s) that recorded the flight time of the vertical jump. The height of rise of the body’s center of gravity was calculated from the flight time. During the jump the hands were kept on the hips to minimize differences in technique. After the practice jumps the subjects performed three to four maximal trials, separated by 1–1.5 min of rest, and the highest jump with an acceptable technique was used for the analyses. The flight time of the two highest jumps showed r and CV values within the range of 0.94–0.99 and 0.6–2.2% in the different age groups.

Statistical analysis. Linear and curvilinear regression analyses were performed to determine the association between age and the dependent variables. The linear model provided a similar or better fit with the present data (excluding sprint performance and training data) than the nonlinear model and is therefore the one described below. In addition, ANOVA was used to compare the age groups in terms of their physical characteristics (Table 1), training and selected performance characteristics (Table 2), and contractile properties of single muscle fibers (Table 3). When a significant difference was found, Tukey’s and Tamhane post hoc tests were used for the specific comparisons. Comparisons of slope differences between cross-sectional areas of the different type II fibers with respect to age (Fig. 2) were assessed by using the F-test. Differences in cross-sectional and relative fiber-type area between different fiber types within age groups were analyzed by ANOVA (Figs. 3B and 4). Pearson correlation coefficients were used to measure the association between MHC I isoform content and relative type I fiber area as well as correlations of strength, sprint, and jump performance with MHC II content. Statistical significance was set at P < 0.05 for all analyses.

RESULTS

Physical characteristics. Body height and mass were lower in the older age groups, whereas percentage body fat did not differ with age (Table 1). Thigh length showed no significant differences with age, but there were aging-associated decreases in thigh circumference and vastus lateralis muscle thickness. The estimated fascicle length of the vastus lateralis muscle did not differ with age.

Fiber cross-sectional area. Type I fiber cross-sectional area showed no differences with advancing age (Fig. 2A). However, there was a progressive aging-associated reduction (P < 0.001) in the cross-sectional area of different type II fibers (Fig. 2, B–D), leading to a decline (P < 0.001) in type IIA/I, IIB/I, and IIB fiber area ratios with age (Fig. 3A). The slopes of the aging-related declines in the fiber areas were not different for

Table 2. Training and performance characteristics of subjects by age group

<table>
<thead>
<tr>
<th>Variable</th>
<th>18–33 yr</th>
<th>40–49 yr</th>
<th>50–59 yr</th>
<th>60–69 yr</th>
<th>70–84 yr</th>
<th>P</th>
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<tbody>
<tr>
<td>Training</td>
<td></td>
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<tr>
<td>Years of sprint training</td>
<td>13.2 ± 1.3&lt;sup&gt;a,b,c,d&lt;/sup&gt;</td>
<td>28.1 ± 2.0</td>
<td>28.8 ± 2.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>35.1 ± 4.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>34.3 ± 4.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.001</td>
</tr>
<tr>
<td>Training hours, h/wk</td>
<td>11.5 ± 0.68&lt;sup&gt;a,b,c,d&lt;/sup&gt;</td>
<td>6.7 ± 0.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.6 ± 0.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.1 ± 0.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.9 ± 0.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Frequency, sessions/wk</td>
<td>5.9 ± 0.3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.9 ± 0.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.4 ± 0.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.2 ± 0.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.0 ± 0.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Strength training, h/wk</td>
<td>5.2 ± 0.4&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
<td>2.2 ± 0.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.5 ± 0.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.8 ± 0.1&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.9 ± 0.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Performance†</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>60-m sprint, s</td>
<td>7.00 ± 0.03&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
<td>7.62 ± 0.09&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>8.03 ± 0.09&lt;sup&gt;b&lt;/sup&gt;</td>
<td>8.61 ± 0.08&lt;sup&gt;b&lt;/sup&gt;</td>
<td>9.53 ± 0.19&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Vertical jump, cm</td>
<td>52.5 ± 1.62&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>42.0 ± 0.97&lt;sup&gt;a&lt;/sup&gt;</td>
<td>33.1 ± 0.89&lt;sup&gt;b&lt;/sup&gt;</td>
<td>28.3 ± 0.91&lt;sup&gt;b&lt;/sup&gt;</td>
<td>22.7 ± 0.99&lt;sup&gt;b&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Isometric force, N</td>
<td>3.865 ± 0.23&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.307 ± 0.187&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.872 ± 1.860&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.440 ± 1.200&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.310 ± 1.175&lt;sup&gt;b&lt;/sup&gt;</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are means ± SE. ANOVA P values and location of significant differences are shown. <sup>a,b,c,d</sup>Group is significantly (P < 0.05) different from the 18- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 84-yr-old group, respectively. <sup>†</sup>Number of subjects for the performance tests was 14, 15, 17, 21, and 19 for the 18- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 84-yr-old group, in that order.
Cross-sectional area, specific tension and maximum velocity of unloaded shortening in single muscle fibers classified according to expression of MHC composition in younger and older subjects

<table>
<thead>
<tr>
<th>Age group</th>
<th>Type I</th>
<th>Type I/IIa</th>
<th>Type IIa</th>
<th>Type IIax</th>
<th>Type IIx</th>
</tr>
</thead>
<tbody>
<tr>
<td>18–33 yr</td>
<td>2,400</td>
<td>2,400</td>
<td>3,200</td>
<td>3,200</td>
<td>2,800</td>
</tr>
<tr>
<td>53–77 yr</td>
<td>3,200</td>
<td>3,200</td>
<td>3,200</td>
<td>3,200</td>
<td>2,800</td>
</tr>
</tbody>
</table>

Values are means ± SE. *P* values reflect level of significance. Statistical analyses were only restricted to muscle fibers expressing the type I or IIa MHC isoform, because of the small number of fibers expressing the type IIx MHC isoform. Compared with the younger group, the older athletes had 21 and 37% smaller (*P < 0.05*) cross-sectional areas in muscle cells expressing the type I (3,450 ± 150 μm², *n* = 43 vs. 4,360 ± 160 μm², *n* = 68) and IIa (3,080 ± 190 μm², *n* = 53 vs. 4,930 ± 140 μm², *n* = 47) MHC isoforms, respectively. The smaller type IIa fiber size and the more pronounced decline in type IIa fibers than in type I is in accordance with morphological measurements from the biopsy cross sections. However, a smaller type I muscle fiber size in the old subjects was only observed at the single muscle fiber level. The advantage with measurements at the single muscle.

Fiber-type distribution. The relative distribution of the histochemically determined fiber types did not differ with age. For all subjects combined, the fiber-type percentages were 45.7 ± 1.3 for type I, 32.0 ± 1.0 for IIa, 11.4 ± 0.6 for IIAB, and 9.8 ± 0.9 for IIB.

Relative area of fiber types. When fiber-type distribution was combined with the corresponding fiber area to form the relative fiber-type area, there was an increase in the area of muscle occupied by type I fibers as a function of age (*P < 0.05*) (Fig. 4). Within the fast fiber subtypes no aging-associated differences in relative area of fiber types were observed, although there was a trend toward a decrease (*P = 0.05*) in the area of type II fibers with age. Among fast fibers, type IIa fibers occupied the largest area in all age groups (*P < 0.001*), whereas no differences were seen in the relative areas of types IIAB and IIB.

MHC isoform content. The analysis of MHC isoform composition revealed an aging-related increase in the relative content of MHC I (*P < 0.01*) and a decrease in that of MHC IIx (*P < 0.05*), whereas no age difference was observed in MHC IIa expression (Fig. 5). MHC I isoform content was highly associated with the corresponding histochemically determined relative area of type I fiber (*r* = 0.92, *P < 0.001).

Single-fiber contractile properties. A total of 144 single membrane permeabilized muscle fiber segments out of 302 fibers fulfilled the criteria for acceptance for contractile measurements in the younger (18–33 yr, *n* = 8) and older group (53–77 yr, *n* = 9) (Table 3). No significant difference was found in the relative number of single-fiber preparations that fulfilled the criteria for acceptance in the younger (44%) and older subjects (51%).

Muscle fiber size was measured in a total of 265 muscle fiber segments in the younger (*n* = 138) and older (*n* = 127) subjects at a fixed sarcomere length assuming an elliptical cross section of the fiber segment. Because of the paucity of muscle fibers expressing the IIX MHC isoform (*n* = 7) and fibers coexpressing the type I and IIa (*n* = 2) or the IIa and the IIX (*n* = 11) MHCs, aging-related differences in fiber size were restricted to analyses of fibers expressing the type I and IIa MHC isoform. Compared with the younger group, the older athletes had 21 and 37% smaller (*P < 0.05*) cross-sectional areas in muscle cells expressing the type I (3,450 ± 150 μm², *n* = 43 vs. 4,360 ± 160 μm², *n* = 68) and IIa (3,080 ± 190 μm², *n* = 53 vs. 4,930 ± 140 μm², *n* = 47) MHC isoforms, respectively. The smaller type IIa fiber size and the more pronounced decline in type IIa fibers than in type I is in accordance with morphological measurements from the biopsy cross sections. However, a smaller type I muscle fiber size in the old subjects was only observed at the single muscle fiber level. The advantage with measurements at the single muscle.
fiber level is that cross-sectional area is measured at a fixed sarcomere length. However, a selective aging-related decrease in type II muscle fiber area has been repeatedly documented in human quadriceps muscle and a bias due to the smaller number of muscle fibers measured at the single muscle fiber level is a more likely explanation for the different results on type I muscle fiber size in old age.

Statistical analyses of differences in contractile properties between younger and older subjects were restricted to muscle cells expressing the type I and IIa MHCs because of the paucity of cells expressing other MHCs or combinations of MHCs. The maximum force generated by the single muscle fibers was higher ($P < 0.001$) in the younger than in the older subjects. However, this difference was primarily due to the larger size of the muscle fibers in the younger subjects, and no aging-related difference was observed in specific tension, i.e., when maximum force was normalized to the cross-sectional area of the fibers (Table 3). In accordance with previous studies in single human muscle fiber segments $V_o$ increased in the following order: β/slow (type I) → IIa → IIaX → IIx MHCs in both the younger and older subjects (Table 3). An aging-related difference in contractile speed was observed in muscle cells expressing the type I MHC isoform, i.e., $V_o$ was 24% slower ($P < 0.05$) in the muscle fibers from the older subjects. The $V_o$ values of muscle fibers expressing the IIa MHC isoform were not significantly different between younger and older subjects.

**Sprint and strength performance.** Table 2 shows the 60-m sprint, vertical counter movement jump and maximal isometric muscle strength ($F_{max}$) values among the different age groups. There were clear age group differences in all of these performance measures. The rate of decrease in vertical jump height (11.1%/decade) and $F_{max}$ (8.3%/decade) was best described as a linear function of age. The rate of lengthening in the 60-m sprint times with age was curvilinear, showing an accelerated rate around age 65–70.

![Fig. 2. Relationship between age and mean cross-sectional area of type I (A) and different type II muscle fibers [IIA(B), IIAB(C), and IIB (D)]. Regression lines are shown for cross-sectional areas of type II fibers, which declined significantly with age. The slope of the regression line among the fast fibers was similar.](http://jap.physiology.org/)

![Fig. 3. A: ratio of type II-to-type I fiber mean cross-sectional areas of vastus lateralis muscle in different age groups. There was a significant decline ($P < 0.001$) in type II/A, IIAB/I and IIIB/I fiber area ratios with age. B: difference between the mean cross-sectional area ($\mu m^2$) of each type II and type I fibers in different age groups. ***$P < 0.001$, **$P < 0.01$, *$P < 0.05$ compared with type I fiber.](http://jap.physiology.org/)
The force-time curves on the absolute scale indicated an aging-related lengthening ($P < 0.001$) in the times needed to reach specific force levels up to 2,000 N (Fig. 6A). Correspondingly, there was an aging-associated decline ($P < 0.001$) in the absolute RFD. The loss of RFD from $25,270 \pm 1,576$ N/s in the youngest group to $13,216 \pm 1,234$ N/s in the oldest group represented a decrease of 9.7% per decade. The time taken to reach RFD lengthened ($P < 0.001$) from $54 \pm 4$ ms (18–33 yr) to $104 \pm 12$ ms (70–84 yr) with increasing age.

When the force in the force-time curves was calculated as a percentage of the maximal force produced, there continued to be an aging-related lengthening ($P < 0.001–0.05$) in the time needed to reach 10–80% of $F_{\text{max}}$, with the significance of association being strongest at force levels between 10–50% of the maximum (Fig. 6B). Moreover, adjustment for $F_{\text{max}}$ also decreased the influence of age on the rate of force development (normalized RFD), although a small but significant decline ($P < 0.05$) remained. In all age groups normalized RFD occurred early in the force-time curve, i.e., before 25% of maximal force was reached (range 21.1–24.9%).

Relation between force production and MHC isoform expression. When all subjects were considered, absolute maximal RFD was associated with the total relative MHC II (IIa+IIX) isoform content ($r = 0.28$, $P < 0.01$). The times taken to reach the specific force levels between 100 N and 1,500 N were also related to MHC II ($r = -0.41$ to $-0.31$, $P < 0.001–0.01$). Adjusting for age, the times to the force levels up to 1,000 N remained correlated with MHC II ($r = -0.35$ to $-0.32$, $P < 0.01$), whereas the effect of MHC II on absolute RFD was no longer statistically significant. Furthermore, the correlation coefficients between $F_{\text{max}}$ and MHC II did not reach the level of statistical significance whether unadjusted or adjusted for age.

The association of normalized force production with MHC II content was also evaluated, to take into account differences in the strength levels of the athletes. For the overall sample, a relationship between normalized RFD and MHC II content was found ($r = 0.27$, $P < 0.05$). In addition, the times taken to reach the force levels of 10–70% of $F_{\text{max}}$ were associated with MHC II, the correlation being progressively weaker with increasing force levels (10–40%, $r = -0.41$ to $-0.38$, $P < 0.001$; 50%, $r = -0.33$, $P < 0.01$; 60–70%, $r = -0.28$ to $-0.23$, $P < 0.05$). After controlling for age, normalized RFD ($r = 0.23$, $P < 0.05$) and the times taken to reach 10–50% of $F_{\text{max}}$ ($r = -0.35$ to $-0.26$, $P < 0.01–0.05$) continued to show a significant correlation with MHC II content.

With regard to dynamic performance, vertical jump height correlated with MHC II content (overall sample: $r = 0.39$, $P < 0.001$; age-adjusted: partial $r = 0.32$, $P < 0.01$). The 60-m running times were related to MHC II only when the subjects were taken as a whole ($r = -0.26$, $P < 0.05$).

DISCUSSION

The present study examined aging-related differences in muscle fiber and force-production characteristics in competi-

![Fig. 4. Proportion (%) of overall muscle fiber cross-sectional area occupied by different fiber types in the vastus lateralis muscle of subjects in different age groups. Relative area of type I fibers increased with age ($P < 0.05$) whereas that of type IIB showed aging-associated decline ($P = 0.05$).](http://jap.physiology.org/)

![Fig. 5. Relationship between age and relative content of MyHC I (A), IIa (B), and IIX (C) isoforms. Lines in A and C are linear regressions.](http://jap.physiology.org/)
tive male sprinters with a very long history of systematic training. The main findings were as follows: 1) With increasing age there was a progressive decrease in the cross-sectional area of fast type II fibers, whereas the area of slow type I fibers remained constant. 2) The percentage of different fiber types showed no age-associated differences; however, when expressed as their relative area, an increase in the area occupied by type I fibers and decrease in type IIb fibers with age was found. 3) There was an aging-associated increase in the proportion of slow MHC I with a concomitant decrease in MHC IIx isoform content. 4) The preferential decrease in the size of muscle cells expressing fast MHC isoforms (type IIa) was confirmed in single membrane permeabilized fiber segments, i.e., at a fixed sarcomere length. 5) $P_o$ was lower in muscle cells from older than young subjects, but this difference was secondary to the smaller size of muscle fibers in older age, and force normalized to fiber cross-sectional area (specific tension) was not affected by age in muscle cells expressing the type I or IIa MHC isoforms. 6) The decline in the explosive force-production capacity of the knee extensor muscles was associated with both quantitative and qualitative changes in the slow MHC isoform, i.e., there was a shift toward a slower MHC composition as well as a decrease in shortening velocity in muscle cells expressing the slow MHC isoform.

**Fiber cross-sectional area and distribution.** The present data showed that sprint-trained vastus lateralis muscle was characterized by a large cross-sectional area of both slow and fast fibers. We can put these results into greater perspective by comparing them with previous data by Häkkinen and coworkers (24–26) obtained from untrained middle-aged (40 yr) and older (70 yr) men with the same analytical methods. This comparison, shown in Fig. 7A, indicates that the older 70-yr-old sprinters had on average 65, 69, and 28% larger cross-sectional area of type I, IIA, and IIB fibers than age-matched untrained men. In fact, the oldest sprinters had fiber area values equivalent to those of untrained 40-yr-old men.

Although the quadriceps muscle of the present sprint athletes appeared to be strongly influenced by regular training, muscle fiber cross-sectional area decreased with age. Consistent with several previous studies in untrained (41, 45, 69) as well as in endurance-trained humans (36, 56, 71), we observed that the reduction in fiber area is mainly confined to fast fibers, leading to a decrease in the type II-to-type I fiber area ratio with age (Figs. 1–3). Our finding that type II fiber cross-sectional area decreases progressively in the vastus lateralis muscle, starting as early as at ~30 yr of age, is also supported by some previous studies of untrained people (45, 52). On the other hand, in the present sprinters the extent of the aging-related decrease in the different type II fiber areas was similar. This result differs from some earlier reports (10, 21) according to which in untrained people type IIB fibers are much more susceptible to the effects of aging than type IIA fibers.

In the present study the distribution of type I and II fibers or different fast fiber subtypes in sprint-trained athletes was unaltered with age. However, when the proportion of fiber types was expressed as their respective cross-sectional areas, the sprinters showed an aging-associated increase in the muscle area occupied by type I fibers (Fig. 4), owing to a decrease in the size of individual fast fibers. On average across all subjects, 53–60% of their muscle area was occupied by fast type II fibers. These values are similar to those observed in earlier studies in young adult sprinters of the same caliber (2, 62).
The measurement of MHC isoform composition in the muscle homogenates showed an aging-related increase in the relative proportion of slow MHC I with a concomitant decrease in MHC IIx content. In line with our results, other electrophoretic studies of muscle homogenates have generally reported that in normally active and endurance-trained people aging is associated with a shift toward greater relative MHC I content in the vastus lateralis. Previous experiments on human limb muscles have demonstrated that given types of histochemically determined muscle fibers mainly express their analogous MHC isoforms (except that type IIB express MHC IIx) (54, 63). This seems to be consistent with the findings that the increase in MHC I with age proceeds in parallel with an increase in the relative area of type I fibers, induced by the atrophy of fast fibers (36). In our study, we too found a strong association between relative type I fiber area and MHC I isoform profile.

The results on fast MHC isoforms revealed that a number of master athletes had an absence of MHC IIx, whereas all of the young adult athletes showed detectable amounts of the MHC IIx isoforms (Fig. 5C). Previous research on young subjects suggests that the loss of MHC IIx could be explained by the increased recruitment of fast fibers containing MHC IIx resulting in deactivation of the MHC IIx genes and activation of the MHC IIa genes, according to the gene default theory (19).

Training studies have also indicated that a decrease in MHC IIx can occur rapidly, within a period of 1–2 wk of intense training (19, 65). As in most other studies with athletes, it was not possible to standardize the level of training for several weeks before the study, and thus the possibility exists that in certain master athletes the absence of MHC IIx reflects an increase in the intensity and duration of premeasurement training. Alternatively, it cannot be ruled out that the loss of MHC IIx in these runners is a result of the aging-dependent transformation of type IIx to slower MHC isoforms and/or selective loss of type IIB fibers containing MHC IIx (40).

Contractile function of single muscle fibers. The slowing of contractile speed in muscle cells expressing the slow MHC isoform in older age is in accordance with the aging-related slowing of V_o in muscle cells expressing the type I and or the type IIa MHC in humans (11, 38, 43) and rodents (46, 68). However, this slowing in human muscle fibers has been reported in subjects with a sedentary lifestyle, and the similar slowing observed in the physically very active subjects in this study indicates that this slowing is not the result of a decreased physical activity level in old age. In addition to the slowing in contractile speed, an aging-related loss in specific tension has been reported in sedentary individuals at the single muscle fiber level (11, 38, 43), but no significant difference in specific tension was observed between the strength- and sprint-trained young and old men in this study. It is accordingly suggested that the aging-related loss reported in specific tension at the single muscle fiber level may be a consequence of a more sedentary lifestyle in old age. This is supported by the increased specific tension reported in response to strength training in old sedentary women (15). Trappe and coworkers (70), on the other hand, did not observe a change in either shortening velocity or specific tension in old sedentary humans.

In vitro motility studies after extraction of myosin from millimeter short muscle fiber segments from human percutaneous muscle biopsies have shown that the aging-related slowing at the single muscle fiber level is primarily caused by altered structural-functional properties of the motor protein myosin (33, 34, 42). Multiple slow MHC isoforms have been identified in skeletal muscle (14, 18, 35, 47), and the possibility cannot be excluded that the slowing in old age is associated with an aging-related upregulation of an isoform not, or less, expressed in young individuals. However, there is to our knowledge no evidence of the expression of novel slow MHC isoforms in old age with migration properties similar to the H9252/slow (type I) MHC on SDS-PAGE. The longer turnover rate of myosin in humans in old age (4) and the increased risk for posttranslational modifications of the motor protein is advanced as a more likely cause underlying the slowing in contractile speed. There are several potential mechanisms by which a protein with a very slow turnover rate, such as myosin, can be modified during the aging process. Nonenzymatic glycosylation (glycation) of myosin has been reported to increase in old age (66), and glycation has a dramatic negative effect on myosin function that could explain the slowing.
observed in old age (3, 8, 57). In addition, there are other mechanisms by which myosin may be modified during the aging process, and oxidation of cysteines has been put forward as a mechanism underlying structural-functional changes of myosin in old age (48, 55).

Relation between muscle force production, MHC isoform expression, and age. An important objective of this study was to examine the relation of the isometric force characteristics of the knee extensor muscles to MHC composition and age. As expected, our data demonstrated an aging-related decline in absolute maximal force that could largely be attributed to a loss of contractile tissue, as indirectly indicated by our fiber size and muscle thickness findings. The results on the absolute scale also showed an aging-related decrease in RFD, and a lengthening of the time taken to reach the specific submaximal force levels (Fig. 6A). When the data were normalized for maximal force, the older runners continued to demonstrate longer times to force production, particularly in the lower part of the force-time curve (Fig. 6B). In line with our findings, several other studies have reported that older subjects exhibit slower isometric force production, whether determined by maximal voluntary (9, 27, 67) or electronically stimulated contractions (29).

In the present study we also found that aging-related slowing of normalized force production (RFD and time to reach 10–70% of Fmax) was associated with a decrease in the relative content of MHC II isoforms. This connection is likely to reflect the differences in intrinsic properties between type I and type II MHC fibers. As reported here or elsewhere, fibers with a fast MHC have significantly higher shortening velocity (30, 44) and develop tension faster (30, 49) than fibers expressing the slow MHC isoforms. This study also indicates that older age in sprinters is associated with a reduction in the shortening velocity of single MHC I fibers, but it is unclear whether this could be a factor influencing the slowing of isometric force production. However, considering that in rapid isometric muscle actions the motor units are recruited in the order of size, e.g., the smaller slow-type before the larger fast-type motor units (32), then an aging-related decline in the shortening velocity of type I MHC fibers could potentially reduce the rate of force production in the initial phase of muscle contraction.

Finally, it should be emphasized that although the older sprint athletes in this study showed an aging-related reduction in force production, their maximal and fast force-production capacity remained at a high level compared with that of untrained men. Figure 7, B and C, shows the Fmax and RFD data for the middle-aged and older runners in this study and data obtained from age-matched normally active men in previous studies using the same strength testing methods and device (22, 23). The oldest sprinters had Fmax and RFD values that were ~31 and 47% higher, respectively, than those of 70-yr-old nonathletes. These values were actually at the same level as the values obtained for untrained subjects at the age of 40 yr. Moreover, in terms of dynamic explosive strength, the oldest subjects in this study had vertical jump values twice as high as those reported earlier for untrained men aged 71–73 yr (6).

Methodological considerations. Our study has certain methodological limitations that should be pointed out. First, the results shown in this paper were obtained with a cross-sectional design and may have been influenced by genetic and constitutional factors. Longitudinal studies in athletes who remain highly trained are required to confirm the present cross-sectional observations. Second, a potential confounding factor in this investigation is that needle biopsy specimens are not fully representative of the whole muscle. Repeated sampling of the same muscle has shown that the CV for fiber distribution and size is 5–10% (58). Third, by recruiting top young and master athletes with the same relative level of competitive performance there were aging-related differences in training patterns, which may have affected the results. For example, it is possible that the reduction in resistance training in older runners may have contributed to the decline in fast fiber size with age. Finally, a strength of this study is that we had a good sample of continuously trained sprinters in 10-yr age groups from young adult to old age. The wide age range, including intermediate age groups, provided a clearer picture of the nature (i.e., rate and linearity) of aging-related changes in muscle fiber and force-production characteristics than would have emerged if only two discrete age groups had been compared.

In conclusion, the present results suggest that highly trained competitive sprint runners experience the typical aging-associated reduction in the size of fast fibers, shift toward a slower MHC isoform profile, and lower V02 in muscle cells expressing the type I MHC isoform, playing a role in the decline in explosive force-production capacity. On the other hand, our master sprinters demonstrated considerably larger fiber size, intact maximum force normalized to cross-sectional area at the single muscle fiber level, and higher maximal and explosive strength characteristics than those previously reported for untrained older people. It is likely, therefore, that systematic sprint training is an effective stimulus in maintaining muscle fiber structure and force-production characteristics during aging. Although our findings are not directly applicable to untrained people, they tend to favor the view that, to minimize the effect of aging on the neuromuscular system, optimal overall physical training might require actions that impose explosive-type overload on muscle.

ACKNOWLEDGMENTS

We thank Erkki Helkala, Aila Ollikainen, Vuokko Kovanen, and Arimantas Lionikas for valuable assistance with the data collection and analysis, and all the subjects participating in this study.

GRANTS

Support for this study was provided by the Finnish Ministry of Education and the Peurunka-Medical Rehabilitation Foundation to H. Suominen and the Swedish Research Council (08651), Swedish Sports Research Council, and National Institutes of Health (AR-045627, AR-047318, AG-014731) to L. Larsson.

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