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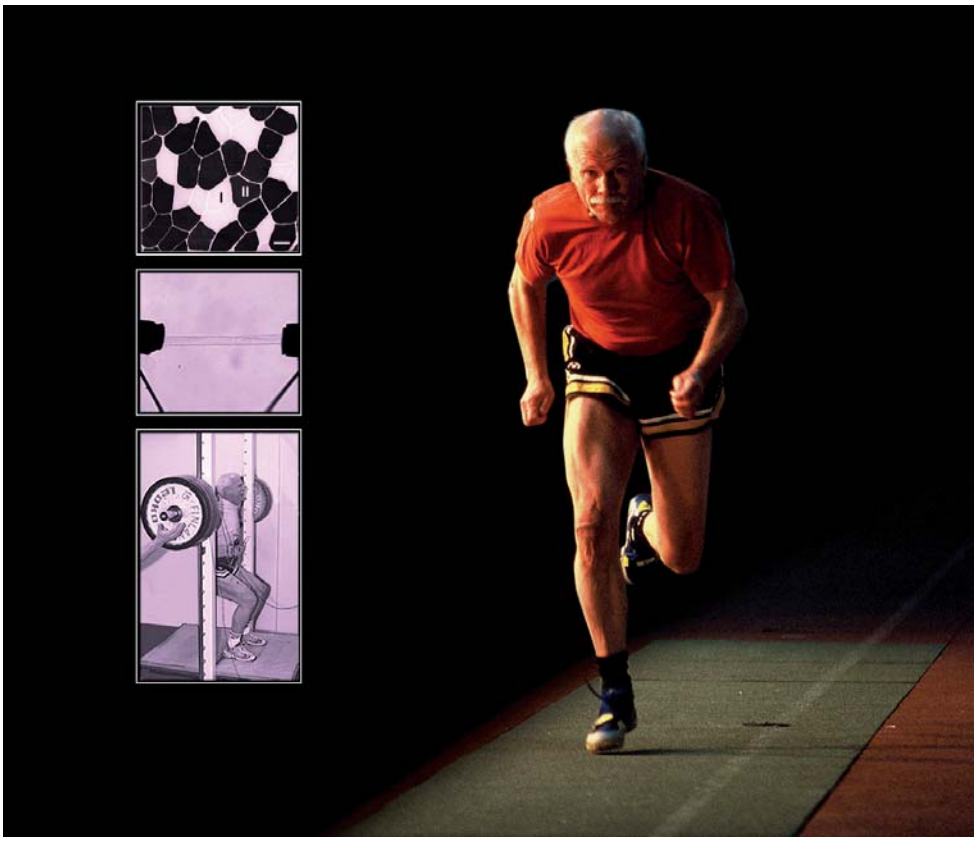
137

Marko T. Korhonen

Effects of Aging and Training on Sprint Performance, Muscle Structure and Contractile Function in Athletes



JYVÄSKYLÄN YLIOPISTO

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JYVÄSKYLÄ 2009

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Cover picture: Jorma Manninen, world champion and 100 m world record (12,50 s) holder in the 65- to 69-year age category in 2007. Photograph by Risto Antikainen

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ABSTRACT

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Cross-sectional studies were conducted to examine sprint running, anaerobic energy production and muscle properties in male sprinters aged 17-88 years. In addition, a 20-week training intervention was carried out to determine whether older runners can further improve their neuromuscular and performance characteristics by a greater emphasis on strength training. With age, sprint performance declined gradually (5-6%/decade). The slowing of maximum speed was characterized by a reduction in stride length and an increase in contact time along with lower ground reaction forces (GRF) and smaller leg and vertical stiffness during the contact phase. Stride frequency showed small decline while swing time remained unaffected with age. Variability in the biomechanical parameters that showed good repeatability (CV 1-6%) was the same in the older as younger runners, and no age effect was seen in the symmetry of the measures. $[La]_{b\ peak}$ declined with age after races over 100-400 m, the decrease becoming more evident from age 70. Running times correlated inversely with $[La]_{b\ peak}$. Leg muscle thickness, type II fiber size and myosin heavy chain (MyHC) II isoform content decreased with age, while type I fiber size, fiber distribution and fascicle length showed no age differences. In single type I and IIa MyHC fibers, neither force adjusted for fiber size nor contractile speed differed between the groups. There was an age-related decline in maximal (8-9%/decade) and explosive (10-11%/decade) isometric and dynamic leg strength. The differences in maximal, but not in explosive, isometric strength were eliminated when normalized for muscle thickness. Muscle thickness was the strongest predictor of GRF in the braking phase, while the countermovement jump explained most of the variance in push-off GRF. The sprint training, including heavy-resistance and high-power strength exercises, resulted in significant gains in maximal and explosive strength and improvements in force production during running. The improvements were mainly related to hypertrophic adaptations. The results show that the deterioration in sprint performance with age is a complex phenomenon that may be affected by the interaction of changes in biomechanical, neuromuscular and metabolic factors. A major contributor appears to be reduced muscle mass, caused partially by decreased type II fiber size, which affects the GRFs required to achieve fast running speeds. However, habitual sprint training seems to maintain speed, strength and glycolytic energy production at high levels into older age and is effective in preventing the age-related decline in single fiber function and fascicle length. The data also suggest that to maximize the training effects on fast fibers, rapid strength and speed performance, the optimal training regimen requires a strength training component.

Key Words: aging, master athlete, muscle, single fiber, speed, sprint running, strength

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Jyväskylä, June 2009 Marko Korhonen

LIST OF PUBLICATIONS

The present thesis is based on the following original papers, which are referred to in the text by their roman numerals:

I Korhonen MT, Mero AA, Suominen H (2003). Age-related differences in 100-m sprint performance in male and female master runners. Med Sci Sports Exerc 35 (8): 1419-1428.

II Korhonen MT, Mero AA, Alén M, Sipilä S, Häkkinen K, Liikavainio T, Viitasalo JT, Haverinen MT, Suominen H (2009). Biomechanical and skeletal muscle determinants of maximum running speed with aging. Med Sci Sports Exerc 41 (4): 844-856.

III Korhonen MT, Suominen H, Viitasalo JT, Liikavainio T, Alén M, Mero AA (2009). Variability and symmetry of force platform variables in maximum-speed running in young and older athletes. Submitted for publication.

IV Korhonen MT, Suominen H, Mero A (2005). Age and sex differences in blood lactate response to sprint running in elite master athletes. Can J Appl Physiol 30(6): 647-665.

V Korhonen MT, Cristea A, Alén M, Häkkinen K, Sipilä S, Mero A, Viitasalo J, Larsson L, Suominen H (2006). Aging, muscle fiber type, and contractile function in sprint-trained athletes. J Appl Physiol 101: 906-917.

VI Cristea A*, Korhonen MT*, Häkkinen K, Mero A, Alén M, Sipilä S, Viitasalo JT, Koljonen MJ, Suominen H, Larsson L (2008). Effects of combined strength and sprint training on regulation of muscle contraction at the whole-muscle and single-fibre levels in elite master sprinters. Acta Physiol (Oxf) 193: 275-289.
*contributed equally

In addition, some previously unpublished results will be presented.

ABBREVIATIONS

ANOVA	Analysis of variance
BF	M. biceps femoris
BW	Body weight
CMJ	Countermovement jump
CSA	Cross-sectional area
CTRL	Control group
CV	Coefficient of variation
EMG	Electromyography
EX	Experimental group
F_{brake}	Resultant GRF of braking contact phase
F_{push}	Resultant GRF of push-off contact phase
$Freq_{step}$	Step frequency
$Freq_{str}$	Stride cycle frequency
GL	M. gastrocnemius lateralis
GM	M. gastrocnemius medialis
GRF	Ground reaction force
iEMG	Integrated electromyography
KE	Knee extensors
L_{step}	Step length
L_{str}	Stride cycle length
$[La]_{b\ peak}$	Peak blood lactate concentration
mATPase	Myofibrillar adenosine triphosphatase
MyHC	Myosin heavy chain
PF	Plantar flexors
RF	M. rectus femoris
RFD	Rate of force development
SD	Standard deviation
SDS-PAGE	Sodium dodecyl sulphate polyagrylamide gel electrophoresis
SE	Standard error
SHBG	Sex hormone-binding globulin
SJ	Squat jump
ST	Specific tension
T	Total testosterone
t_{aer}	Aerial time
t_c	Ground contact time
t_{str}	Stride cycle time
t_{sw}	Swing time
V_o	Maximum unloaded shortening velocity
VIM	M. vastus intermedialis
VL	M. vastus lateralis
VM	M. vastus medialis
V_{max}	Maximum running velocity
1 RM	One repetition maximum

CONTENTS

1 GENERAL INTRODUCTION

The physiology of human body is characterized by its ability to undertake diverse physical activity. During aging, however, a gradual deterioration takes place in structure and function in the different tissues of the body, leading to declines in endurance, muscle strength and speed capabilities. Speed and explosive strength/power, as evaluated by the ability to achieve high running velocity and by the ability to produce the greatest possible force in the shortest possible time, are particularly susceptible to age-related changes in non-athletes. These physical qualities are recognized as major components of successful performance in many sports and recreational physical activities. Moreover, fast force production is needed to adequately perform the tasks of daily living while, conversely, decline in it could increase the risk for mobility limitation in advanced age.

In view of the importance of preserving the ability to run and develop force rapidly, lifestyle factors that could have effect on the maintenance of speed performance with aging merit consideration. In this connection, investigation of the age-related differences in performance and associated skeletal muscle characteristics of sprint-trained master athletes may provide valuable information. Several authors have emphasized that master athletes constitute an ideal research model, as in these individuals the confounding influences on physical changes of decreased physical activity and diseases are minimized and thus reflect the aging process itself (Bortz and Bortz 1996, Morley 2000, Hawkins et al. 2003, Tanaka and Seals 2003, Spirduso 2005, Lazarus and Harridge 2007, Rittweger et al. 2009). Studying master athletes is also likely to sustain motivation while at the same time lowering the risks to musculoskeletal and cardiovascular systems attendant on the measuring and training of maximal speed and force production.

Until now, studies of the age differences on sprinting ability have focused primarily on sprint track records. Few data have been gathered on the biomechanical characteristics associated with the age-related decline in sprint performance. For example, little is known about kinematic stride cycle parameters, and no data exist regarding age differences in ground reaction

forces, which is a representative performance measure reflecting the sum of segment forces transferred to the ground. Similarly, it is unknown whether aging affects anaerobic energy production during sprint running in master athletes.

In order to understand the age-related decline in speed and strength it is important to have reliable knowledge of the age-related changes in the skeletal muscles. Studies in young sprinters have shown that large muscle mass composed predominantly of fast fibers is critical for sprint performance as it allows great forces to be generated quickly (Mero et al. 1992, Andersen et al. 2000, Weyand and Davis 2005). The question arises: How do skeletal muscle structural and functional properties change with age in systematically trained sprint athletes and what is the contribution of different muscle characteristics to the age-related decline in sprinting speed? It also remains unclear how much and what types of training are optimal in limiting the decline in muscle mass and functional losses in force and power in master athletes.

Therefore, the following review of the literature and summary, based on the six original articles, focus on the effects of aging on sprinting ability and its determinants in high-performance male sprinters. This thesis also examined whether in older sprint runners speed performance and neuromuscular characteristics can be further increased by applying the modern training methods used with young athletes.

2 REVIEW OF THE LITERATURE

2.1 Age-related changes in sprint performance

Sprint running performance can be evaluated from several different perspectives. This first section reviews studies that are closely related to the present research project. Therefore, references to 100-m competition performance times, velocity curves and biomechanical characteristics of maximum speed running in male athletes are included. Since some of these areas have not been investigated previously in master sprint runners, the literature is supplemented with information found in young sprinters.

2.1.1 Competition performance times

Age group records for the 100-m sprint have been investigated in a number of studies and provide the basis for understanding the effect of aging on the ability to run fast. Competitive sprint performance overall is dependent not only on a high maximum speed but also a short reaction time, the ability to produce fast starting acceleration and speed-endurance to maintain maximum velocity for as long as possible (Mero et al. 1992, Ae et al. 1994, Delecluse 1997). Accordingly, success in sprinting is affected by the interaction of various neuromuscular and technical capabilities and anaerobic metabolic capacity.

The first scientific reports on the relationship between age and world record performances of running appeared in the middle of the 1970s, about a decade after the first organized masters' track competitions (Moore 1975, Salthouse 1976). These studies indicated that the men's world record speed for the 100-400-m events declined at a rate of about 0.8% per year from the peak level attained at age 20-25 until 55 years of age, with steeper declines thereafter (Salthouse 1976). However, since the 1970s all age group sprint world records have improved considerably.

Examination of the current age records for the 100-m sprint shows that the age at which a steeper, exponential decline in performance starts, has risen to about 80 years of age (Fig. 1A). In record performances the overall decline in mean running velocity is 32.5% (0.56%/yr) in the span of about 60 years (from 10.32 m/s at age 22 to 6.97 m/s at age 80) (Fig. 1B). The improvements in master sprint track records are likely to reflect the larger number of elite runners (from various countries) competing in masters sprint running as well as more systematic year-long training (Spirduso et al. 2005). While most of the current age-group 100-m sprint records have been achieved in this decade the improvements have been small in recent years. This suggests that the current records are increasingly more representative of the real performance capabilities that can be attained in different age groups.

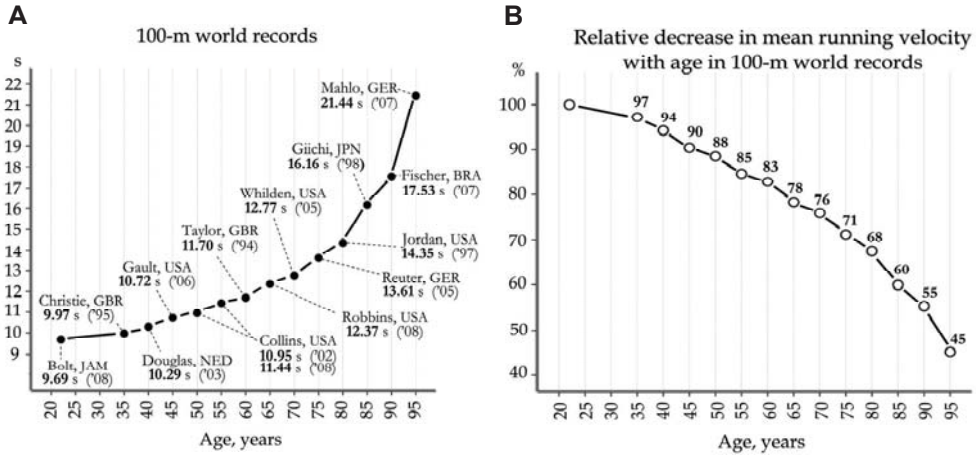

FIGURE 1 The official 100-m world records (year 2008) in open and each 5-year age (≥35 yr) classes in men (A), and relative decrease of the mean running velocity in age-based world records from that of the open class (B). The year when the record was made is shown in brackets.

Only one investigation providing longitudinal data on sprinting performance ability from peak levels at age 20-25 until age 70-75 was found in the literature (Conzelmann 1997). The retrospective study carried out by Conzelmann (1997) identified the best German male runners with repeated participation in sprinting competition. In the selected athletes the rate of longitudinal decline in the mean running velocity for the 100-m event from age 20-25 to ages 70-75 was about 0.3-0.4%/year and smaller than the cross-sectional decline (~0.6%/year) of the ten highest ranked all time national performances. However, it would seem that competitive status influences longitudinal change in sprinting ability, and in those runners who have reached a very high level of competitive performance during their adulthood the rate and magnitude of sprint performance decline will be greater than in those with a lower level of performance at young adult age. In relation to this, an interesting observation is that the individual best 100-m performance times of the world record holder for the women's 100- and 200-m events at 35-, 40- and 45-year-old groups (Merlene

Ottey) have over 10 years declined from 10.74 s (36 yrs) to 11.34 s (46 yrs) corresponding to a decrease of the mean running velocity of about 0.53%/year. Given that she is at a stable, optimal level of training (still trying to qualify for major championships), the decrement in performance reflects mainly biological aging itself.

2.1.2 Biomechanical characteristics of sprint performance

VELOCITY CURVE. Velocity curve, which describes acceleration from a resting position to maximum velocity and deceleration at the end of the run, has often been used to evaluate overall sprint performance. The distance required to reach maximum velocity appears to be dependent on the runner's performance level. For example, Gundlach et al. (1963) found that in the 100-m sprint, lower level male runners (100 m: 13.0-14.4 s) reached their maximum velocity of about 8.2 m/s at around 30 m, whereas in the fastest sprinters (100 m: 10.8-11.7 s), a maximum velocity of ~9.6 m/s was reached at about 45 m. Biomechanical studies conducted at the major international championships since the 1980s have provided information on velocity curves among the world's fastest sprinters. For example, the laser radar results of the recent World Championships showed that male 100-m finalists (9.86-10.10 s) consistently attained their maximum velocity (11.6-11.9 m/s) at about 60 m, after ~6.5 s (Kersting 1999). However, the increase in velocity after about 20 m was quite small and the sprinters were capable of reaching ~89% and ~95% of their maximum velocity already at the 20-m (~2.9 s) and 30-m (~3.84 s) marks. The decrease in velocity from the peak value to the finishing line of the run ranged from 4% to 10% for male finalists (Kersting 1999). Further, analyses of 100-m performance in national and international level sprinters have suggested that although each velocity section is important, the final time is not as much affected by the ability to accelerate (0-30 m) as the maximum attainable velocity and the time it can be maintained (Bruggemann and Glad 1990, Li 1991, Andersson and Alnes 1996, Gajer et al. 1999, Kersting 1999). In support of these results, the current 100-m world record performance (9.69 s) was characterized by the maintenance of maximum velocity (11.8-12.2 m/s, unofficial values) throughout the latter half (50-90 m) of the race (Eriksen et al. 2008, Tucker and Dugas 2008). Although several studies have been conducted on velocity curves, it remains unclear how increasing age affects velocity curve characteristics.

STRIDE CYCLE PARAMETERS. At the first level of mechanical analysis, running velocity is the product of stride frequency and stride length, thus velocity can be increased by changing either or both of the two components. The interaction between stride frequency and stride length is different during the acceleration, maximum velocity and deceleration phases. For example, in the 60-m and 100-m events elite young sprinters typically reach their maximum stride frequency between 10 and 20 m, at which distance stride length is about 75% of its value during the maximum velocity phase. On the other hand, during the last 10-20 m

of the 100-m sprint stride frequency typically decreases and stride length increases.

Age-related differences in kinematic stride patterns during maximum velocity running have been described in two studies (Hamilton 1993, Roberts et al. 1997a). In the competition study by Hamilton (1993), the results (derived from graphs) for 83 men showed that maximum velocity declined from about 9.9 m/s among 30- to 40-year-old runners to about 6.6 m/s in runners aged 80-89. This decline in maximum velocity was primarily related to reduction in stride cycle length (2 x step length) from about 4.6 m to 3.1 m. Stride cycle frequency showed a small decline with advancing age and was explained by an increase in contact time while swing time, the other component of the stride frequency, remained largely unaffected. Similar results for the relationship between age, velocity and stride cycle parameters were reported by Roberts et al. (1997a) who studied five master runners aged 60-65 years and three middle distance runners aged 20-22 years. Their results further indicated that the older runners were not able to generate forces and velocities in the swing limb comparable to those of the younger runners, but the timing of limb movement with regard to peak values was not affected by age. In addition, one major change in sprint performance with age is a decrease in the range of motion in the hip and knee joints (Hamilton 1993, Roberts et al. 1997a).

GROUND REACTION FORCES. While previous studies have provided insights into age changes in kinematics during constant velocity sprinting, how older age affects ground reaction force (GRF) characteristics in sprint running is still unknown. Ground-leg interaction is the major factor in sprint running because it is during the contact phase of the step cycle that segmental forces can act on and thus influence horizontal speed. GRFs consist of vertical, horizontal anterior-posterior and medial-lateral directional components. Vertical force is the greatest GRF in magnitude and dominates the resultant force vector. It is likely to have strong effect on the minimum time needed to be spent on the ground to produce a sufficient impulse to support body weight and to create a long enough aerial time for repositioning the swing leg (Weyand et al. 2000). Horizontal (anterior-posterior) force produces a negative braking effect at touchdown and a positive propulsive effect during the latter part of the contact phase. For optimal force production during sprinting, the runner should be able to rotate the body's center of gravity as fast as possible forward of the contact phase to direct vertical acceleration to the intended horizontal movement. Technical aspects, such as high backward velocity of the foot prior to touchdown (Ae et al. 1992), and a small horizontal distance between the front support foot and the body's center of gravity (Mann 1998) have been suggested to favor effective horizontal force production. Of note, recent evidence suggests that certain kinematic characteristics that are thought to be related to efficient stride pattern (e.g., good knee flexion and high knee lift during forward swing, and short braking distance and active knee and ankle joint extension of the contact leg) may not be optimal for the world best sprinters capable of running about 12 m/s (Ito et al. 2008).

Mero and Komi (1986, 1987) combined vertical and horizontal forces and used mean net resultant GRFs as a specific force indicator in maximum speed in good (100 m: 10.62 s, n=5) and average male (100 m: 10.96 s, n=6), and female sprinters (100 m: 12.22 s, n=8). The GRFs increased with sprinting ability in both the braking (~1600-2200 N; 2.7-2.9 BW) and propulsive (~1200-1800 N; 2.1-2.4 BW) phases. The contact time for the braking phase was about 10% and for the propulsive phase about 3% shorter in the fastest runners (43 ms, 58 ms, respectively) than in the two other groups. It was suggested that maximum speed is limited by the magnitude of the force that the runner is capable of producing during the minimum contact time, whereas stride frequency showed no differences between runners of different ability. These findings are in general agreement with the study of treadmill running by Weyand et al. (2000) that examined the average vertical GRF and stride parameters in 33 subjects with different sprinting abilities (6.2–11.1 m/s). The results indicated that maximum speed was related to the magnitude of GRFs, which positively affected stride length, contact time, and stride frequency, whereas swing time did not differ between the runners.

MUSCLE GROUPS IN SPRINTING. Electromyographic (EMG) data have provided an insight into the contribution of various muscle groups and neuromuscular coordination during sprinting. With respect to the muscle group involvement during maximum speed (Fig. 2), many authors have emphasized the critical role of the hamstrings and gluteal muscles in bringing about downward and backward leg movement throughout the contact phase (Simonsen et al. 1985, Mann et al. 1986, Mero and Komi 1986, Wiemann and Tidow 1995, Jönhagen et al. 1996, Mann 1998). It has also been suggested that the adductor muscles contribute to hip extension and counteract the abducting effect on the leg of the gluteal muscles (Mann and Hagy 1980, Wiemann and Tidow 1995). Knee extensors and plantar flexors are of particular importance during the braking phase of contact in providing stability for the knee and ankle joint angles. Of the knee extensors, the double-joint rectus femoris could also play a role in hip flexion during the swing phase. However, much of the force required to reposition the limb could be produced passively through elastic recoil and transfer of energy between body segments (Chapman and Caldwell 1983, Weyand et al. 2000). During acceleration, when the body has a pronounced forward lean, the knee extensors can also be important producers of forward propulsion (Mero and Komi 1990, Wiemann and Tidow 1995, Delecluse 1997). The plantar flexors (gastrocnemius, soleus) in conjunction with dorsiflexor (tibialis anterior) muscles have a major influence how effectively the forces of different body segments are transferred to the ground. In this regard, high muscle preactivity and stiffness are thought to play an important role in the toleration of high impact loads and in enabling the effective utilization of tendomuscular elasticity during the brief contact phase (Cavagna et al. 1971, Dietz et al. 1979, Gollhofer et al. 1984, Mero and Komi 1987).

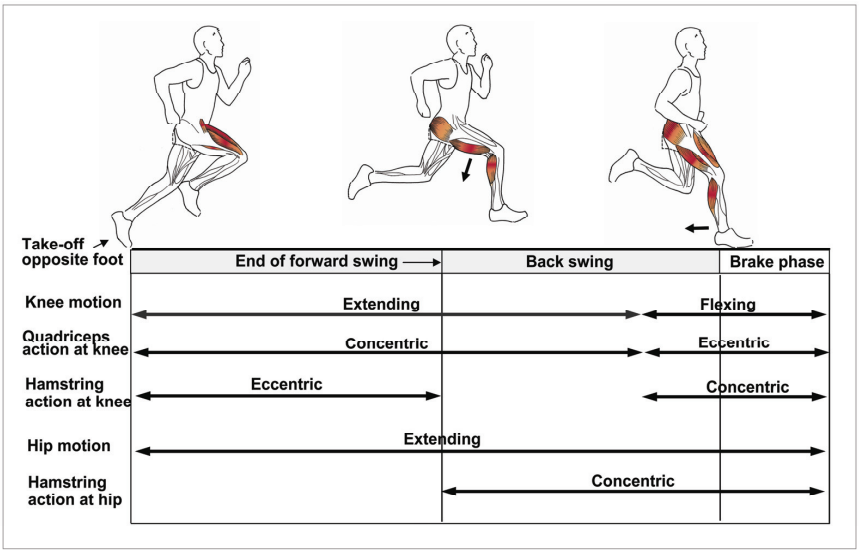

FIGURE 2 Dominant quadriceps and hamstring muscle contraction modes during later part of swing phase and braking phase of the contact in sprinting. Redrawn with modifications from Sugiura (2008).

During the stride cycle, the same muscles require a high level of intramuscular coordination to perform maximal movements with different forms of contraction (Fig. 2). For example, in the end of the forward swing the hamstrings do a substantial amount of eccentric work to decelerate the swing of the forward leg, which allows the storage of elastic energy, used in subsequent concentric action during back swing (Simonsen et al. 1985). However, the exact pattern of force production (muscle-tendon interaction) during the critical contact phase of human sprinting remains to be determined. Interestingly, although not definitely comparable, evidence from direct measurement of ankle extensors force, length and activity of the running animals suggests that at contact the muscles operate near-isometrically with a stretch-shorten pattern of the tendinous tissue (Roberts et al. 1997b, Biewener et al. 1998, Gabaldon et al. 2008). The top sprinting speed of humans depends on the magnitude of the forces that can be developed during the contact phase (Weyand et al. 2000), and near-isometric force generation could favor force output as the muscle contracts over the high force region of its force velocity curve (Gabaldon et al. 2008). Good balance between the eccentric, concentric and isometric strength of muscle as well as between agonists and antagonists (e.g. quadriceps vs. hamstrings) is very important for the performance and injury prevention of athletes of different ages and must be considered in training (Jönhagen et al. 1994, Kallinen and Alen 1994, Galloway and Jokl 1996, Aagaard et al. 1998).

LOWER EXTEMITY STIFFNESS. Sprint running is characterized by fast reactive contact that requires an appropriate mechanical stiffness of the integrated musculoskeletal system. The exact process by which muscle stiffness is regulated is not fully understood, but may reflect a complex interaction of centrally-programmed prelanding activation and reflex potentiation after the impact phase (Dietz et al. 1979), stiffness of tendons and other connective tissues (Butler et al. 2003) and muscle force-generating capacity. During running the vertical GRF and center of mass displacement trajectory shows a linear relationship, and in this case the contact leg is considered to act as a linear spring (Brughelli and Cronin 2008). Therefore, it is possible to express the complex multijoint body system as a simple spring-mass system and to describe the lower extremity stiffness (elasticity) during the contact phase. For analytical purposes, stiffness of the leg spring (k_{leg}) is calculated from the ratio of maximal vertical GRF to the change of leg spring length at the middle of the contact phase. Vertical stiffness (k_{vert}) is defined as the ratio of the maximal vertical GRF to the vertical length change of the runner's center mass.

A number biomechanical studies have shown an increase in k_{vert} with increasing running velocity (Luhtanen and Komi 1980, Mero and Komi 1986, Farley and Gonzalez 1996, Arampatzis et al. 1999, Morin et al. 2005). In particular, k_{vert} may determine the minimum ground contact time the runner is capable of effectively using and is thus reflected in stride frequency (Farley and Gonzalez 1996). In the early experiment, Mero and Komi (1986) calculated vertical stiffness ("apparent spring constant") from kinematic data and found that the higher eccentric stiffness of better runners was related to the smaller vertical displacement of the center of mass. More recent studies suggest that the increase in k_{vert} at higher velocities is due to a decrease in vertical displacement of the center of mass (Δy) together with an increase in vertical GRF, but with a greater contribution from the decrease in Δy (Farley and Gonzalez 1996, Arampatzis et al. 1999).

Some studies also indicate an increase in k_{leg} with increasing running speed (Arampatzis et al. 1999, Brughelli and Cronin 2008). The results of the study by Arampatzis et al. (1999) on 13 runners showed that the progressive increase in k_{leg} with increasing velocity at slow to moderate velocities (2.5-6.5 m/s) was mainly due to increases in GRF, while there was no change in the leg spring compression. A more recent study by the same authors in 10 experienced male sprinters suggest increases in k_{vert} and k_{leg} but no change in leg compression (ΔL: from foot to hip) even at higher (~9.2-9.5 m/s) running velocities (Stafilidis and Arampatzis 2007). Other studies have suggested that leg stiffness remains constant from slow to moderate running speeds because the leg spring experiences larger compression with increased Fz_{max} (Farley and Gonzalez 1996). It has been argued that these contradictory findings for k_{leg} can be partially explained by different velocities, and that k_{leg} increases at velocities over about 5 m/s (Brughelli and Cronin 2008). Moreover, the use of different methods of calculation may have influenced the length changes in spring-mass models, and may thus have influenced k_{leg} (Arampatzis et al. 1999).

Limited evidence suggests an age-related reduction in leg stiffness during maximal vertical countermovement jump (Liu et al. 2006) and a decline in ankle joint stiffness in intensive drop jumping on sledge apparatus (Hoffren et al. 2007). However, the stiffness is likely to be motor-task specific and it remains unclear whether aging leads to a decline in mechanical stiffness during maximal sprint running.

VARIABILITY AND SYMMETRY OF BIOMECHANICAL MEASURES. In performance analysis, the reliability refers to the degree of stability and consistency of the data. In biomechanical experiments, especially when measuring basic temporal-spatial and GRF variables with a force platform, the errors related to instrumentation and scoring are small and the major source of variability is expected to be due to the biovariation associated with human movement (Hamill and McNiven 1990). In contrast to walking and endurance running, studies evaluating variability during sprinting have been very limited in scope. In fact, a search of the literature disclosed only one reliability study on maximum-speed running (Mero and Komi 1986), but this was limited to two GRF variables. The few other studies have addressed accelerated sprinting (Hunter et al. 2004, Bradshaw et al. 2007), and no data exist for older runners. However, evidence from studies of accelerated sprinting suggests that intra-subject variability differs with the parameter of interest. For example, the study by Hunter et al. (2004) showed that the variation in L_{step}, $Freq_{step}$, t_c and t_{aer} was low, with CVs in the range of 1.2-2.9% for 4 steps. In contrast, parameters based on vertical displacement of the body's center of gravity or horizontal braking GRF showed higher variability (6-11.5%).

Bilateral symmetry, defined as agreement between the parameters of the dominant vs. non-dominant, or right vs. left leg, is another aspect of sprinting that has been remained virtually unstudied. The few sprint studies reporting the values for both sides have not evaluated the data in terms of the magnitude and significance of asymmetry for the reliability of measurements (Atwater 1982, Andersson and Alnes 1996). Examination of the results suggests, however, that perfect symmetry can not be assumed. From data collection standpoint information on biomechanical data concerning symmetry is very important. If asymmetry between the limbs exists, monolateral trials may not provide representative values.

2.2 Energy production during sprint running

Muscle contractile (actomyosin power stroke) as well as non-contractile (Ca^{2+} and Na^+/K^+ pump) events are dependent on the supply of chemical energy from adenosine triphosphate ($ATP \rightarrow ADP + P_i + Energy$), and brief all-out exercise requires very high rate of ATP resynthesis for muscle contractions to continue. ATP can be regenerated rapidly by phosphocreatine (PCr) depletion

and glycolysis with the production of lactic acid (Boobis et al. 1982). With continued sprint exercise, the oxidative metabolism also begins to play a role in energy production. Our current understanding of the interaction of these energy systems during brief whole-body sprint exercise is largely based on muscle biopsy studies of changes in muscle metabolites (e.g., ATP, PCr, pH, lactate, glycogen) and oxygen. However, muscle biopsy sampling instantly after sprint exercise is very challenging in many aspects and lacks applicability to a wider population. For example, one concern is that the small time delay (<1 min) between the end of exercise and freezing of the muscle tissue can influence PCr concentration (Söderlund and Hultman 1986). Therefore, many researchers have used peak post-exercise blood lactate concentration and oxygen deficit measurements to provide "indirect" information on energy production during sprinting. The following section reviews the literature on the direct measurement of the interaction of the energy systems during track sprinting and the use of the post-exercise blood lactate method in sprint exercise studies. Since no information exists on aging sprint runners, studies of blood lactate response to brief maximal exercise in master sprint swimmers and untrained people are discussed.

2.2.1 Interaction of energy systems during sprint exercise

Two muscle biopsy studies of anaerobic metabolic response to 100- and 400-m track sprinting were found in the literature. Hirvonen et al. (1987) determined muscle metabolites in a group of seven young male sprinters (100 m records: 10.60-10.99 s) using biopsy samples (special needle of 1.5 mm diameter) from the m. vastus lateralis taken at rest and before and after running 40, 60, 80 and 100 m at maximum speed (Fig. 3).

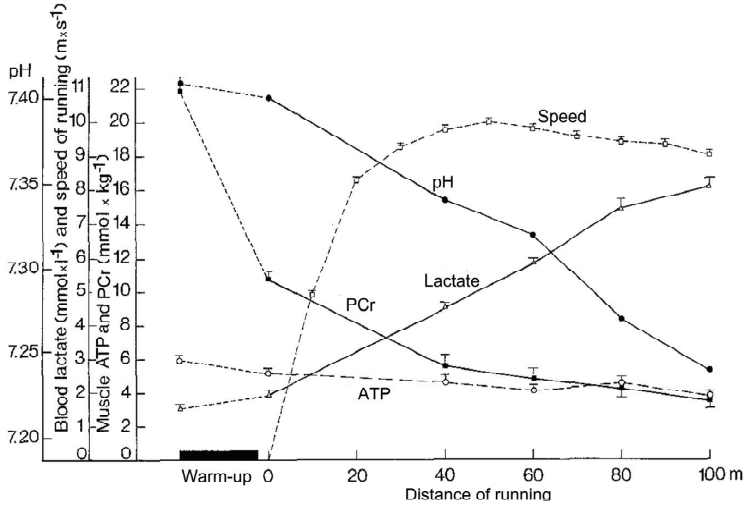

FIGURE 3 Changes in running velocity, blood lactate, capillary blood pH, and muscle ATP and phosphocreatine (PCr) for the different running distances. Adapted from Hirvonen et al. 1987.

The intramuscular PCr stores were depleted by about 55% of the pre-run values already during the first 40 m (~5.5 s) and were further depleted by only another 10% for the rest of the run. The muscle and blood lactate accumulated linearly from the start of the run, suggesting that the contribution of anaerobic glycolysis was constant throughout the 100-m sprint. PCr depletion was thought to be the main factor responsible for the loss of speed (~6-11%) towards the end of the run, as the energy transfer from glycogen may not be fast enough to maintain the high rates of ATP utilization needed for sprinting. The post-exercise muscle (6.7 mmol/kg) and blood lactate (8 mmol/l) and pH (7.25) levels were not considered to be high enough to suggest that acidosis was the primary reason for muscle fatigue. It is important to add that in competition conditions, peak post-race blood lactate levels over 13 mmol/l have been reported (Kindermann and Keul 1977, Hirvonen 1984, Locatelli and Arsac 1995), suggesting that acidosis-induced fatigue could possibly have some influence on 100-m performance.

A later unique experiment by the same authors (Hirvonen et al. 1992) studied changes in muscle metabolites during simulated 400-m track race in six male runners (400-m records: range 47.5-50.5 s). The experiment was carried out in two consecutive days, so that in the first day the athletes ran 400-m and 100-m distances and in the second day 300 m and 200 m. The velocity for 100-300-m distances was based on split times in the initial 400-m sprint (51.9±0.7 s). It was found that compared with pre-run values, the greatest, ~48%, decline in PCr occurred during the first 100-m section. Muscle lactate increased throughout the run (from ~1.8 to 17.3 mmol/kg), but with a significant increase between 100-200 m. This suggests that in the 400-m run, anaerobic glycolysis became the primary energy source after about 14 s (simultaneously with loss of PCr) and that this dominance increased toward the end of the run. The speed of running decreased from 200 m and most markedly during the last 100-m section when there was almost complete depletion of PCr (-89%), a drop in ATP (-27%), and a reduction in the rate of anaerobic glycolysis. These results about the changes in muscle metabolites appear to be in line with a study on treadmill sprinting in sixteen non-athletes (8 males and 8 females) showing that PCr decreased by about 67% and ATP by 28% from resting values after 30-s all-out sprinting (Nevill et al. 1989). Hirvonen and coworkers (1992) suggested that the decrease in glycolytic ATP production (inhibition of enzymes) and loss of running speed at the end of the run may be connected to the high level of muscle acidity. However, they also pointed out that it is unclear whether acidosis is a direct cause of fatigue in the 400 m since a variety of biochemical changes occur simultaneously with the development of fatigue. In fact, the exact mechanism of fatigue during sprint running is still unclear. Studies in young adult sprinters have suggested that fatigue during sprint-type of activity is mainly due to processes in muscle itself (peripheral fatigue) (Mero and Peltola 1989, Nummela et al. 1992, Nummela et al. 1994) but existence of neural fatigue cannot be completely ruled out. With respect to more detailed laboratory studies on muscle fatigue, many experiments have confirmed elevated H^+

concentration as a major factor impairing contractile function, either directly (inhibition of cross-bridge kinetics) or indirectly (e.g., via glycolytic inhibition) (Fitts 2004). However, some studies have linked fatigue to other components of the metabolism, such as increased inorganic phosphate (P_i) from PCr breakdown and potassium ion (K^+) concentrations (Westerblad et al. 2002, Allen and Westerblad 2004).

2.2.2 Peak blood lactate concentration and sprint performance

During maximal sprint performance, anaerobic glycolysis is activated immediately at the onset of exercise (Bergström et al. 1971, Hultman and Sjöholm 1983). Most of the lactic acid ($C_3H_6O_3$) formed in muscle quickly dissociates into lactate ($C_3H_5O_3^-$) and hydrogen ions (H^+) and are transported and diffusing at a high rate out of muscle in the blood (Sahlin et al. 1978, Juel 1988). The rationale behind the use of indirect $[La]_{b\ peak}$ method is that blood lactate levels after maximal performance correlate strongly with muscle lactate at the cessation of exercise and thus gives a rough estimate of anaerobic energy production from glycolysis (Cheetham et al. 1986, Hirvonen et al. 1992). On the other hand, $[La]_{b\ peak}$ underestimate the lactic acid production in active muscles, because some of the lactate is metabolized within working muscles and also due to its rapid uptake by various tissues (kidney, liver, heart, inactive muscles) of the body (Brooks 1987).

It has been estimated that in world-class 100-m performance (10 s) the energy provision is 50% from both anaerobic glycolysis and PCr, whereas in the 400 m, the relative energy contribution is about 62.5% from anaerobic glycolysis, 12.5% from PCr, and 25% from aerobic processes (Newsholme et al. 1992). However, the relative contribution of the three energy systems to the total energy supply has varied a little in different studies due to the different methods to evaluate energy release and training background of the subjects (Nummela and Rusko 1995, Gastin 2001). For example, biopsy studies on metabolic response to cycle ergometer sprinting have suggested higher glycolytic energy production in the beginning of exercise. It has been reported that during all-out cycle sprinting of 6-s, the contribution of glycolytic energy production may be even 50% of the total energy production (Boobis et al. 1982, Gaitanos et al. 1993). The increased anaerobic glycolytic stress with increasing sprint distance is reflected in $[La]_{b\ peak}$. In previous studies in young high-performance male sprinters, $[La]_{b\ peak}$ following 100-, 200- and 400-m sprint races have been found to be about 12-14 mmol/l, 17-20 mmol/l, and 20-25 mmol/l, respectively (Kindermann and Keul 1977, Hirvonen 1984, Lacour et al. 1990, Locatelli 1996).

A number of earlier studies have shown that maximal levels of blood lactate accumulation can predict performance in sprint running, although this finding is not complete. $[La]_{b\ peak}$ has been found to correlate significantly with competitive 200-m (Locatelli 1996) and 400-m track performances (Ohkuwa et al. 1984, Lacour et al. 1990, Nummela et al. 1992, Hill 1999), the maintenance

of maximal velocity during 200-m races (Hautier et al. 1994, Locatelli 1996), as well as with the treadmill sprinting over 100-400 m (Fujitsuka et al. 1982, Cheetham et al. 1986, Weyand et al. 1994) and maximal power during anaerobic running test (Nummela et al. 1996). Hirvonen (1984) investigated $[La]_{b\ peak}$ in Finnish male sprinters (n=66) and hurdlers (n=19) in competitive conditions. The results showed that $[La]_{b\ peak}$ was highly correlated with race times in 100 m (10.6-11.2 s), 200 m (20.9-22.3s) and 400 m (46.9-49.8s) sprinting events, but not in 110-m (13.8-14.5s) or 400-m (51.9-54.4s) hurdle performance. This lack of significant association between $[La]_{b\ peak}$ and hurdle times could be explained by the fact that hurdle performance is also highly dependent on technical factors.

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Studies based on measurements in master swimmers have produced inconsistent results on age-related changes in the blood lactate response to short duration maximal exercise. Reaburn (1990) reported no significant age differences in the capillary $[La]_{b\ peak}$ following a 100-m freestyle swimming test (1 min – 1 min 37 s) when comparing groups of male swimmers aged 25–35, 36–45, 46–55, and 56+ (n=4 in each age group). $[La]_{b\ peak}$ values of 14.3 mmol/l were observed in 25– to 35-year-olds and 13.1 mmol/l in 56-year-olds. Recently, Benelli et al. (2007) examined the capillary $[La]_{b\ peak}$ after competitive swims at distances of 50-400 m (34 s to 8 min 50 s) in male (n=52) and female (n=56) athletes between 40 and 79 years of age. For the analyses, all the distances and swimming styles were combined. The male swimmers showed an age-associated decline in $[La]_{b\ peak}$ between the group in the 5^{th} decade vs. the groups in 7^{th} and 8^{th} decades, but no significant age-group differences existed among the female swimmers. Because the male swimmers also showed an age-related reduction in muscle-bone thigh volume, the authors proposed that the greater reduction in $[La]_{b\ peak}$ with age in males than in females could be caused by their greater loss of skeletal muscle mass (Benelli et al. 2007).

Other investigations of the influence of age on blood lactate response to brief maximal exercise have examined untrained males and report a decline in $[La]_{b\ peak}$ with age (Kindermann and Keul 1977, Makrides et al. 1990, Marsh et al. 1999). Using a 30-s cycle sprint as an exercise test, Makrides et al. (1990) and Marsh et al. (1999) found that $[La]_{b\ peak}$ is lower in older men (ages 60-70 yrs) than young men. In the study on running, Kindermann and Keul (1977) examined 87 young, young adult, and middle-aged subjects. It was found that $[La]_{b\ peak}$ after an all-out 400-m run increased from 9.8 mmol/L in runners aged 8.9 years (running time 78.5 s) to 18.7 mmol/L in 25-year-old group (63.4 s) but thereafter decreased steadily to value of 14.9 mmol/L in the oldest, 56-year-old group (92.5 s). Nevertheless, it is difficult to interpret the $[La]_{b\ peak}$ result found in untrained older people due to the confounding effects of age-related physical inactivity on the determinants of anaerobic metabolism. For instance, at least in females, there is evidence for an age-related decline in total blood plasma, and

red blood cell volume in sedentary, but not in master athletes (Jones et al. 1997), which could lead to bias in the evaluation of post-exercise blood lactate concentration. Moreover, a potential concern is age-related change in lactate diffusion and distribution kinetics following maximal exercise. The available evidence is limited to studies on untrained animals and some of those issues will be covered in discussion part.

2.3 Skeletal muscle characteristics and aging

A primary factor determining athletic ability is the properties of the neuromuscular system that provide the force and power for movement. The following section reviews the literature on age effects on the structural and functional characteristics of skeletal muscle properties in master sprinters. In order to understand the effectiveness of continued sprint training in preventing any decline in muscle properties, it is necessary to discuss the results on changes in muscle characteristics with age in normally active people and in endurance- and strength-trained master athletes.

2.3.1 Muscle structure

Skeletal muscle contains diverse motor units and associated fiber types that display differences in their contractile as well as metabolic properties. Muscle fibers are traditionally classified into slow type I, fast type IIA and fast type IIB by myofibrillar ATPase histochemistry (Brooke and Kaiser 1970). Fiber identification is based on differences in mATPase activity determined by specific myosin heavy chain (MyHC) isoform profiles (Smerdu et al. 1994, Ennion et al. 1995). Fibers classified as type I and IIA have a predominance of MyHC I and MyHC IIa, respectively. However, type IIB fibers in humans contain a predominance of MyHC isoform similar to the rat IIx, not IIb (Smerdu et al. 1994). Intermediate "hybrid" fiber types IIAB, IC and IIC possess two types of MyHC isoforms (Staron et al. 2000).

Normal aging is associated with changes in muscle morphological characteristics. On the basis of the histological data on whole vastus lateralis muscle obtained at autopsy, the reduction in muscle cross-sectional area is approximately 40% over the 60 years from age 20 (Lexell et al. 1988). A primary mechanism for the atrophy is thought to be loss of fibers due to declining numbers of motor units and fiber denervation (Campbell et al. 1973, Lexell and Downham 1992a, Doherty 2003). The reduction in fiber number seems to begin as early as in the third decade of life and the decline becoming evident after age 50 (Lexell et al. 1988). In human m. vastus lateralis, the decrease in fiber number has been reported to be about 50%, from 650 000 to 325 000 fibers between 20 and 80 years of age. The slope of motor unit loss with age seems to be quite similar to that of muscle fiber, although some of the denervated fibers become

reinnervated through axonal sprouting of existing motor units (Faulkner et al. 2008). Evidence from animal and human studies suggests that the loss of motor units with aging may be greatest among the largest and fastest units involving type II fibers (Doherty et al. 1993). The question as to whether fiber type distribution changes with advancing age is, however, unclear as cross-sectional studies have shown both a relative increase in type I fibers (Larsson et al. 1978, Poggi et al. 1987, Jakobsson et al. 1990, Jansson 1994) and no change (Grimby et al. 1984, Essen-Gustavsson and Borges 1986, Lexell et al. 1988) in fiber type composition. Furthermore, the few existing longitudinal studies have not been consistent showing no change (Aniansson et al. 1986, Frontera et al. 2008), decrease in type I fiber percentage (Frontera et al. 2000a) and decrease in type IIb percentage (Aniansson et al. 1992).

Another factor in muscle atrophy is a reduction in fiber size. In several cross-sectional studies, the decrease in fiber size has been more pronounced in type II fibers (Grimby et al. 1982, Essen-Gustavsson and Borges 1986, Lexell et al. 1988, Lexell and Downham 1992b, Hortobagyi et al. 1995, Andersen 2003), leading to a decrease in the type II-to-type I fiber area ratios (Tomonaga 1977, Larsson et al. 1978, Clarkson et al. 1981). Because the area of each fiber type is closely related to MyHC isoform expression, the greater atrophy of fast fibers contributes to the decreasing relative MyHC II content observed in older muscle (Klitgaard et al. 1990a, Hameed et al. 2003, Short et al. 2005). In longitudinal studies, a decline in type IIa and IIb fiber size (Aniansson et al. 1986), no change (Frontera et al. 2000a), increase in type IIa (Frontera et al. 2008) and increase in both type I and II fibers (Aniansson et al. 1992) have been reported. However, the variability between the cross-sectional and longitudinal findings could be attributed to relatively short follow-up periods (4-12 yr) and small sample size in longitudinal investigations.

Aging-related atrophy may be accompanied by changes in the muscle architecture (i.e., spatial arrangement of muscle fibers in relation to the axis of force development), and muscle composition. These changes include reductions in fascicle (fiber) length and pennation angle (Kubo et al. 2003a, Kubo et al. 2003b, Narici et al. 2003, Morse et al. 2005) and an increase in intramuscular fat (Overend et al. 1992, Kent-Braun et al. 2000). Some of these studies show, however, no changes in fascicle length when expressed relative to limb length (Kubo et al. 2003a) or relative to muscle length (Morse et al. 2005).

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It is clear that in many older people changes in muscle structure do not reflect a true aging effect but are largely due to inactivity and disuse of the muscles. Studies on athletes have provided an insight into the effects of long-term training on muscular changes with age. Regarding adaptations to endurance training, several investigations have suggested that years of endurance running with a high volume of low-force contractions is not a sufficient stimulus to maintain whole-muscle or fiber size above the average level (Klitgaard et al. 1990a, Coggan et al. 1993, Proctor et al. 1995, Trappe et al. 1995, Alway et al.

1996, Dinenno et al. 2001). Some investigations in elite athletes have shown that regular endurance training may lead to smaller type I and II fibers (Widrick et al. 1996a) and type II-to-I fiber area ratio (Trappe et al. 1996) compared with untrained subjects. However, this decrease in fiber size was thought to be beneficial for aerobic performance, as it can facilitate oxygen diffusion into the center of the cell. Furthermore, there is some evidence that older endurance athletes do not exhibit a significant decline in fascicle length (Karamanidis and Arampatzis 2006) or increase in intramuscular fat (Sipilä and Suominen 1991, Martin et al. 2000) in trained leg muscles, suggesting that endurance training supports the maintenance of muscle architecture and quality.

Concerning strength training, in a study by Pearson et al. (2002), 54 elite level olympic-style weight lifters aged 40-87 showed a progressive loss of lean lower limb volume (0.8%/yr). These results compare favorably with the muscle fiber observations of Brown and Coggan (1990) for 18 power and olympic lifters aged 18-74 years. In comparison with young lifters (18-23 yr), older (60-74 yr) lifters showed a 30-36% decline in type I and IIa fiber size in m. vastus lateralis. In the older lifters, the size of type I (5500 μm^2) and type IIa (6800 μm^2) fibers remained clearly above the average level. Klitgaard et al. (1990a) and Aagaard et al. (2007) also reported that in older (68-78 yr) men with several years of strength training, type IIa and IIb fibers in m. vastus lateralis were significantly larger than those in age-matched endurance-trained or sedentary men.

Few data exist on the effects of regular sprint training on muscle fiber characteristics in master sprinters. Comparing muscle structure in five 21- to 25-year-old and five 61- to 69-year-old sprinters, Reaburn (1993) found age-related decreases in the size of type IIa and IIb fibers (-20%- -27%) and in the anthropometrically-estimated whole-muscle area (-23%). Within the group of older sprinters, type IIa (5080 μm^2) were 35% larger in size than either type I or IIb fibers, suggesting that sprint training with high-velocity and high–power contractions may overload type IIa fibers more than the others. Further, one study found that a group of elderly sprinters and jumpers had similar quadriceps muscle thickness and cross-sectional area than age-matched controls, but the muscle quality was better preserved in athletes (Sipilä and Suominen 1991).

The larger proportion of slow-twitch fibers in the trained leg muscles of endurance athletes than in the leg muscles of power/sprint athletes is probably largely a result of their genetic aptitude. However, whether years of intensive training can induce changes in the type I to type II percentage ratio is unclear. This possibility is supported by a longitudinal study that found that 6 of the 11 middle-aged male distance runners had a significantly greater proportion of slow fibers in follow-up measurement than in baseline 20 years earlier (Trappe et al. 1995). Another study indicated that in older elite orienteers the arm muscles had an equally high percentage of slow fibers as the leg muscles despite the highly different usage of these limbs over the years, suggesting that with continuous endurance training no shift occurs between the major fiber types (Saltin 1986).

2.3.2 Muscle activation

The central nervous system creates motor commands that activate the musculoskeletal system for movement. A characteristic feature of skilled motor performance is the ability to fully activate agonist muscles in coordination with synergists and antagonist muscles (Sale 1991). However, aging may impair the effectiveness of the neural mechanism. For older untrained people, the most important concern could be central activation and an inability to activate all motor units of the agonist muscles. This has been shown by experiments using the twitch interpolation technique for the quadriceps femoris (Harridge et al. 1999, Stevens et al. 2003) and biceps brachii (Yue et al. 1999) muscles. For example, Stevens and colleagues (2003) estimated an ~11% deficit in the quadriceps femoris muscle activation as between old (64-84 yr, n=46) and younger (18-32 yr, n=46) adults.

In theory, the central activation and neural drive required to activate the agonist muscles could be impaired by the smaller number of motor units recruited, a lower rate of motor unit recruitment and impaired motor unit synchronization. However, not all these mechanisms may be involved: while some studies have suggested that the maximal motor unit discharge rate declines with age (Kamen et al. 1995, Connelly et al. 1999, Klass et al. 2005), other data have indicated no age-related changes in motor unit synchronization (Kamen and Roy 2000) or in the ability to recruit all motor units during a maximum voluntary contraction (Kent-Braun 2009). It has recently been suggested that the reduction in motor unit discharge rate with age might not be a consequence of age-related neural adaptation, but a selective cell death of larger motor neurons that have higher discharge rates than those of surviving motor neurons serving type I muscle units (Lehman and Thomson 2009). At the cellular level, aging can lead to impairments in excitation-contraction coupling due to various changes in sarcoplasmic reticulum characteristics, e.g., loss of dehydropyridine and ryanodine receptors that translate membrane depolarization into intracellular Ca^{2+} release (Payne and Delbono 2004).

In addition to the intramuscular factors, aging could impair intermuscular mechanism. For example, in some studies using surface EMG recordings, older people have showed increased coactivation of antagonist muscles in simple dynamic and isometric actions that could decrease the net strength development of the agonists (Häkkinen et al. 1998a, Izquierdo et al. 1999). One hypothesis is that in untrained older people the increased coactivation of the antagonists serves to compensate for the reduced activation capacity of the agonists and protects against instability of the muscle joint complex (Macaluso et al. 2002, Larsen et al. 2008). On the other hand, the findings concerning highly impaired tapping test speed suggest that muscle contraction/muscle relaxation ratio could also be suboptimal in movements requiring almost no muscle strength (Kent-Braun and Ng 1999).

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Some evidence suggests that during aging the maximal muscle activation ability is maintained above average level by regular practice. In the study by Leong et al. (1999) seven older male weight lifters (67-79 years) and five untrained age-matched controls performed 50% and 100% maximal voluntary contractions (MVC) of knee extensors while recordings from groups of motor units were obtained from the rectus femoris muscle by using an indwelling electrode. It was found that at 100% of MVC, the weight lifters' maximal motor unit discharge rate was approximately 20% higher compared to the untrained men. The only study to examine rapid neural activation in master athletes (Ojanen et al. 2007) involved 32 middle-aged and older (36-85 years) strength-trained master male throwers (shot put, discus, hammer throw) and 28 controls (35-74 years). In order to allow reliable comparison between individuals, the surface EMG was normalized so that the data obtained during the initial 0-100 ms and 0-500 ms of isometric bilateral leg extension was related to maximum activation during peak force phase (500-1500 ms) in the same action. The relative agonist iEMG activation during the first 100 ms compared to maximum was lower in 50- to 75-year-old groups, but not in 40-year-old athletes. Moreover, only the youngest, 40-year-old group could already produce maximal activation during the first 500 ms. However, no clear differences could be seen in the rate of relative muscle activation as between master throwers and untrained people.

2.3.3 Single-fiber contractile function

In recent years, aging-related changes in contractile properties of single (skinned/permeabilized) muscle fibers have received increasing scientific interest since these factors could explain the deterioration in whole-muscle function. The single fiber contractility has been examined by both intact and permeabilized (skinned) muscle fiber preparations. The force generation of intact fibers, which have the membrane with a functional excitation-contraction coupling system, provide insight into the dihydropyrine and ryanodine receptors that are critical for efficient excitation-contraction coupling (Payne and Delbono 2004). In contrast, the permeabilized muscle fibers without excitation-contraction coupling system, provide more direct information on the interactions of the myosin and actin.

The maximum velocity of unloaded shortening (V_o) has been described as an important design parameter of muscle (Rome et al. 1990), especially since the mechanical power produced by muscle is a product of force and shortening velocity. Fiber V_o is thought to reflect the maximum rate of cross-bridge cycling and correlates with myosin ATPase activity (Barany 1967). Studies on young adult subjects have shown that the MyHC isoform expressed in a fiber has strong influence on V_o in the order I < IIa < IIx. In humans, V_o of type IIa and IIx fibers in m. vastus lateralis has been reported to be about 3.3-4.7 and 8-10 times faster, respectively, than that of type I (Larsson and Moss 1993, Bottinelli

et al. 1996, Harridge et al. 1996, Harridge et al. 1998). For hybrid fibers that contain two MyHC isoforms, V_o is an intermediary, with the slower myosin type possibly having a greater effect on V_o than the faster type (Larsson and Moss 1993, Larsson et al. 1996). Further, it has been found that V_o can vary considerably in the fibers of the same MyHC isoform profile. This variability may relate, in part, to the contribution of the composition of the myosin light chain isoform to fiber V_o (Larsson and Moss 1993). It is noteworthy that experiments using in vitro motility assay have suggested that the difference in contractile speed between different fast fiber subtypes (IIa vs. IIx) could be significantly smaller at the human physiological temperature (35 °C) than at the normally reported lower (12 °C or 15 °C) temperatures (Lionikas et al. 2006).

Specific tension (ST, maximal force divided by cross-sectional area) is another principal single fiber contractile parameter. ST is dependent on the number of cross-bridges acting in parallel and the tension generated by each cross-bridge. As to the effect of fiber type on ST, the results of the many (Bottinelli et al. 1996, Harridge et al. 1996, Larsson et al. 1997, Harridge et al. 1998, Frontera et al. 2000b) but not all (Larsson and Moss 1993) studies in young adult subjects suggest that ST is higher (~14-50%) in type II than in type I MyHC fibers. Further, power curves constructed from the force-velocity relationship indicate that the peak power of type IIa and IIx fibers is ~5- and 10-fold greater, respectively, than that of type I MyHC fibers (Widrick et al. 2002).

To date, no clear consensus has emerged about the effect of aging on contractile function of single, permeabilized fibers in normally active men. With respect to V_o, studies have shown aging-associated slowing of both type I and IIa fibers (Larsson et al. 1997, D'Antona et al. 2003, Ochala et al. 2007, Yu et al. 2007), slowing of type IIa fibers (Krivickas et al. 2001, D'Antona et al. 2007) as well as no change in V_o (Trappe et al. 2003). Similarly, there are conflicting results about the effect of age on ST, with investigators reporting declines in type I (D'Antona et al. 2007), in type IIa (Hvid et al. 2009), in both type I and IIa fibers (Larsson et al. 1997, Frontera et al. 2000b, D'Antona et al. 2003, Ochala et al. 2007) and no age differences in ST in either fiber type (Trappe et al. 2003). In a recent longitudinal study, no changes were found in V_o or ST over 9 years in five elderly (at the age of 71-80 years) untrained men (Frontera et al. 2008). In support of the contractile dysfunction with age hypothesis, studies using in vitro motily assay have found alterations in the properties of the myosin molecule that might partially explain the slowing of contractile speed with age (Höök et al. 2001, D'Antona et al. 2003). Further, based evidence from human and rat studies, the age-related loss of ST could relate to reductions in the myosin concentration (D'Antona et al. 2003) and a decrease in the fraction of myosin heads in the strong-binding structural state (Lowe et al. 2001).

MASTER ATHLETES

At least four studies have been published on the effects of years of physical training on single fiber contractile parameters. D'Antona et al. (2007) compared V_o and ST in type I and IIa fibers in m. vastus lateralis among young physically

active controls (30 yrs, n=5), and older (73 yrs) untrained (n=7) and regularly endurance-trained (n=3) men. The results showed no significant differences in ST in either fiber type between the young controls and older endurance-exercised men, whereas elderly untrained men showed a decrease in ST in type I fibers. With respect to V_o, both the older endurance-trained and untrained men showed an age-related reduction in V_o for type IIa but not for type I fibers. These findings differ from those of Larsson et al. (1997) who reported a decrease in V_o and ST for type I and IIa fibers in m. vastus lateralis in both older (73-81 yr) untrained (n=2) and physically active (n=2) men when compared with young untrained men (25-31 yr, n=4). The physically active older subjects had, however, different training backgrounds one being strength-trained and other endurance-trained. Widrick et al. (1996a, 1996b) examined single fibers from the gastrocnemius lateralis muscle of middle-aged elite distance runners (42 yr, n=6) and their age-matched untrained counterparts (44 yr, n=5). They found that in type I MyHC fibers, V_o was significantly faster (19%) in the distance runners than in a control group, whereas no differences were found in type IIa and IIx fibers. In addition, there were no group differences in the ST of the fibers, but the absolute force and peak power of type I and IIa fibers were lower in runners than in untrained men due to reduced fiber size (Widrick et al. 1996a, Widrick et al. 1996b). A methodological criticism which can be raised with all of the above mentioned studies is small number of subjects per age or athlete groups (n=2-6). Within that limitation, the data tend to suggest that years of endurance training may lead to adaptations (maintained/higher V_o of type I fibers, and reduced fiber size) that could optimize the fiber's ability to work for prolonged periods, while having a detrimental effect on the fiber's ability to perform fast and high-intensity exercise. However, it is likely that muscle fiber adaptations are dependent on the nature of the exercise stimulus. The effectiveness of long-term sprint and strength training in the prevention of age-related changes in single fiber function remains unclear.

2.3.4 Whole-muscle strength

MAXIMAL STRENGTH. Since the 1800s (Quetelet 1835), an extensive body of literature has been accumulated demonstrating that aging is associated with inevitable loss of maximal force-generating capacity. In recent cross-sectional studies comprising large numbers of subjects across the adult age span (20-80 yrs), maximal isometric and concentric strength of lower extremity muscles has been found to decline from about 40 years of age (Hortobagyi et al. 1995, Lindle et al. 1997, Samson et al. 2000, Akima et al. 2001, Pearson et al. 2002, Lauretani et al. 2003). The average decline in leg strength has been estimated to be about 0.5-1.0% per year with an accelerated decline toward the higher age groups. Further, follow-up studies (3-12 yr) have suggested that, at least in older ages, longitudinal declines in strength could be somewhat higher (Aniansson et al. 1986, Winegard et al. 1996, Frontera et al. 2000a, Hughes et al. 2001, Goodpaster et al. 2006). One exception to the pattern of aging-associated strength reduction may be eccentric strength, which has found to be little

affected by age in some studies (Poulin et al. 1992, Hortobagyi et al. 1995, Porter et al. 1997)

Of the various age-related neuromuscular changes, loss in muscle mass appears to be the major cause of the reduction in maximal strength with age (Frontera et al. 1991, Häkkinen and Häkkinen 1991, Reed et al. 1991, Izquierdo et al. 1999, Frontera et al. 2000a, Akima et al. 2001). Another possible explanation is a reduction in force per unit area (specific force). The differences in whole-muscle specific force between young and older adults have been about 10-30% (Young et al. 1985, Bruce et al. 1989, Klitgaard et al. 1990a, Jubrias et al. 1997, Macaluso et al. 2002) and could reflect, among other factors, reduced muscle-fiber specific tension (Larsson et al. 1997, Frontera et al. 2000b), an increase in coactivation of the antagonist muscles (Izquierdo et al. 1999, Macaluso et al. 2002) and a decrease in agonist muscle activation (Harridge et al. 1999, Stackhouse et al. 2001, Kamen and Knight 2004, Morse et al. 2004, Kubo et al. 2007).

EXPLOSIVE STRENGTH**.** Explosive strength is often used as a general term for expressing rapid force production capacity in different simple and complex muscle actions and is also used in this thesis. In isometric force task, explosive strength can be assessed as rate of force development (RFD), calculated from the slope of force-time curve. In dynamic action explosive strength can be defined by examining the ability produce greatest possible power output (speed) in single acyclical and cyclical activities against light-to-heavy external loads (e.g., jumping, weight-lifting).

The assessment of explosive strength has received increasing interest among gerontologists as it has been recognized that for mobility, being able to produce force quickly, could be more significant than maximal force (Bassey et al. 1992, Bean et al. 2003). For example, with respect to recovery from trip, the time available to make appropriate initial responses is about 0.3 s, and thus impose high demands on fast force production of hip extensors of swing leg and plantar flexors of stance leg (Schultz 1995). Of specific concern is, however, that with age explosive strength declines more radically than maximal strength. Cross-sectional studies have shown that the average rate of decline of 0.9-1.5% per year in dynamic and isometric explosive strength begins at around 30-40 year of age (Bosco and Komi 1980, Bemben et al. 1991, Jubrias et al. 1997, Pearson et al. 2002, Lauretani et al. 2003). There are also few studies that have provided insight into the speed of rapid repetitive movements (Radford and Upton 1976, Kent-Braun and Ng 1999). In their study, Radford and Upton (1976) examined 139 males and females ranging in age from 5 to 75 years and found that the speed of alternated hand and foot tapping in 20-s test increased until ~20 years of age, but decreased then with clear decline about the age of 55 years. On average, tapping speed declined 30-35% from peak level from 20 to 75 years of age, an average rate of about 0.6% per year.

The decrease in muscle size and maximal strength with age plays a critical role in the ability to generate explosive force, especially in actions where muscle

contraction must overcome relatively high external resistance (Grassi et al. 1991, Izquierdo et al. 1999, Lanza et al. 2003, Thom et al. 2005). Some studies suggest that even half of the loss of vertical jump ability and triceps surae power is accounted for by loss of muscle size (Grassi et al. 1991, Thom et al. 2005). Another critical factor seems to be MyHC isoform composition, as expected from force and power properties of different muscles (Fitts and Widrick 1996). For example, type II MyHC has been related to the ability of older muscles to produce rapidly plantar flexion torques at faster isokinetic velocities (Harridge et al. 1995), and vertical jump (Sipilä et al. 2004). Among numerous other proposed factors are a decrease in the rapid neural activation (Häkkinen et al. 1998a, Kent-Braun and Ng 1999, Liu et al. 2006) possibly affected by decreased nerve conduction velocity (Wang et al. 1999), decline in fascicle/fiber length (Thom et al. 2007), increase in tendon compliance that can slow force transmission (Reeves et al. 2003), impaired ability to use the elasticity of muscle-tendon complex in vertical jumping (Bosco and Komi 1980, Hoffren et al. 2007), and a reduction in the rate of Ca^{2+} release-reuptake by the sarcoplasmic reticulum affecting isometric contractile speed (Klitgaard et al. 1989a, Delbono et al. 1995). The mechanism of decline in fast repetitive movements could also relate to motor processing speed of the central nervous system (Kent-Braun and Ng 1999, Sosnoff et al. 2004).

MASTER ATHLETES

Investigations on endurance athletes have indicated that while prolonged training does not attenuate the age-associated loss of absolute maximal leg strength (Alway et al. 1996) it could limit the extent of loss of relative strength, because whole-muscle force scaled to unit area (specific force) is greater in older endurance athletes than in untrained controls (Klitgaard et al. 1990a, Alway et al. 1996, Trappe et al. 1996, Dreyer et al. 2006, Aagaard et al. 2007). However, the experiment by Harridge et al. (1997) in older orienteers (70-100 yr, n=15) and untrained (68-92 yr, n=18) men suggest that the endurance training stimulus may aggravate the age-associated slowing of the twitch contractile properties of the knee extensors. Recent studies have also indicated that, elite master long-distance runners (40-87 yr, n=116) had about 8-14% lower counter-movement jump power in comparison with age-matched untrained men (n=89), the differences becoming smaller with increasing age (Runge et al. 2004, Michaelis et al. 2008). These findings that endurance running might cause restraining of explosive muscular contraction was also observed in an experimental trial on middle-aged and older untrained men (30-71 yr, n=36) showing an inhibitory effect of daily running exercise (~4 km/day, for 18-week) on the vertical jump height (Ono et al. 1976). On the basis of some previous studies, the slowing of contractile properties and loss of power in older endurance athletes could be linked to the lower expression of fast fibers (Harridge et al. 1997, Martin et al. 2000) and smaller type I and II fiber diameters (Widrick et al. 1996b).

Reports on strength-trained athletes have indicated that high-intensity resistance training is associated with good muscle function but that it cannot totally prevent the age-associated loss of strength (Brown and Coggan 1990, Meltzer 1994, Pearson et al. 2002, Anton et al. 2004). For instance, one study found that in 60- to 74-year-old weight and power lifters dynamic knee extension torque and squat lift (110 kg) were 75% and 66%, respectively, of the values in young strength athletes aged 18-23 years (Brown and Coggan 1990). Anton et al. (2004) performed a retrospective analysis of top age-group weightlifting and powerlifting records (USA) and found that the age-related decline in squat and bench press (powerlifting) performance was smaller than that in olympic-style weightlifting characterized by more complex and powerful effort of the entire body. In a study by Pearson et al. (2002) comparison of 40- to 87-year-old male weight lifters (n=54) with age-matched untrained controls (n=54) indicated that the rate of decline in leg extension power (1.1-1.5%/yr) and isometric knee extension strength (0.5-0.6%/yr) with age are similar for highly-trained and untrained men. In the athletes, however, power and strength values were about 32-35% higher. This has been confirmed by other studies (Klitgaard et al. 1990a, Leong et al. 1999, Aagaard et al. 2007, Ojanen et al. 2007). The age-related decline in muscle force production in strength-trained master athletes could be attributed both to muscle wasting mediated by reduction of fiber size (Brown and Coggan 1990), and decreases in rapid neural activation (Ojanen et al. 2007).

Cross-sectional data have shown that older athletes involved in systematic sprint– and jump–type training may exhibit positive adaptations in both maximal and explosive strength/power. A case study comparing a world champion master male long jumper aged 71 and seven sedentary controls (63-81 yrs) was one of the first to show that large positive effect in the rapid and maximal isometric strength of knee extensors (about 60% higher values in jumper) could be achieved by regular training that emphasizes plyometrics (Iwaoka et al. 1989). Recent findings on male track athletes aged 35-90 (n=295) indicated that in sprinters countermovement jump power (W/kg) was greater (+15-29%) than that in middle-distance runners, who in turn had higher power (+17-40%) than endurance runners (Michaelis et al. 2008). However, the power demonstrated quite similar rate of decline with age (~1.0%/yr) in all athlete groups. In a study by Sipilä et al. (1991), elderly (71-80 yr, total n=97) sprint/jump athletes performed better in the vertical counter-movement jump than either sedentary or strength- or endurance-trained men of the same age. The maximal isometric knee extension strength of the sprinters/jumpers was comparable to that of the strength-trained athletes, but higher than that of the endurance athletes or untrained individuals. Reaburn (1993) found that while older sprinters had 22-29% lower isokinetic strength of knee extensors than young runners, they were able to produce higher torque compared with sedentary controls at slow (60°/s) angular velocities and compared with both the sedentary and endurance-trained older men at higher angular velocities (180 and 240°/s). In the same study, older sprinters showed 31-37% higher peak

power and 10-s work capacity in cycle ergometer sprints than their sedentary or endurance-trained counterparts, but had 29-35% lower values when compared to younger sprinters. The author suggested that the major mechanism for the decline in strength and anaerobic power is a decrease in active muscle mass secondary to an age-related reduction in type II fiber area (Reaburn 1993). On the basis of the above literature, possible age-related neuromuscular changes affecting maximal and explosive strength are summarized in Figure 4.

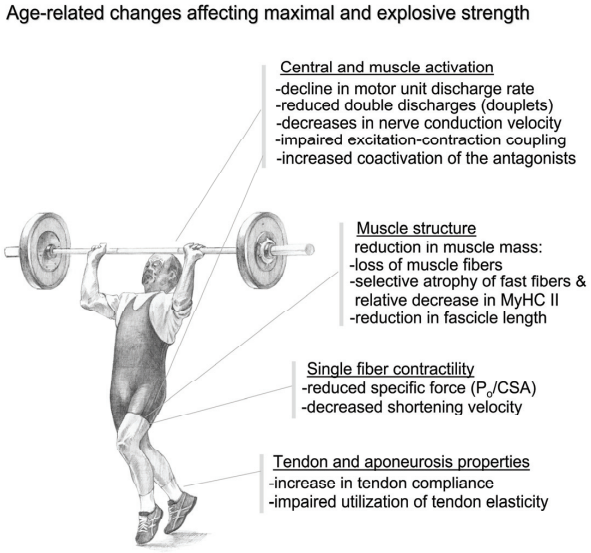

FIGURE 4 Potential age-related changes in neural and muscular factors affecting maximal and explosive strength.

2.4 Effect of training on sprint performance and skeletal muscle characteristics

Analysis of the training methods of young sprinters indicates that the optimal development of sprinting ability requires not only specific sprint training but also well-designed strength training throughout the year (Mero et al. 1987, Joch 1992, Andersen et al. 1994, Delecluse 1997, Young et al. 2001, Blazevich and Jenkins 2002). In general, the aim of strength training for sprinters is, to produce very fast maximal actions against high (80-100%) and lower loads (30-60%), to address force and velocity components of the power equation and to improve motor unit recruitment and the selective hypertrophy of the fast fibers so as to increase the proportion of type II myosin. Additional muscle mass that does not increase sprint-specific strength and power is likely to be performance-limiting for weight-bearing sprinting and should be avoided in training.

However, as described earlier, muscle involvement and force requirements are quite complex in sprinting as they consist of different phases (Mero et al. 1992, Wiemann and Tidow 1995, Young et al. 2001). While the hip extensors and reactive strength of the calf muscles could be the main factors for maximum speed when contact duration lasts 80-100 ms, the maximal strength and rate of force development of the hip and knee extensors also play important role, especially in the start and acceleration phases (Mero et al. 1992, Wiemann and Tidow 1995, Young et al. 2001, Mero et al. 2006). Accordingly, it is clear that strength training for speed development must involve various muscle groups and strength qualities.

In order to achieve successful transfer of training, the movements used in strength training should be sport-specific in their biomechanical characteristics. For example, the movements used in competitive weightlifting (the snatch, clean, and jerk) and especially their variations may simulate mechanically (power, velocity) sprinting movements more effectively than strength training machines. Thus, such explosive lifting exercises may be especially valuable for enhancing sport-specific strength (Kraemer and Häkkinen 2002, Stone et al. 2002). The use of various fast contact bounding/plyometric exercises is a way to develop force production and elastic abilities with similar force-time characteristics to those required in sprinting (Mero and Komi 1994). In addition, plyometrics may help to convert the increased strength into sprinting movement by enhancing the intermuscular coordination of separately weight-trained muscle groups (agonists, antagonists, synergists).

In modern training methods it has also become evident that to avoid overtraining and to maximize the transfer of strength gains to sprint performance there is need for the appropriate sequencing of volume, intensity and exercise selection in a periodized manner during various phases (preparation, competition) of the training period (Joch 1992, Delecluse 1997, Young et al. 2001, Kraemer and Häkkinen 2002). Finally, one notable finding regarding periodization for peak performance is that heavy training may down-regulate the most powerful MyHC IIx isoforms whereas the opposite can result from detraining (Andersen and Aagaard 2000, Andersen 2001).

TRAINING INTERVENTIONS IN YOUNG SPRINT ATHLETES. Very few intervention studies exist on the effects of the interaction of strength and sprint training on both sprint performance and neuromuscular characteristics in already trained sprint athletes. Cadefeau et al. (1990) found that when sprint training (30-500 m) was combined with strength training (weight, plyometrics), 60-m and 300-m sprint times decreased 3.3% and 4.1%, respectively, over 8 months training in young runners (16-17 yr, n=16) with no prior strength training experience (Cadefau et al. 1990). On the basis of their muscle biopsy results, improved sprint performance with the combined training may have been partially related to the enlargement (20%) of type II and type I fibers and increases in the activity of the key glycogenolytic and glycolytic enzymes, while there was 8% significant increase in slow type I fiber percentage.

A study by Andersen et al. (1994) showed that three months of normal preparatory strength- and interval-training (15-18h/wk) improved 20-m block- and flying-start sprint times by about 2-3% and average power output during a 45-s knee extension test by about 20% in a group of young elite male sprinters (20-27 yr, n=6). These changes were accompanied by the transformation of both MyHC isoform I and IIx towards MyHC isoform IIa (I→IIa←IIx), suggesting that one possible explanation for improved performance could be related to changes in MyHC composition. Interestingly, a subsequent study by Andersen (2001) showed a great inter-individual variability in MyHC IIx response to strength training (12 wk) followed by interval running (8 wk) in six young previously untrained men. Those who had high proportions of MyHC IIx (~26%) in their m. vastus lateralis, exhibited complete loss of the MyHC IIx with strength training, but it returned to the initial level (to ~18%) after running training. In contrast, in the subjects with low level MyHC IIx (~4%), the isoform content remained largely unchanged during the whole training. Although small sample size disallow firm conclusions, it seems possible that strength training is more effective than interval running for decreasing MyHC IIx and that individuals might experience different MyHC IIx responses.

MASTER SPRINTERS

In the past, before 1970s, the training for sprinters consisted mainly of running practices. This tradition appears to be reflected to current training of many master sprinters who have continued their career into older age (Sipilä et al. 1991). In their study, Reaburn and coworkers (1995, 1997) hypothesized that an increase in resistance training stimulus could lead to further improvements in strength and sprint performance in competitive master runners who had limited or no previous experience of strength training. It was found that in response to an 8-week combined sprint (2x/wk) and hypertrophic strength (3x/wk) training program, the male sprinters (55±6 yr; n=6 and 4 controls) increased dynamic leg extensor strength by 25%, quadriceps strength by 10%, hamstring strength by 12%, anthropometrically-measured thigh circumference by 3%, and 100-m and 300-m sprint performance by 4% and 2%, respectively. Based on these findings, the authors recommended that master athletes involved in sprint/power events should include hypertrophy strength training as an essential component of the training program. The results also indicate that the master male sprinters, at least between ~45 and 60 years of age, were able to tolerate the increased training volume (5 sessions per week) without signs of overtraining, as indicated by their maintained basal serum testosterone levels (Reaburn et al. 1997). While this study indicates that combined training is more effective than sprint training alone, it remains to be determined how the training is reflected to muscle fiber and neural characteristics. This information would increase the knowledge of the plasticity of already trained neuromuscular system to altered exercise stimulus and could have implications of planning of training programs for aging athletes.

3 AIMS OF THE STUDY

Studying peak performances and related physiological characteristics in aging master athletes can provide valuable information about potential benefits of different types of training and increase our understanding what is possible in physical aging with regular exercise. In athletes the physical changes reflect the aging process itself, with minimal confounding influences of decreased physical activity and diseases. Most of the previous studies have dealt with endurance and strength capabilities. However, to date very little is known about the speed performance characteristics and this led to the interest in the present thesis. The general aims of the study were a) to investigate the associations between age, sprint performance characteristics, anaerobic energy production and functional and structural properties of skeletal muscle in systematically trained male sprinters, and b) to study the effects of combined sprint and strength training on the running performance and neuromuscular in older runners in an experimental trial (Fig. 5).

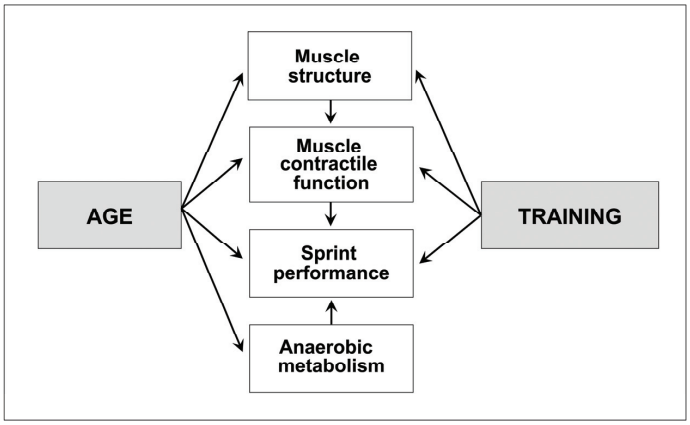

FIGURE 5 The general aims of the study.

More specifically the aim was to answer the following research questions:

(1) How do the overall 100-m and 60-m sprint performances decline with age? What are the biomechanical changes associated with maximum velocity decline with age (I, II), and does age have an influence on the repeatability and symmetry of the performance variables? (III)

(2) Is anaerobic lactacid energy production, estimated by blood lactate response, affected by age, and is the peak blood lactate concentration associated with overall 100-400-m sprint performances? (IV)

(3) What is the effect of age on various muscle structural and functional properties at the single-fiber and whole-muscle levels in systematically trained sprint athletes? (II, V)

(4) What is the contribution of structural and functional characteristics of leg muscles to age-related decline in maximum-speed sprinting? (II)

(5) Are older sprinters able to further enhance sprint performance and neuromuscular characteristics by incorporating heavy-resistance and high-power strength exercises into the overall training program? (VI)

4 MATERIALS AND METHODS

4.1 Study subjects and design

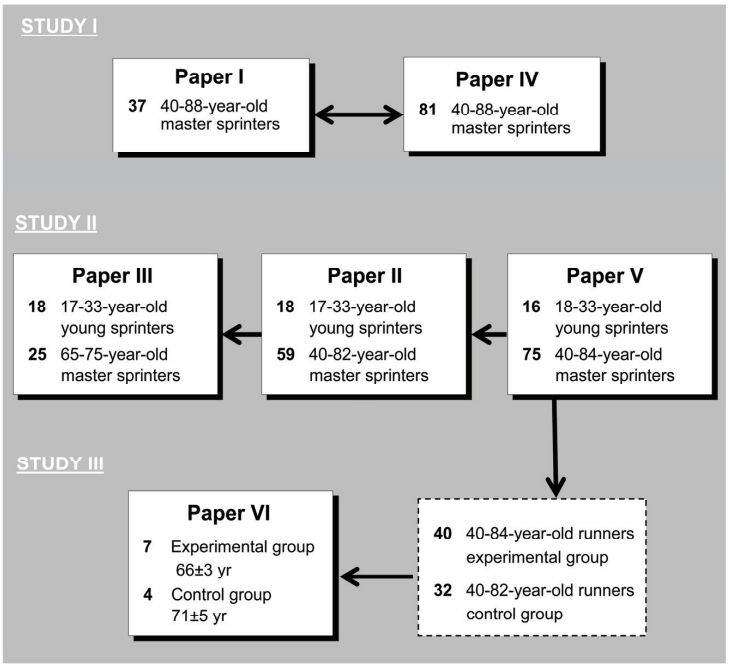

FIGURE 6 Scheme of the subjects in the studies.

The present thesis consists of three larger study phases shown in Figure 6.

STUDY I. The sprinters aged 40-88 years qualified for the finals and semifinals in the 100-400-m sprint events in the European Veterans Athletics Championships (in July 2000) were recruited for the first phase of the study (I, IV). Thirty-seven

sprinters (the fastest 3-4 finalists in each 5-yr age category) were analyzed for biomechanical aspects of the 100-m sprint performance (I). Examination of blood lactate response to sprinting comprised 81 runners who had qualified for finals in 100-, 200- and 400-m sprint events (IV). Information on current and former training, competition performance and sport injuries was obtained with questionnaires (translated into 8 languages) and interviews. Most of the athletes had in their youth competed in sprint running events and maintained regular year-round training. With age, there were no significant differences in total training hours per week (~6-8 h) and the percentage of sprint training (~70-90%) of total training.

STUDY II. Laboratory-based measurements of running biomechanics and skeletal muscle properties were undertaken in the second study phase in 2002 (II, III, V). A total of 108 athletes aged 17-84 years were recruited from among the members of Finnish track and field organizations. To qualify for the study the subjects had to have a long-term sprint-training background and success in international or national championships in 100- to 400-m sprinting events. On the basis of the questionnaire, the weekly training hours (from about 11.5 to 5.9 h/wk), training frequency, and strength training hours declined with age, with the greatest decline from the youngest to the 40- to 49-year-old athletes (V). Medical histories and a focused medical examination (for subjects >55 yr) indicated that the actual health of the subjects was good without any functionally limiting chronic neurological, cardiovascular, endocrinological or musculoskeletal conditions. Basal serum concentrations of total testosterone (T), sex hormone-binding globulin (SHBG) and T/SHBG -ratio (free androgen index) were examined in subjects aged 40-84 (Table 1). T concentration remained unchanged with age, while SHBG values increased and T/SHBG ratio declined. However, both T and SHBG were within the normal references values. The daily dietary intake of macronutrients and micronutrients was registered by food diaries for 3 workdays and 2 weekend days. Subjects were given detailed instructions on completing the foods records (portion sizes, exact brand names, preparation techniques). The diaries were analyzed using the nutrient analyses software Nutrica 3.0 (Social Insurance Institution, Turku, Finland). The results showed no age-related differences in energy and macronutrient intake. The daily dietary protein intake followed the recommendations for older athletes (1.2-1.4 g/kg vs. recommendation 1.2-1.4 g/kg), whereas daily carbohydrate intake was below the recommendations (3.6-4.3 g/kg vs. recommendation 6 g/kg/day) (Campbell and Geik 2004). In micronutrients, only vitamin D intake (8.6±6.1 µg) was below recommendations (10-15 µg) and its intake decreased with age ($r=-0.35$, $p<0.01$). The selected characteristics of the study subjects are listed in Table 1 (V).

TABLE 1 Selected characteristics of the subjects in the second study phase (V). Values are means ± SE.

Variable	Age					r with age
	18-33 yr	40-49 yr	50-59 yr	60-69 yr	70-84 yr	
N	16	16	18	21	20	
Age, yr	24.3±1.0	44.0±0.9	53.9±0.6	65.8±0.6	75.3±0.9	
Height, cm	178.0±1.1	180.5±1.9	175.5±1.1	172.7±0.9	171.1±1.2	**-0.48*****
Body mass, kg	77.2±1.4	79.7±1.9	74.3±1.4	71.2±0.9	69.8±2.0	**-0.39*****
Body fat, %	16.5±0.9	13.3±1.0	14.9±0.9	13.6±1.0	15.7±1.1	**0.23*****
Serum hormones, N		15	18	21	18	
T, nmol/L		18.0±1.8	14.8±0.8	15.7±0.8	16.2±1.0	-0.10
SHBG, nmol/L		40.5±3.54	41.2±5.1	39.3±2.8	57.4±4.3	**0.33****
T/SHBG ratio		0.46±0.04	0.39±0.02	0.44±0.04	0.30±0.02	**-0.31****
Dietary intake, N		10	13	19	12	
Energy, kcal		2 430±120	2 230±110	2 370±70	2 070±60	-0.24
Energy, kcal/kg		31.0±1.4	30.8±1.7	33.1±1.3	28.4±1.0	-0.07
Carbohydrates, E%		50.0±1.0	50.4±1.7	51.7±1.9	51.0±1.6	0.11
Carbohydrates, g/kg		3.9±0.2	3.9±0.2	4.3±0.2	3.6±0.2	0.03
Protein, E%		15.9±0.9	17.6±0.4	17.1±0.6	16.1±0.6	-0.24
Protein, g/kg		1.2±0.1	1.4±0.1	1.4±0.1	1.2±0.1	-0.05
Fat, E%		30.9±1.5	29.3±1.3	28.6±1.2	31.6±1.6	0.00
Fat, g/kg		1.06±0.06	1.00±0.08	1.06±0.07	0.99±0.18	-0.07

STUDY III. The subjects for the third study phase were recruited from among the 40- to 84-year-old men in the second phase. The men in the experimental (EX) group (n=40) participated in a periodized strength and sprint training program for 20 weeks (December 2002 - May 2003), while the controls (CTRL, n=32) continued their accustomed run-based training routines. A subset of twelve 52- to 78-year-old elite sprinters with no background of intensive strength training was chosen for the study VI, which involved single-fiber analyses of biopsy samples. There were 7 subjects in EX group and 5 subjects in CTRL group. One subject from CTRL group could not complete the study. The baseline performance and training characteristics did not differ between groups suggesting that the randomization was successful. The measurement and analyses performed in the second phase served as a baseline and they were repeated after 20 weeks. The study included also field tests for running speed and muscle force-production properties. The overall experimental design and the timetable of various measurements performed during the whole training intervention are summarized in Table 2.

In each study, the subjects were informed about the possible risks and discomfort associated with the investigation before giving written consent to participate in the measurements (I-VI). This study was approved by the Ethics Committee of the University of Jyväskylä and conformed to the Declaration of Helsinki.

TABLE 2 Experimental design of training intervention (VI).

	Training (January – May)						Follow-up (June, August)	
Measurements **weeks**	1	5	10	11	16	21	26	35
Maximum velocity and stride variables	●	●		●	●	●		
Ground reaction force characteristics	●			●		●		
Vertical and leg stiffness	●			●		●		
Competition analysis (100-m, speed curve)			●				●	●
Maximal dynamic strength	●			●		●		●
Jumping performances	●	●		●	●	●		●
Maximal and explosive isometric strength	●			●		●		
Electromyographic measurements	●			●		●		
Muscle biopsy	●					●		
Ultrasound measurements	●			●		●		
Body fat %	●			●		●		
Body weight	●	●		●	●	●		●
Serum hormones	●			●		●		
Nutritional intake		●						

4.2 Measurements

The measurements and variables used in the studies are listed in Table 3 and only briefly described in this chapter. For detailed information the reader is referred to the original reports.

4.2.1 Sprint running performance

COMPETITION PERFORMANCE ANALYSIS. Velocity and kinematic stride parameters during 100-m sprint finals in the European Veterans Athletics Championships were analyzed from video data (I). For data collection two high-speed cameras with a panning technique were placed parallel to track behind back straight at the points of 32 m and 72 m from starting line. Progressive velocity was determined from the time between the consecutive 10-m sequences. Average step length and step rate values were calculated for each 10-m sequence, and contact and flight times were examined for peak velocity and deceleration phases. For the time analysis of the 200-m and 400-m races, the race distance was divided to four 50 m and 100 m sections, respectively. For 200 m, the two camera locations were the same than during 100-m race, while during 400-m event the two panning cameras were positioned at opposite sides of the track area.

TABLE 3 The variables measured in the original studies and methods used.

Variables	Study	Methods/references
Sprint running performance		
Speed-curve	I, II	Chow (1987), Kersting (1999)
Maximum speed	I, II,VI	"
Kinematic stride cycle parameters	I–III,VI	Mero (1987), Weyand et al. (2000)
Ground reaction forces	II,III,VI	Mero (1987)
Vertical and leg stiffness (k_{vert}, k_{leg})		Morin et al. (2005)
Blood lactate concentration	IV	Lactate Pro, Pyne et al. (2000)
Muscle fiber properties		Biopsy m. vastus lateralis, Bergström (1962)
Fiber-type distribution	II,V,VI	ATPase histochemistry, Staron et al. (2000)
Fiber cross-sectional area	II,V,VI	Tema image-analysis, Sipilä et al. (2004)
MyHC isoforms in muscle homogenates and single fibers	V,VI	SDS-PAGE, Andersen and Aagaard (2000), Larsson et al. (1993)
Specific tension (Po/CSA)	V,VI	Moss (1979)
Unloaded shortening velocity (Vo)	V,VI	Edman (1979)
Whole-muscle structure		
Muscle thickness	II,V	Ultrasound, Aloka SSD-1400, Kubo et al (2003)
Pennation angle of fascicles	II,V	" "
Fascicle length	II,V	" "
Dynamic strength		
Bilateral concentric half-squat 1-RM	II,VI	Smith machine, Häkkinen et al. (2002)
Jumping performances		Contact mat, force plate
Squat jump	VI	Viitasalo (1985)
Counter movement jump	II,V,VI	"
Reactive jumping (5 s)	VI	Bret et al. (2002)
Standing triple-jump	VI	Mero et al. (1981)
Isometric strength		Isometric dynamometry
Bilateral leg extension force and RFD	II,V	Viitasalo et al. (1978, 1980)
Unilateral knee extension torque	VI	Häkkinen et al. (1998)
Unilateral knee flexion torque	VI	"
Electromyography		Vastus lateralis and medialis, biceps femoris
Maximal iEMG during strength tests	VI	Häkkinen et al. (1988)
Basal serum hormone concentrations		
Total testosterone, T		Chemiluminescent immunoassays,
Sex-hormone binding globulin, SHBG		(Immulite 1000 analyzer)
Anthropometry		
Height and body mass	I–VI	
Body fat, %	II,V,VI	BIA, Spectrum II, RJL System
Questionnaires		
Training history	I–VI	
Health status	II,V,VI	
Nutritional intake		Nutrica 3.0 (5-day dietary records)

FORCE PLATFORM MEASUREMENTS. For the assessment of specific biomechanical characteristics of sprinting the subjects run 60-m trials on an indoor running track. The measurement setup is shown in Figure 8. The kinematic stride cycle parameters (II: Fig. 1) were defined according to Weyand et al. (2000). In studies II and VI average net resultant GRF of braking (F_{brake}) and push-off (F_{push}) phases was used as specific force indicator (Mero and Komi 1986).

Lower extremity stiffness during running was determined according to Morin et al. (2005), using the concept of the spring-mass model (Fig. 7) (McMahon and Cheng 1990, Farley and Gonzalez 1996). The method is based on the modeling of the force-time curve by a sine function. The range of the stiffness values calculated by the sine-wave method was 0.67 to 6.93% lower than the corresponding values obtained using the force-platform derived measurements, thereby confirming the validity of the former method (Morin et al. 2005).

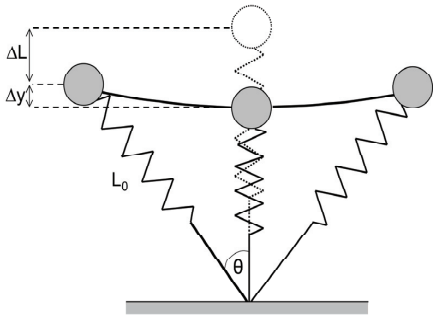

FIGURE 7 Schematic drawing showing the spring-mass model of running. The model consists of a point mass equivalent to body mass and a single leg spring. The initial length of the leg spring at the beginning of the contact phase is L_0 with θ denoting the half angle swept by the leg during the ground contact time. The compression of the leg is symbolized by ΔL and the vertical displacement of the center of mass during contact is represented by Δy.

The stiffness measurements calculated by the sine-wave method were based on the following formulas (Morin et al. 2005): vertical stiffness (k_{vert} in kN/m) was defined by

$$k_{vert} = Fz_{max} \cdot \Delta y_c^{-1} \qquad \text{(eq. 1)},$$

where Fz_{max} is the maximal vertical GRF (in kN) and Δy_c is the vertical displacement of the center of mass (in m) at the instant when the center of mass reaches its lowest point. In this study, Fz_{max} was obtained from the force platform recordings and Δy_c estimated by

$$\Delta y_c = -\frac{Fz_{max}}{m} \cdot \frac{t_c^2}{\pi^2} + g \cdot \frac{t_c^2}{8} \qquad \text{(eq. 2)},$$

where m is the subject's body mass (in kg), g is the acceleration due to gravity, and t_c is the contact time (in s) registered by the force platform.

Leg stiffness (k_{leg} in kN/m) was defined as the ratio of Fz_{max} to maximal displacement of the leg spring (in m) during the contact:

$$k_{leg} = Fz_{max} \cdot \Delta L^{-1} \quad \text{(eq. 3)},$$

$$\text{with} \qquad \Delta L = L_0 - \sqrt{L^2 - \left(\frac{vt_c}{2}\right)^2} + \Delta y_c \quad \text{(eq. 4)},$$

where L_0 is the initial leg length of the subject (measured as the distance between the greater trochanter and the ground in the standing position), v is mean 10 m velocity (in m/s) measured by photocell gates and Δy_c is the vertical displacement of the center of mass (in m). Vertical and leg stiffness are also given in body-weight normalized (kN/m/kg) values. The stiffness parameters were calculated from 2 contacts for each side and averaged.

In the methodological study (III), the intra-subject variability, calculated as coefficient of variation (CV%=(SD/mean) · 100%) of 4-trial mean, and symmetry of 15 GRF and 4 kinematic parameters were examined. The asymmetry between dominant and non-dominant limb was quantified by utilizing symmetry index:

$$SI(\%) = \frac{X_D - X_{ND}}{1/2(X_D + X_{ND})} \cdot 100\% \quad \text{(eq. 5)},$$

where X_D and X_{ND} are variables for the dominant and non-dominant leg, respectively (Herzog et al. 1989). Accordingly, when SI is zero there is perfect symmetry between legs, while a positive and negative SI values indicate greater magnitude of the variables for dominant and non-dominant side, respectively. The data were also examined using the absolute symmetry index ASI=|SI|, in which case averaging positive and negative symmetry indices over several subjects does not lead to a zero value (Giakas and Baltzopoulos 1997).

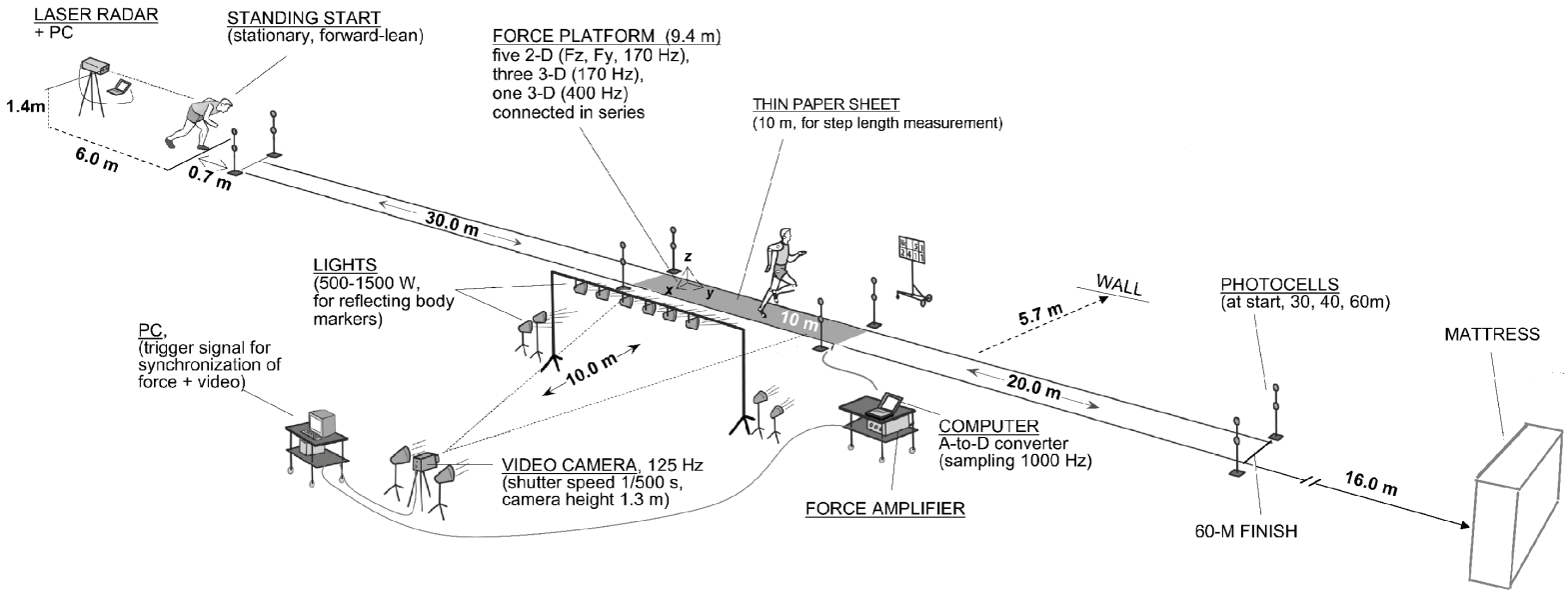

FIGURE 8 Schematic presentation of the experimental set-up for the analysis of speed curve, ground reaction forces and kinematic stride parameters during sprint running.

4.2.2 Post-competition blood lactate measurements

In order to estimate the level of anaerobic glycolytic energy production during maximal sprinting, blood samples were collected after the 100-m, 200-m, and 400-m finals in the European Veterans Athletics Championships. Two recovery blood samples for peak blood lactate concentration ($[La]_{b\ peak}$) were taken from the fingertip within 2-8 min after the runs. The $[La]_{b\ peak}$ was determined using Lactate Pro (Arkray, Inc. Japan) and the highest value of blood lactate concentration was included in the analyses. The measuring range of Lactate Pro is from 0.8 to 23.3 mmol/L. Previous studies have shown that the Lactate Pro gives reliable results for blood samples with high lactate concentrations (up to 20 mmol/L) and exhibits a high degree of accuracy with other laboratory lactate analyzers (Shimojo et al. 1993, Pyne et al. 2000). It should be recognized that in this method the lactate measurement is based on small plasma sample. Short high-intensity exercise can lead to loss of plasma from vascular space that might cause a concentration of constituent in the vascular space, including lactate (Davis et al. 2007). Accordingly, a possibility exists that peak blood lactate values uncorrected for potential exercise-induced plasma volume loss could overestimate the actual blood lactate level. Nevertheless, the same analytical method was used for all participants and the main finding should not be biased.

4.2.3 Muscle fiber measurements

MUSCLE BIOPSY AND HISTOCHEMICAL ANALYSES. Muscle samples were taken from the middle portion of the vastus lateralis of the dominant leg using a needle biopsy technique with suction. Transverse sections of 10 µm were stained for myofibrillar ATPase after acid (pH 4.37, 4.60) and alkaline (pH 10.30) preincubations (Brooke and Kaiser 1970). Six different fiber types (I, IC, IIC, IIA, IIAB, and IIB) were identified (Staron et al. 2000). The fiber area and relative proportion of the various fiber types were analyzed from the entire transverse section using an image analysis system (Sipilä et al. 2004). In earlier studies from the laboratory in our faculty, the reproducibility of fiber type and size determinations has been reported to be in the order of ~13–18% and 17–18%, respectively, when calculating the fibers from the whole transverse sections (Suominen 1978, Viitasalo et al. 1980).

GEL ELECTROPHORESIS. The myosin heavy chain (MyHC) isoform content of the muscle homogenates and single fibers was determined by sodium dodecyl sulphate-polyacrylamide gel electrophoresis (SDS-PAGE) (Andersen and Aagaard 2000). For the homogenate electrophoresis, 10-15 cryosections (10 µm) from each biopsy were placed into 700 µL of a lysine buffer 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 at 4°C at a constant voltage of 70 V for 42 h. Following the run, the

gels were fixed for 24 h in 5% acetic acid and 50% methanol and stained using Coomassie blue. In the stained gels three distinct protein bands could be separated and identified as MyHC I, IIa, or IIx isoforms.

SINGLE-FIBER CONTRACTILE MEASUREMENTS. Intrinsic ability of permeabilized muscle fibers to generate force and speed was studied by using single muscle fiber technique (Larsson and Moss 1993). All contractile measurements were carried out at 15 °C. A single fiber segment was mounted in an experimental apparatus leaving an average fiber segment length of 2 mm exposed to the solution between the connectors leading to the force transducer and a motor lever arm (Fig. 9). The composition of the solution is varied by altering concentrations of free Ca^{2+} and the mechanical responses are examined. Maximum tension (P_o) is determined from the difference between the fiber force in activating ($10^{-4.5}$ M) and relaxing (10^{-9} M) solutions that are expressed as pCa (-log[Ca^{2+}]). While the fiber is in relaxing solution, the sarcomere length was set to 2.79±0.01 µm (range 2.66–2.85 µm) by adjusting the overall segment length. Specific tension was calculated as P_o normalized to CSA and was corrected for the 20% swelling that is known to occur during skinning (Godt and Maughan 1981).

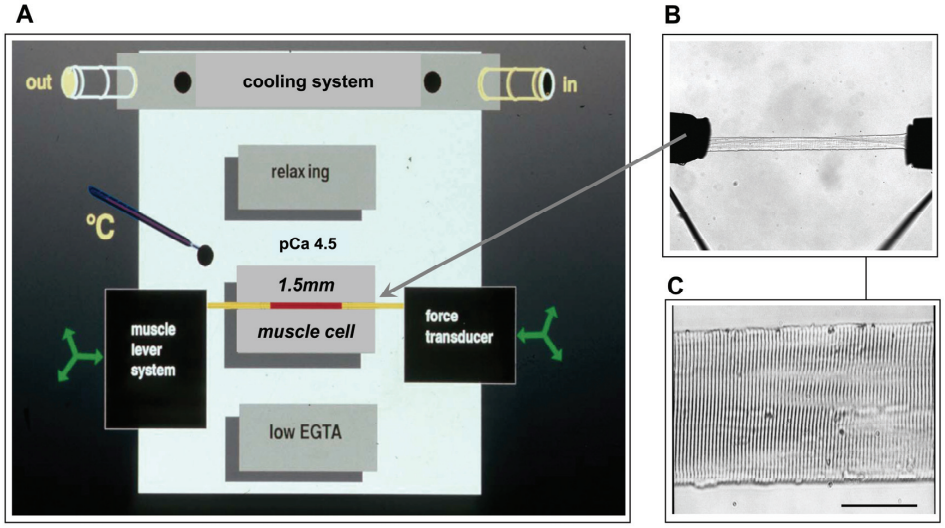

FIGURE 9 Design of experimental apparatus for recording force and movement of single muscle fibers (A) and microscopy photos of fiber segment mounted between the force transducer and motor lever arm (B) and micrograph of the fiber in relaxing solution (C). The horizontal bar denotes 50 µm (C).

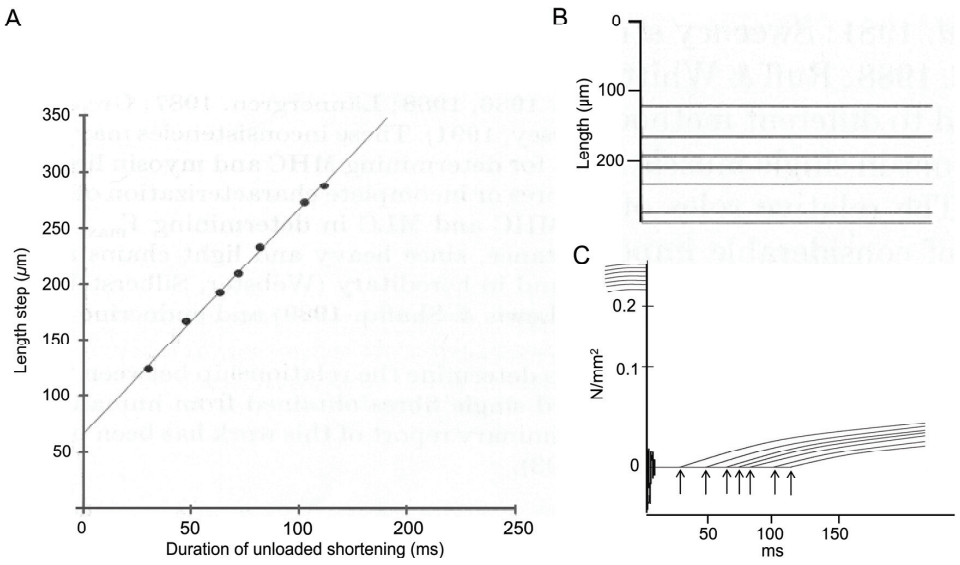

FIGURE 10 Determination of single fiber unloaded shortening velocity (V_o) using the slack test. After reaching of peak force at maximal Ca^{2+} activation (pCa 4.5) and optimal sarcomere length, the fiber is rapidly (within 1-2 ms) released, causing force to fall to zero. The fiber shortens under zero load until slack is taken up (arrows, time of unloaded shortening) and then begins to redevelop force (C). The fiber is activated and released 8-10 times using different length steps (B), and V_o is determined as the slope of resultant straight line (Δlength vs. Δduration of unloaded shortening) (A).

Maximum velocity of unloaded shortening (V_o) is measured by the slack test procedure as shown in Figure 10. The contractile recordings were accepted for subsequent analyses if a V_o value was based on linear regressions including four or more data points. Data were discarded if the coefficient of reliability r for the fitted line was less than 0.97, if the sarcomere length during isometric tension development changed by more than 0.10 μm compared with the sarcomere length in the relaxed fiber, or if maximum force changed more than 10% from first to final activation (Moss 1979). These acceptance criteria for contractile measurements were met by 44-51% of the analyzed fibers (V, VI). After mechanical experiments, each single fiber, dissolved in sample buffer, was loaded onto a 6% SDS-PAGE gel and ran at 120 V for 24 h at 10°C. Gels were subsequently silver stained and MyHC isoforms were determined.

Of note, our research and previous investigations have demonstrated a large range of variance in single fiber contractile properties within and between individuals of the same age and consequently the single fiber findings could be biased by small number of fibers analyzed. For that reason, to improve the reliability of the results the following results combine data from study V with additional data obtained from the training intervention study VI (new analyzed fibers from the same older athletes).

4.2.4 Whole-muscle structure

Knee extensor and ankle plantar flexor structural/architectural characteristics were measured using B-mode ultrasonography (Kubo et al. 2003a). The thickness of the knee extensor (KE) muscles (vastus lateralis, VL; vastus medialis, VM; vastus intermedius, VIM; rectus femoris, RF) and plantar flexor (PF) muscles (gastrocnemius medialis, GM; gastrocnemius lateralis, GL) was determined as the distance between the upper and deeper aponeuroses (Fig. 11). The measurements for the VL, VIM and RF were taken at 50% (for VIM two anatomic sites: under VL and under RF) and for the VM at 30 % of the distance between the lateral condyle of the femur and greater trochanter (Fig. 11A). The corresponding measurement sites for the GM and GL were at 30% proximal between the lateral malleolus of the fibula and the lateral condyle of the tibia (Fig. 11B). The sum of the muscle thicknesses of all the knee extensor and plantar flexor muscles (KE+PF muscle thickness) was used as an indicator of muscle mass. Fascicle pennation angle and length were determined from the VL, GM and GL. The pennation angle was measured as the angle between the fascicle and the deeper aponeuroses. The fascicle length was calculated as the straight fascicular path distance between the upper and deeper aponeuroses of the most clearly visualized fascicles. However, in most cases the entire fascicle was not fully visible within the image are, and the fascicle length was estimated from the muscle thickness and pennation angle as follows: Fascicle length = Muscle thickness $\cdot \sin(\text{pennation angle})^{-1}$. The inter-day reproducibility (coefficient of variation, CV%) of the muscle architectural measurements using this method has previously been shown to be 2.5% for muscle thickness, 3.8% for pennation angle and 5.0% for fascicle length (Kubo et al. 2003b). In addition, previous studies have provided evidence that muscle thickness as determined by ultrasonography is a good estimate for muscle volume (Miyatani et al. 2004) and muscle mass (Sanada et al. 2006).

A

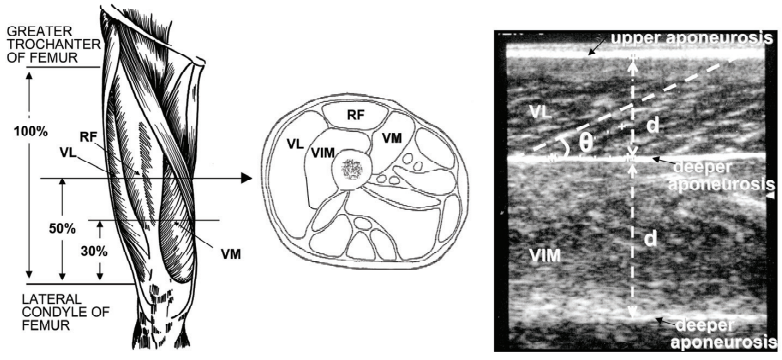

FIGURE 11 Analyzed knee extensor (A) and plantar flexor (B) muscles and examples of ultrasound images for vastus lateralis (VL) and vastus intermedialis (VIM) (50%) and gastrocnemius lateralis (GL). θ, fascicle pennation angle; d, muscle thickness. (continues)

B

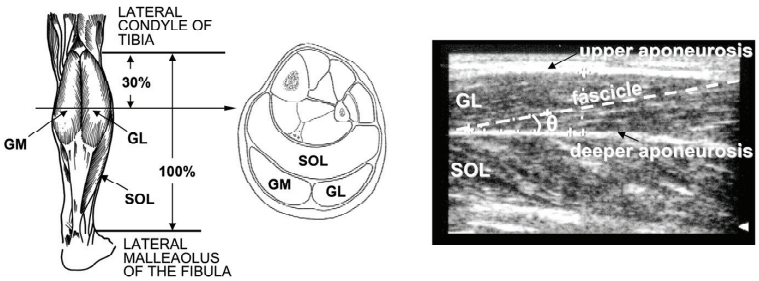

FIGURE 11 (continued)

4.2.5 Dynamic and isometric muscle strength

One-repetition maximum (1-RM) dynamic strength of the leg extensors was measured using a concentric half squat exercise in the Smith-machine (Fig. 12) (Häkkinen et al. 2002). Static squat jump (SJ) was used to examine explosive force production in concentric muscle action and countermovement jump (CMJ) under eccentric-concentric action (Asmussen and Bonde-Petersen 1974). The capacity to benefit from active stretch, i.e., potentiating influence of slow-type stretch-shortening cycle on jump performance was evaluated by comparing CMJ and SJ heights (prestretch augmentation %= [(CMJ–SJ)/SJ] · 100) (Walshe et al. 1996). The reactive jump test (series of straight legged vertical jumps for ~5 s) investigated the ability to rapidly change from an eccentric to concentric contraction (Bret et al. 2002) and standing triple-jump was used to measure explosiveness in cyclic action in horizontal direction (Mero et al. 1981). In previous studies, inter-day reproducibility of the rise of the body center of gravity has been reported to be good for squat jump (CV= 3.6%) and countermovement jump (CV=3.4%) in young adult athletes (Viitasalo 1985).

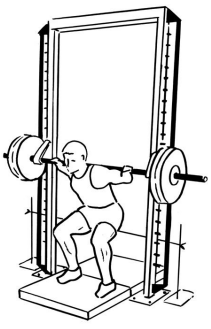

FIGURE 12 Half-squat exercise utilizing Smith-machine and force platform. The subject started from standing position and bended the knees to 90 degrees. This position was maintained for 1 s before extending up on a command. Assistants were in attendance during the lifts to ensure the subjects safety.

Bilateral isometric force of the leg extensors was measured by a dynamometer with the subject seated with knees and hip at 107° knee and 110° flexion, respectively (Fig. 13D) (Häkkinen et al. 1998b). The subjects were asked to perform contractions as explosively as possible. In the force-time analyses, maximal force, maximal rate of force development (RFD, 5 ms) and times taken to produce different absolute and relative force levels were calculated (Viitasalo and Komi 1978, Viitasalo et al. 1980). Maximal unilateral isometric torque of the knee extensors (Fig. 13A) and flexors (Fig. 13B) was measured by a dynamometer on the dominant leg with knee and hip angles of 90° and 110°, respectively (Häkkinen et al. 1998b). Maximal bilateral upright bench-press (Fig. 13C) was also tested isometrically with the 90° elbow flexion and the shoulders in a 90° abducted position. The inter-day reproducibility of the maximal isometric strength of lower limb muscles has reported to be high for different subject groups including young males and females (CV=4.1-6.5%, ICC=0.90-0.93 (Viitasalo and Komi 1975, Thorstensson 1976, Bamman et al. 1997) and middle-aged untrained women (CV 5.4-5.8%) (Heinonen et al. 1994). The reproducibility of isometric RFD has shown to be poor or moderate between days (e.g. CV 5-14%, ICC 0.3-0.7 (Heinonen et al. 1994) and in test-retest comparisons (8-18%) (Viitasalo et al. 1980, Heinonen et al. 1994), and that was also shown in study V (CV= 4.9-9.0%). On the basis of these findings, only maximal force was selected as the isometric strength variable in the training study (VI).

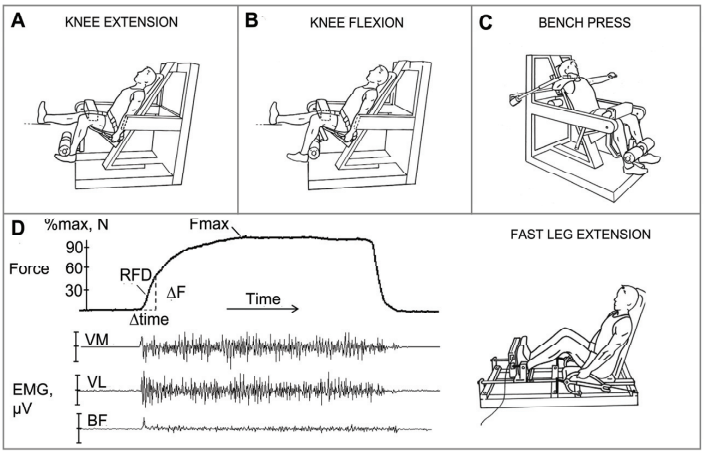

FIGURE 13 Isometric unilateral knee extension (A), unilateral knee flexion (B) and bench press (C) exercises using the David 200 dynamometer. Isometric bilateral leg extension, performed as fastly as possible, was measured by customized dynamometer (D). From the generated force-time curve, maximal force (Fmax), maximal rate of force development (RFD=ΔF/Δtime) and time taken to increase force certain absolute and relative force levels were measured. Lower tracings show raw electromyography (EMG) during rapid leg extension. EMGs were recorded from the vastus medialis (VM), vastus lateralis (VL) and biceps femoris (BF) muscles.

4.2.6 Electromyography

Electromyographic (EMG) activity during bilateral isometric leg extension, unilateral isometric knee extension, dynamic squat 1-RM and squat jump tests was recorded from the agonist muscles of the vastus lateralis (VL) and vastus medialis (VM) and from the antagonist muscles of biceps femoris (BF) of the dominant leg (Häkkinen et al. 1998b). The EMG activity was also measured during unilateral isometric knee flexion for the BF, when it acted as an agonist. The results are expressed as the mean integrated EMG activity (iEMG, (VL+VM)/2) analyzed for the peak force phase of the isometric actions (over a period of 1 s around the peak torque) and for the whole concentric phases of the half squat 1-RM and squat jump exercises. The iEMGs of the VL and VM during dynamic actions were also expressed as a percentage of their maximum isometric activity during knee extension (normalized iEMG). In previous studies, the agonist muscle iEMG has shown poor to satisfactory reproducibility for maximal isometric leg strength (CV=5-14%, ICC=0.3-0.7) in day-to-day comparison (Viitasalo and Komi 1975, Bamman et al. 1997) and for vertical jumping (ICC=0.70) in the 2-week test-retest evaluation (Goodwin et al. 1999) in young adults. The iEMG variability has found to be minimized by increasing the width of the signal averaging window up to 0.5-1.0 s (Heinonen et al. 1994, Bamman et al. 1997), and this was also taken into consideration in these measurements (VI).

4.2.7 Anthropometry

Body mass was measured using an electrical scale and a balance beam scale, and body height with a height gauge. Body fat percentage was estimated with the use of bioelectrical impedance. Leg length was measured with a ruler as the distance from the lateral malleolus to lateral condyle of the femur and thigh length from lateral condyle of the femur to the greater trochanter. Thigh circumference was measured at 50% thigh length using a tape while the subjects stood with their weight distributed evenly between both legs.

4.2.8 Hormone measurements

Subjects aged 40-84 years participating the second study phase were examined for serum basal concentrations of total testosterone (T), sex hormone-binding globulin (SHBG), and T/SHBG –ratio to obtain basic information about the hormonal status. Laboratory tests were run on fasting (12-h, ~8 h sleep) blood samples drawn from an antecubital vein in the morning (between 07:00 and 08:00 AM) of the second experimental day. Specimens were centrifuged (3500 rpm, 4°C for 10 min) and frozen at -75°C until assayed. The serum concentrations of total testosterone and were measured by automated chemiluminescent immunoassays using the IMMULITE 1000 system (Diagnostic Products Corporation, Los Angeles, California, USA). Analytical sensitivity of testosterone assay was 0.5 nmol/L. The intra-assay and inter-

assay CV of total testosterone varied, respectively, from 7.1% (at the level of 7.0 nmol/L) to 6.9% (at 27.4 nmol/L), and from 9.6% (at 7.0 nmol/L) to 7.7% (at 27.4 nmol/L). For SHBG, the sensitivity of assay was 0.2 nmol/L, with range in intra- and inter-assay CV, respectively, from 7.7% (at the level of 11 nmol/L) to 7.5% (121 nmol/L), and from 9.2% (12 nmol/L) to 5.8% (105 nmol). All samples for each participant were analyzed in the same assay for each hormone. In previous study from our laboratory, the physiological reproducibility (CV%; combined methodological and biological variability) was 18.9% for T, 9.8% for SHBG and 16.7% for T/SHBG –ratio in 45 men aged 49-73 years during 2-week control period measurements (Sallinen 2007).

4.3 Training program

The effects of increased strength training on the neuromuscular and sprint performance characteristics of older athletes were examined in third study phase (VI). The 20-week program, consisting of 3-4 training sessions per week, was designed by researchers and coaches in collaboration and utilized the knowledge from earlier studies in young adult athletes (Joch 1992, Delecluse 1997, Kraemer and Häkkinen 2002). In order to reduce the potential for overtraining and to optimize the adaptation, attention was paid to the proper periodization of training. The training program consisted of two 11- and 9-week periods that were further divided into three 3-4 week phases with variations in training intensity, volume and type (Fig. 14).

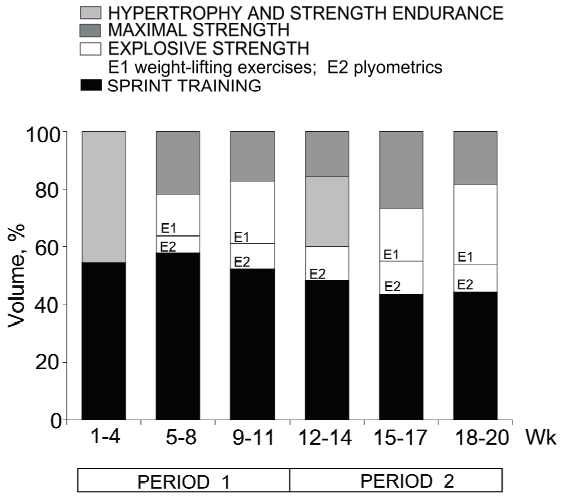

FIGURE 14 The planned training program and relative volume of different training modes during the 20-week study.

Briefly, strength training focused on leg extensor, hamstring and gluteal muscle groups and included different hypertrophic (3-4 sets x 8-12 reps at 50-70%), maximal strength (2-3 x 4-6 reps at 75-85%) and explosive-type weight lifting (2-3 x 4-6 reps at 35-60%; for high-load speed strength) and plyometric exercises (2-3 x 3-10 reps; for low-load speed strength) aimed at specifically stimulate fast motor units (VI: Appendix). Sprint type of training followed the athletes' usual training regimen but the overall volume of sprint exercises was decreased during this experiment when strength exercises were incorporated into the program. Sprint training included preparatory speed-endurance exercises (3-5 x 200-250 m at 75-85% of max speed), short sprints at high velocity (2-3 x 30-80 m at 90-98%), acceleration practices and technique drills. The training data of the subjects were investigated from the training logs that were collected every 5th week. The overall training adherence rate in EX, calculated as the percentage of training sessions successfully completed, was 86±4% for strength training and 83 ±6 % for sprint training across the 20-week study period. In EX, the average training hours and frequency over the training period were 2.1±0.2 h and 1.6±0.1 times per week for strength training and 2.1±0.3 h and 1.6±0.1 times per week for sprint training. Other exercises (ball games, aerobic running, skiing) were performed 0.5±0.2 h and 0.6±0.2 times per week. The controls maintained their previous training schedules and volumes (1.9±0.3 h and 1.8 times/week for sprint training, 0.6±0.1 h and 0.9±0.1 times/week for strength training, and 1.2±0.4 h and 1.1±0.3 times/week for other exercises) (VI).

4.4 Statistical analyses

The results are described as means with standard deviations (SD) or standard errors of the mean (SE). Interrelationships between different variables were studied by simple and age-adjusted Pearson correlation coefficients. Linear and polynomial regression analyses were used to estimate the rate of change in sprint and strength performance and fiber characteristics with age (I, II, IV, V). Differences in slopes of regression lines were assessed by using *F*-test (I, V). Stepwise multiple regression analyses were conducted to find out the combinations of muscle characteristics and age that explained the most variance in sprint performance (II). For inter-group comparisons, the normality of the distribution was analyzed with Kolmogorov-Smirnov test. In the case of normally distributed data, comparisons of parameters between groups were carried out by the Student's non-paired *t*-test or by one-way analyses of variance (ANOVA) when assessment included more than two groups. The Tukey's and Tamhane methods were used as the post hoc tests in ANOVA. In methodological study (III), a 2 x 2 ANOVA (two age groups x two sides) was used to examine the influence of age and leg dominance on the CV values. A paired *t*-test with Holm correction for multiple comparisons was used to compare the mean values of the biomechanical measures between dominant

and non-dominant legs within groups (III). In training intervention the data was non-normally distributed so the differences in changes between the study groups were analyzed using the Mann-Whitney non-parametric test and the Wilcoxon signed-ranks test was used for within-group comparison of baseline and post-training measurements (VI). The statistical analyses were done by the SPSS-statistical program (SPSS, Chicago, IL, USA). A $p<0.05$ was accepted as statistically significant in all analyses.

5 RESULTS

The result section includes the main findings for the male athletes from the total information obtained. More detailed information is given in the original articles I-VI. Some unpublished results are also included.

5.1 Sprint performance characteristics

5.1.1 Running times and velocity curve

The 100-m race times increased in curvilinear fashion with age from 11.14±0.19 s in 40–44 yr- to 17.80±0.57 s in 85–89 yr-old runners (Fig. 15A) (I). The same pattern of decline was also observed in indoor 60-m sprint trials in athletes across the adult age span (Fig. 15B) (II). In the 17–33-yr-old runners the 60-m time was 6.98±0.17 s, which then increased to 9.23±0.41 s among 70- to 82-yr-olds ($p<0.001$; +6%/decade) (II: Appendix 1).

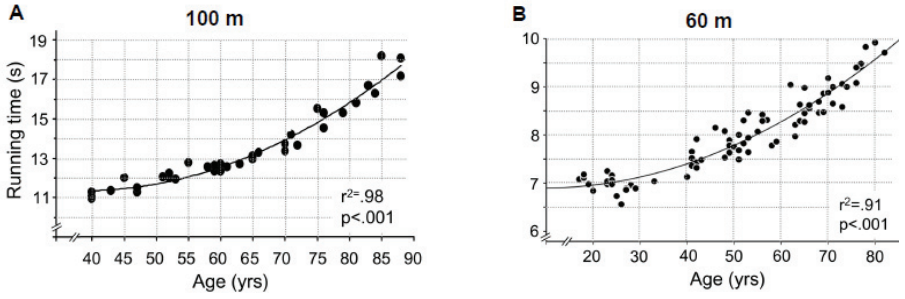

FIGURE 15 Individual values of 100-m and 60-m sprinting times with age.

The velocity curves of the 100-m races indicated that the distance required to reach the V_{max} in the 40- to 49-yr-old runners differed ($p<0.05$) from that in the 80–89 yr, whereas no differences were observed between groups in time required to

reach the V_{max} (I: Fig. 2). The results were confirmed in the 60-m trials using laser radar analyses (II), where distance required to reach V_{max} decreased from 37.5±5.8 m in 17–33-yr-old runners to 25.0±4.2 m in runners over 70 ($p<0.001$). No age group differences existed in time to peak velocity (range 4.07–4.68 s) (II). In the 100-m races, the relative decrease of velocity from peak velocity sequence to the final phase of the run ranged from 5.4 to 10.6% and was positively associated with age ($r=0.51$, $p<0.01$) (I). The decrease in relative speed from peak velocity sequence to the final phase of the run in 200 m (range 12.4 % to 14.9%) and 400 m (range 13.8% to 18.4%) did not differ with age (data not shown).

5.1.2 Maximum velocity and kinematic stride cycle parameters

Figure 16 shows the V_{max} and kinematic stride cycle parameters measured during the maximum speed phase of the 60-m trials (II). From youngest to oldest age group (average age difference 51.1 yr), there was a gradual decline in V_{max} (–5%/decade), L_{str} (–4%/decade) and $Freq_{str}$ (–1%/decade). When expressed relative to leg length, the age-related decline remained for L_{str} ($r=-0.77$, $p<0.001$; –3%) but not for $Freq_{str}$ ($r=0.10$). Of the temporal variables, t_{sw} remained unchanged, t_c (+5%), t_{brake} (+9%/decade) and t_{push} (+2%/decade) increased (Fig. 17) and t_{aer} decreased (–5%/decade, $p<0.001$) with age (II: Fig. 3).

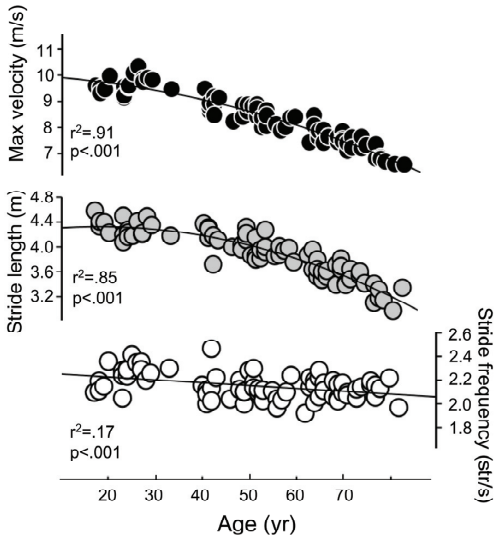

FIGURE 16 Relationships between age and maximum running velocity, stride length (L_{str}) and stride frequency ($Freq_{str}$). L_{str} was determined as the distance between consecutive contacts of the same foot (step length x 2) and $Freq_{str}$ from the inverse of the total stride cycle time [$1/t_{str}$, (=step frequency/2)].

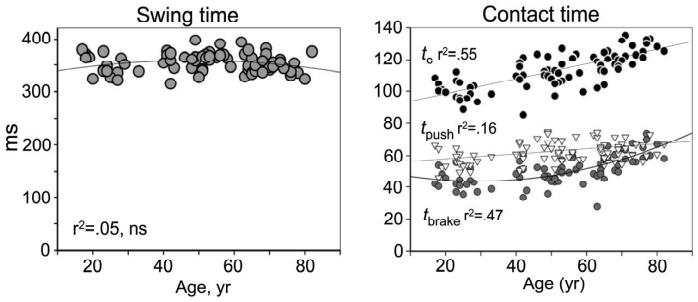

FIGURE 17
Relationships between age and swing time, total contact time (t_c), push-off time (t_{push}) and braking time (t_{brake}).

5.1.3 Ground reaction forces

Force production during maximum velocity sprinting was described using resultant GRFs as a specific force indicator (Fig. 18 A-C) (II). The average net resultant GRFs of the braking (F_{brake}) and push-off (F_{push}) phases declined with age (–4% and –6%, respectively). The F_{push}/F_{brake} ratio showed lower values in the 70- to 82-yr-old group. The mean angle of F_{push} became more vertically-oriented with age, whereas no age effect existed in the braking GRF angle.

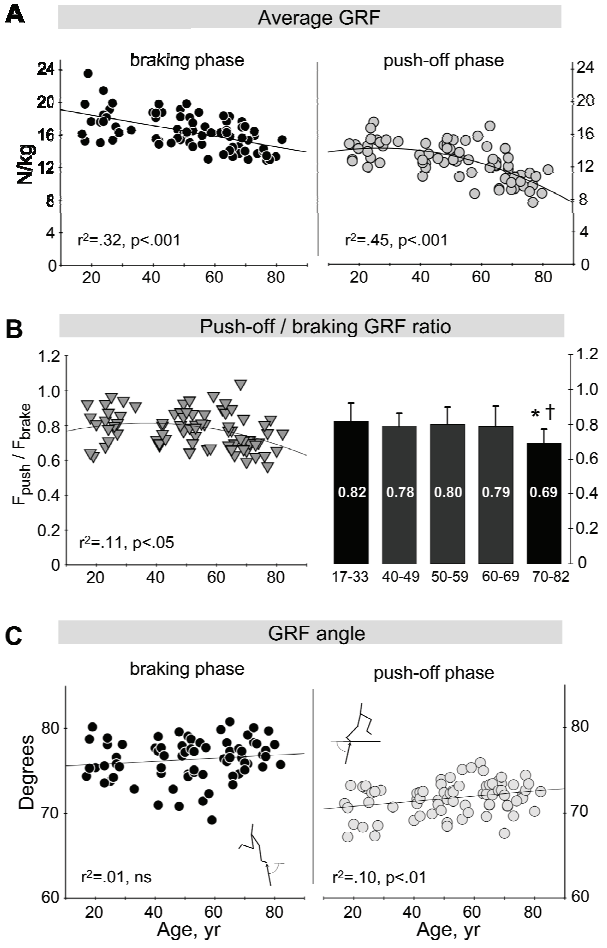

FIGURE 18
Relationships between age and the resultant GRF of the braking (F_{brake}) and push-off (F_{push}) contact phases (A), F_{push} as a fraction of F_{brake} (B), and mean angle of the resultant GRF from vertical, averaged over the braking (F_{brake}°) and push-off (F_{push}°) phases (C). *p<0.05 vs. 17- to 33-yr-old group, †p<0.01 vs. 50- to 59-yr-old group.

5.1.4 Lower extremity stiffness

A progressive age-related decrease in the vertical stiffness ($k_{vert}=Fz_{max}/\Delta y$) and leg stiffness ($k_{leg}= Fz_{max}/\Delta L$) in both absolute and body-weight normalized values was observed (Fig. 19C,D). The decline in k_{vert} was due to the combination of the decrease in Fz_{max} and increase in Δy. k_{leg} decreased with age because Fz_{max} decreased, while no change occurred in ΔL (Fig. 19A,B). In a separate analysis for the youngest athlete group (n=18) significant positive correlations between maximum velocity (range 9.1-10.3 m/s) and k_{vert} ($r=0.61$, $p<0.01$) and k_{leg} ($r=0.48$, $p<0.05$) were found.

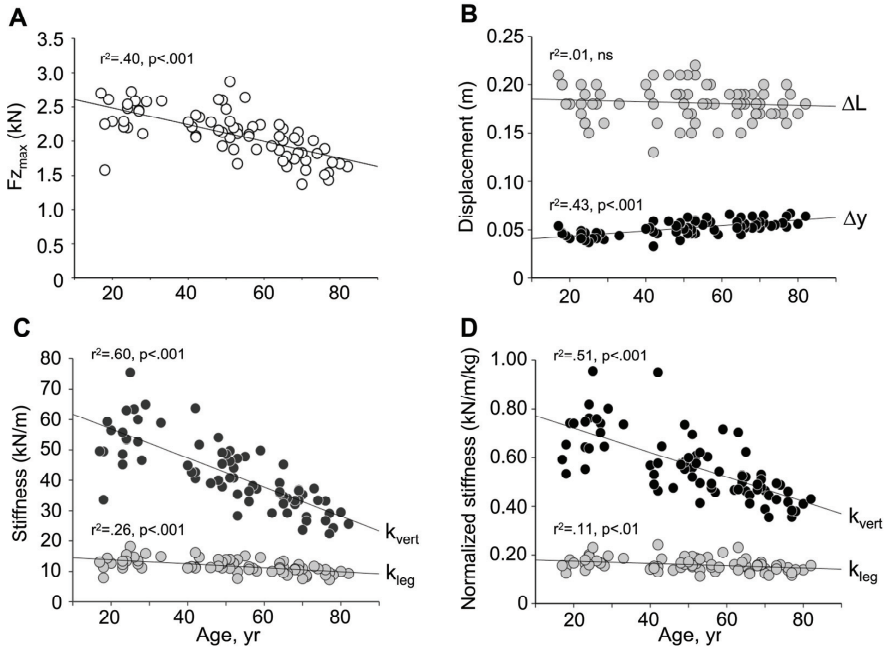

FIGURE 19 Relationships between age and the maximal vertical GRF (Fz_{max}) (A), vertical displacement of center of mass (Δy), displacement of the leg spring (ΔL) (B), vertical stiffness (k_{vert}), leg stiffness (k_{leg}) (C), body-weight normalized k_{vert} and k_{leg} (D).

5.1.5 Relationships among the biomechanical parameters

In the overall sample, V_{max} was significantly associated with all parameters, except t_{sw} (Table 4) (II). Of the parameters, L_{str}, t_c, F_{push}, and k_{vert} were the best correlates of V_{max}. When controlled for age, most of the significant correlations between the parameters remained, with t_c showing the strongest relationship with V_{max}.

TABLE 4 Simple and age-controlled correlations among the biomechanical variables. Values are correlations r (n=77). Significant correlations are in bold (*p<0.05, †p<0.01, ‡p<0.001). ˣ body weight adjusted values (F_{brake} and F_{push}= (N-N_{bw})/kg_{bw}), (k_{vert} and k_{leg}= kN/m/kg_{bw}).

Variables	V_{max}	L_{str}	$Freq_{str}$	t_c	t_{brake}	t_{push}	t_{sw}	t_{aer}	F_{brake}	F_{push}	k_{vert}
	Pearson correlation coefficient										
Max velocity (V_{max})											
Stride length (L_{str})	**.89‡**										
Stride freq ($Freq_{str}$)	**.53‡**	.09									
Contact time (t_c)	**–.84‡**	**–.62‡**	**–.70‡**								
Braking time (t_{brake})	**–.73‡**	**–.61‡**	**–.48‡**	**.81‡**							
Push-off time (t_{push})	**–.42‡**	–.21	**–.53‡**	**.57‡**	–.02						
Swing time (t_{sw})	–.11	**.33†**	**–.85‡**	**.27***	.08	**.35***					
Aerial time (t_{aer})	**.40‡**	**.69‡**	**–.41‡**	**–.35†**	**–.41‡**	.03	**.79‡**				
Braking GRF (F_{brake})ˣ	**.59‡**	**.61‡**	**.26***	**–.66‡**	**–.58‡**	**–.32†**	.11	**.52‡**			
Push-off GRF (F_{push}) ˣ	**.71‡**	**.65‡**	**.33†**	**–.74‡**	**–.83‡**	–.10	.08	**.54‡**	**.63‡**		
Vertical stiffness (k_{vert})ˣ	**.82‡**	**.57‡**	**.74‡**	**–.96‡**	**–.81‡**	**–.50‡**	**–.31†**	**.28***	**.64‡**	**.77‡**	
Leg stiffness (k_{leg}) ˣ	**.47‡**	**.25***	**.56‡**	**–.79‡**	**–.76‡**	**–.28***	–.20	**.28***	**.55‡**	**.76‡**	**.85‡**
	V_{max}	L_{str}	$Freq_{str}$	t_c	t_{brake}	t_{push}	t_{sw}	t_{aer}	F_{brake}	F_{push}	k_{vert}
	Partial correlation adjusted for age										
Max velocity (V_{max})											
Stride length (L_{str})	**.35†**										
Stride freq ($Freq_{str}$)	**.48‡**	**–.65‡**									
Contact time (t_c)	**–.65‡**	.12	**–.65‡**								
Braking time (t_{brake})	**–.56‡**	–.15	**–.32†**	**.67‡**							
Push-off time (t_{push})	–.14	**.33†**	**–.44‡**	**.45‡**	**–.36‡**						
Swing time (t_{sw})	**–.29***	**.73‡**	**–.93‡**	**.39‡**	.09	**.38†**					
Aerial time (t_{aer})	–.02	**.74‡**	**–.71‡**	–.04	–.20	.17	**.88‡**				
Braking GRF (F_{brake}) ˣ	.21	.15	.04	**–.44‡**	**–.35†**	–.13	.14	**.37‡**			
Push-off GRF (F_{push}) ˣ	**.44‡**	**.28***	.10	**–.53‡**	**–.73‡**	.21	.12	**.39‡**	**.44‡**		
Vertical stiffness (k_{vert})ˣ	**.62‡**	–.19	**.69‡**	**–.91‡**	**–.67‡**	**–.34†**	**–.43†**	–.04	**.41‡**	**.60‡**	
Leg stiffness (k_{leg}) ˣ	**.50‡**	–.08	**.49‡**	**–.86‡**	**–.75‡**	–.17	–.21	.17	**.47‡**	**.75‡**	**.93‡**

5.1.6 Variability and symmetry of biomechanical parameters

There were wide-ranging levels of variability in biomechanical parameters, with CVs ranging from 1.5% to 17.4% in the young participants, and from 1.4% to 21.1% in the older participants (Fig. 20) (III). In general, the variation in both groups was low (CV < 6%) in the vertical and resultant forces and in all the step temporal-spatial variables, but clearly higher for vertical loading (LR_{max}, LR_{ave}, Fz_{impact}) and horizontal forces. The CVs of LR_{max} (p=0.037), $Fy_{brake\text{-}max}$ (p<0.001), $Fy_{push\text{-}max}$ (p<0.001), $Fy_{push\text{-}ave}$ (p=0.003), and t_{aerial} (p<0.001), showed higher variability in the older than in the younger participants.

The symmetry index, SI (i.e., dominant vs. non-dominant leg), ranged from –5.4% to +5.6% in the young group, and from –2.6% to +2.7% in older group (III: Table 1). There was one significant age-related difference in the SI, with younger participants showing greater asymmetry for t_{aerial} than older participants (p=0.037).

The absolute inter-leg asymmetry (ASI) values ranged from 2.7% to 14.3% in the young group, and from 2.2% to 18.8% in the older group (III: Table 1). Age had a significant effect on absolute symmetry index of Fz_{max} that was higher in the young (3.8%) than in older (2.2%) group (p=0.016).

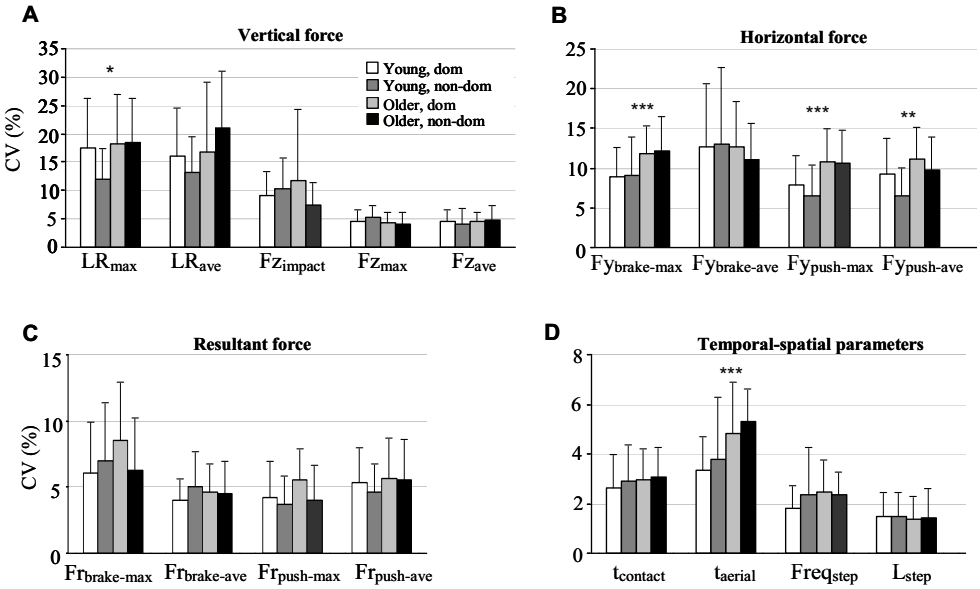

FIGURE 20 Intra-individual coefficient of variations (CV %) of the (A) vertical, (B) horizontal, and (C) resultant components of the GRF, and (D) the temporal-spatial step variables for the dominant (dom) and non-dominant (non-dom) legs in young and older sprinters. Bars indicate group means ± SD calculated from four steps of each leg. Significant age effect: *p<0.05, **p<0.01, ***p<0.001.

5.2 Blood lactate response to sprint running

Figure 21 shows the $[La]_{b\ peak}$ measured after competitive 100-m, 200-m, and 400-m sprinting (IV). The $[La]_{b\ peak}$ declined linearly in 100- and 200-m events and in curvilinear fashion in 400-m. However, 10-yr age-group comparisons with ANOVA showed no group differences in the $[La]_{b\ peak}$ before the oldest, +70 yr group (IV: Table 4). When all athletes in different age groups were considered, the $[La]_{b\ peak}$ correlated significantly with running times in all sprint events (IV: Table 5). Adjusting for age, the $[La]_{b\ peak}$ remained correlated with 400-m times.

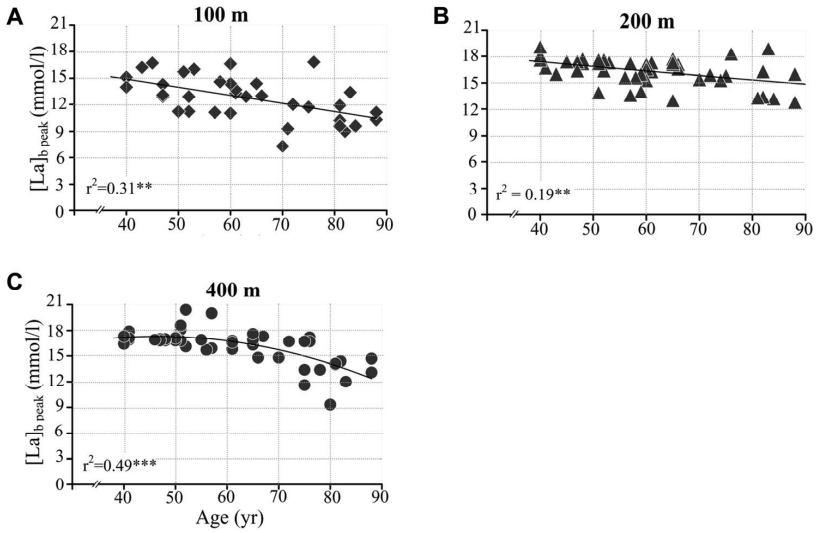

FIGURE 21 Relationships between age and $[La]_{b\ peak}$ after 100-m (A), 200-m (B), and 400-m (C) sprint races. Regression equations: **p<0.01, ***p<0.001.

5.3 Muscle structure

5.3.1 Muscle fiber area, distribution and MyHC isoform content

The mean cross-sectional area of type I fibers showed no significant association with age (Fig. 22A) (V). However, there was a progressive aging-associated reduction in type II fiber areas (Fig. 22B-D), leading to a decline in type IIA/I, IIAB/I, and IIB/I fiber area ratios with age (Fig. 22E). The relative distribution of the histochemically determined fiber types did not differ with age and was 54±11 for type II in the overall study sample.

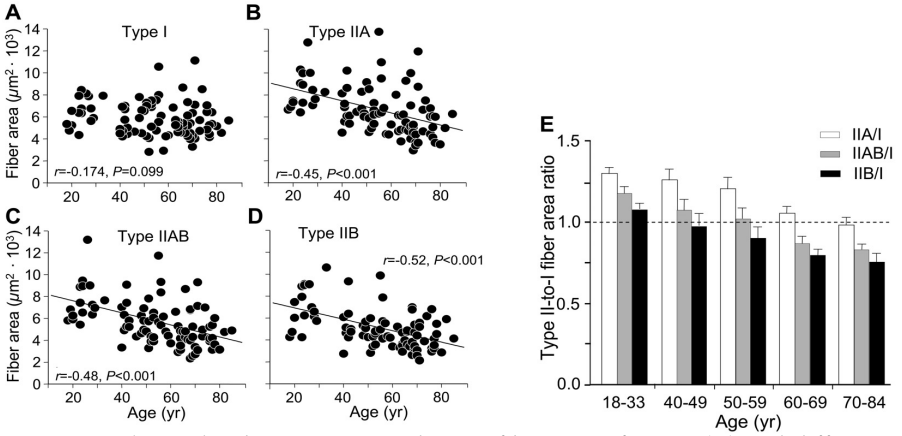

FIGURE 22 Relationship between age and mean fiber area of type I (A) and different type II muscle fibers (B-D), and the ratio of type II-to-type I fiber areas (E) of m. vastus lateralis. Type II fiber area and type IIA/I, IIAB/I, and IIB/I fiber area ratios decline with age (p<0.001). Mean ± SE.

The analysis of MyHC isoform composition of muscle homogenates showed an aging-related increase in relative content of MyHC I and a decrease in that of MyHC IIx, whereas no difference was observed in MyHC IIa expression (V: Fig. 5). The MyHC I isoform content was strongly associated with the corresponding histochemically determined relative area of type I fiber ($r=0.92$, $p<0.001$) (V).

5.3.2 Muscle thickness, pennation angle and fascicle length

The ultrasound results for the knee extensor and ankle plantar flexor muscle thickness and pennation angle are shown in Figure 23 (II). There was an age-related decline in KE (–6%/decade), PF (–2.5%/decade) and KE+PF muscle thickness (–5.5%/decade). This decline in muscle thickness was also observed in all individual KE and PF muscles (data not shown). Pennation angle was associated with age for VL but not for GM or GL. No significant relationships existed between age and fascicle length for VL, GM, or GL (II: Appendix 2).

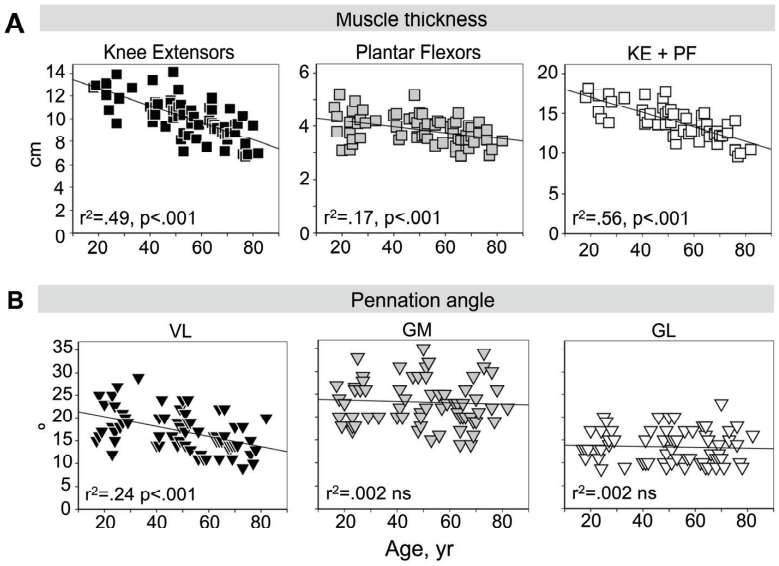

FIGURE 23 Association of age with knee extensor (KE) and ankle plantar flexor (PF) muscle thickness (A), and with pennation angle of m. vastus lateralis (VL), m. gastrocnemius medialis (GM), and m. gastrocnemius lateralis (GL) (B).

5.4 Muscle contractile function

5.4.1 Single-fiber contractile properties

Single fiber contractile function was analyzed in subgroups of young adult (18-33 yr, n=8) and older (53-77 yr, n= 9) runners (V). Maximal tension (P_o) of type IIa MyHC fibers was lower in the older than in younger subjects, but the difference in type I fibers remained only at trend level (p=0.13) (Fig. 24A). Older runners showed smaller fiber sizes (Fig, 24B). Difference in P_o of type IIa fibers was eliminated after adjusting for differences in fiber size (specific tension, ST), whereas the type I fibers from older runners showed higher ST values than those from younger athletes (Fig. 24C). The maximal unloaded shortening velocity of fibers (V_o) did not differ between young and older runners (Fig. 24D). In both age groups, there were no significant differences in P_o, CSA or ST values between fiber types. However, a trend towards greater P_o (p=0.055) and CSA (p=0.087) of type IIa than type I fibers was found in younger runners. Type IIa fibers were faster than type I fibers for both young and older runners (p<0.001).

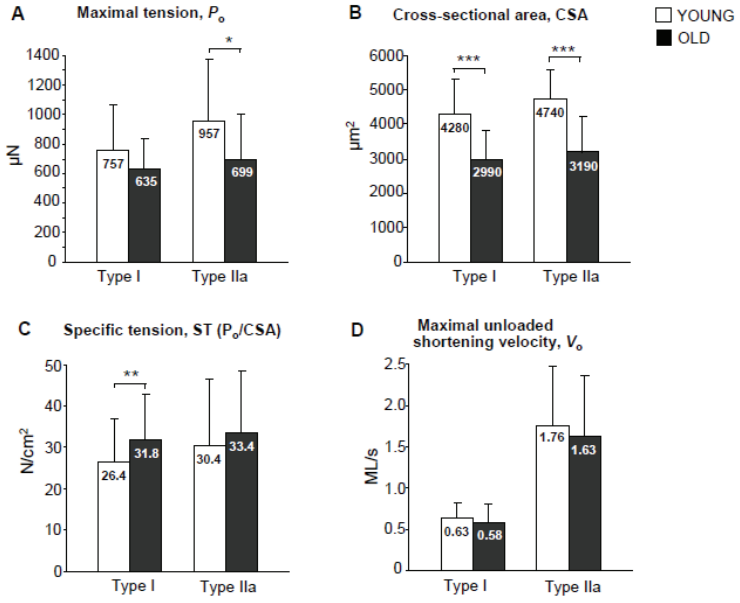

FIGURE 24 Maximal tension (A), cross-sectional area (B), specific tension (C), and maximal unloaded shortening velocity (D) in skinned single muscle fibers expressing type I and IIa MyHC isoforms in young (18-33 yr, n=8 and older (53-77 yr, n=9) runners. Values are mean ± SD. Significant age effect *p<0.05, **p<0.01, ***p<0.001.

5.4.2 Maximal and explosive muscle strength

Figure 25 shows selected dynamic strength variables (II, V). There were gradual age-associated declines in maximal concentric strength (half-squat 1-RM, –9%/decade; half-squat 1-RM/KE muscle thickness, –4%/decade, p<0.01), and in jumping performance (CMJ, –11%/decade; squat jump, –10%/decade, p<0.001) reactive jump, –11%/decade, p<0.001; standing triple jump –8%/decade, p<0.001 [40-82 yr]. The relative increase in jump height from SJ to CMJ (prestretch augmentation) did not differ with age.

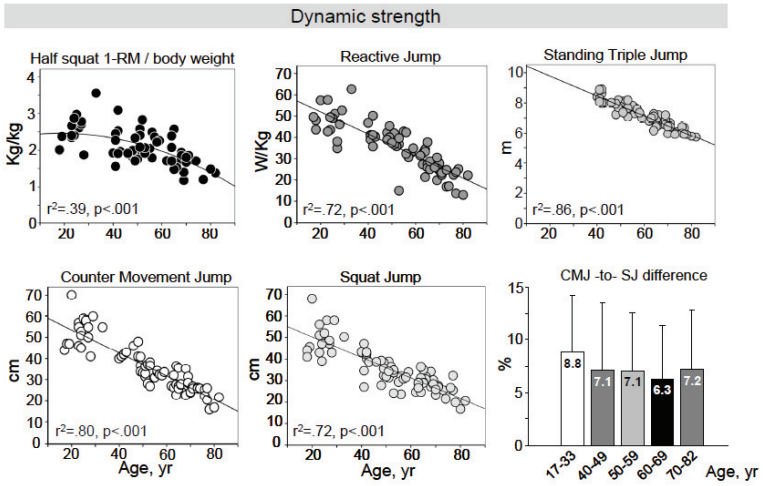

FIGURE 25 Relationship between age and dynamic strength measures.

Clear age-associated declines were also found in maximal isometric force of lower and upper body muscle groups (bilateral leg extension –8%/decade; bilateral leg extension RFD, –11%/decade; unilateral knee extension –7.5%/decade [40-82 yr]; unilateral knee flexion –8%/decade, p=0.013 [40-82 yr]; bench press –9.5%/decade, p<0.001)(Fig. 26). The knee flexor/knee extensor torque ratio did not differ with age (40-82 yr) (data not shown). When isometric force production was calculated as a force per KE muscle thicknesses (specific force), age-related difference in maximal force was eliminated in both bilateral leg extension and in unilateral knee extension (40-82 yr) (Fig. 26). Age differences in bilateral leg extension RFD persisted after adjusting for KE muscle thickness (Fig. 26).

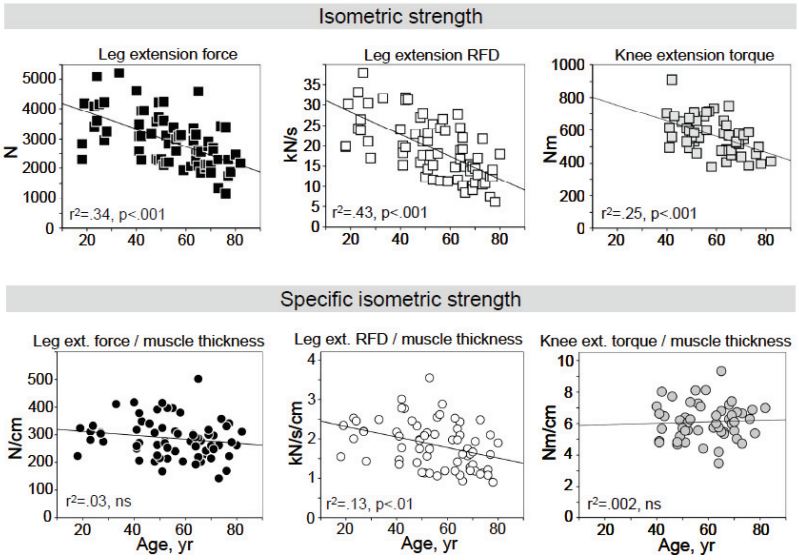

FIGURE 26 Relationship between age and isometric strength measures.

Force-time curves of fast bilateral isometric leg extension action indicated an aging-related decline in the absolute rate of force development (RFD: Δforce/Δtime) along with lengthening in times needed to reach specific force levels 100 N up to 2 000 N (Fig. 27). Furthermore, when the force-time curves were normalized to maximal force produced, there continued to be an aging-related lengthening in times time needed to reach 10-80 % of maximal force.

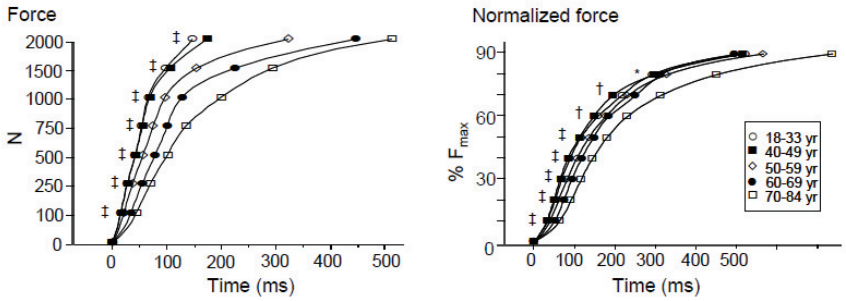

FIGURE 27 Force-time curves calculated for absolute values and normalized force-time curves calculated as a percentage of maximal force (F_{max}) developed in fast bilateral isometric leg extension action in different age groups. Asterisks at the side of force-time curve indicate significant age effect obtained by regression analysis: ***p<0.001; **p<0.01; *p<0.05.

5.5 Relative agonist and antagonist electromyographic activity during fast bilateral isometric leg extension

Figure 28 shows the agonist (VL, VM) iEMG activity in fast bilateral isometric leg extension action during 0-100 ms and 0-500 ms relative to maximum activation in the same action. There was an overall age-related decline in the relative iEMG of VL and VL+VM for the 0-100 ms time period (Fig. 28A). The VL and combined VL+VM activities during the first 100 ms were significantly lower from the maximum activity of the corresponding muscles in the two oldest group and 60-69-yr age group, respectively. Slight but nonsignificant overall age-related declines were observed in relative VL (p=0.06), VM (p=0.21), VL+VM (p=0.09) activity for 0-500 ms time period (Fig. 28B). On the other hand, the iEMG values differed significantly from the maximum activity in 50-59 yr and 70-84 yr age groups for VL and in the oldest group for VM and VL+VM.

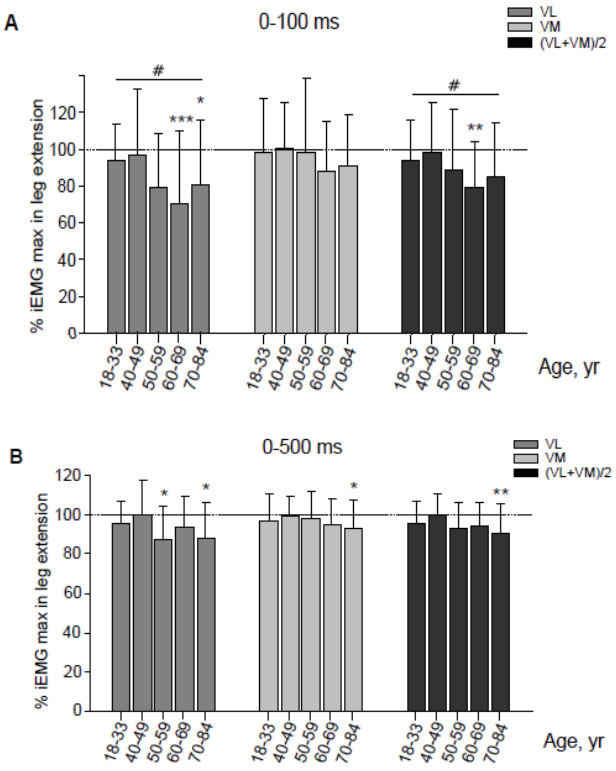

FIGURE 28 Agonist iEMG activity in leg extensors (m. vastus lateralis, VL; m. vastus medialis, VM) in fast bilateral isometric leg extension between 0 and 100 milliseconds (ms) and 0 and 500 ms. Significantly different from max iEMG value: * p<0.05; ** p<0.01; ***p<0.001. Significant age effect obtained by the regression analysis: #p<0.05.

Figure 29 presents the antagonist (BF) activities during rapid bilateral isometric leg extension action relative to maximum agonist values of the BF recorded during unilateral isometric knee flexion. The antagonist BF activity values during 0-100 ms (p=0.36), 0-500 ms (p=0.15) or maximal force phases (p=0.15) were not significantly related to age.

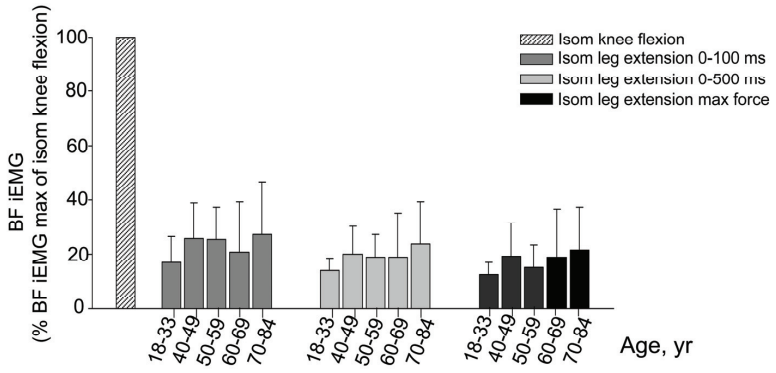

FIGURE 29 Antagonist biceps femoris (BF) iEMG coactivity (relative to maximal agonist values of the BF in knee flexion) during the rapid bilateral isometric leg extension in 0-100 ms, 0-500 ms and maximal force phase of the contraction.

5.6 Interrelationships between muscle and sprint performance characteristics

5.6.1 Relation of strength to muscle architecture, composition and electromyographic activity

Table 5 shows the age-adjusted correlations of selected muscle strength, structure and iEMG characteristics. Of absolute isometric strength, F_{max} correlated with KE thickness, while RFD correlated with the relative agonist [(VL+VM)/2] iEMG of 0-100 ms and 0-500 ms. When the isometric force was calculated as a percentage of maximum produced, the time to reach 30% of maximum was inversely associated with MyHC II%, iEMG 0-100 ms and iEMG 0-500 ms. The time to force level of 60% correlated with iEMGs 0-100 ms and 0-500 ms, while time to 90% force level was related to iEMG of 0-500 ms. With regard to dynamic strength, CMJ and SJ height correlated with MyHC II %. The squat 1-RM did not correlate with MyHC II% or KE muscle thickness, although the latter association approached statistical significance (p=0.06).

TABLE 5 Age-adjusted correlations between selected muscle structural, iEMG and strength characteristics. Values are correlations (r). Significant correlations are in bold: *p<0.05, †p<0.01, ‡p<0.001.

	Isom F_{max}	Isom RFD	t 30% F_{max}	t 60% F_{max}	t 90% F_{max}	Squat 1-RM	Squat Jump	CMJ
	Age-controlled partial correlation							
MyHC II %	0.06	0.18	**-0.32†**	–0.19	–0.03	0.15	**0.29***	**0.32†**
KE thickness	**0.30***	0.23	–0.18	–0.14	–0.24	0.26	0.06	0.09
VL fascicle length	–0.18	0.08	–0.09	–0.23	–0.08	0.09	0.07	–0.03
GM fascicle length	–0.08	0.04	–0.07	–0.16	–0.18	–0.13	–0.16	0.09
VL+VM iEMG 0-100ms	0.14	**0.48‡**	**-0.48‡**	**-0.36†**	–0.12	-	-	-
VL+VM iEMG 0-500 ms	0.00	**0.43‡**	**-0.50‡**	**-0.48‡**	**-0.31†**	-	-	-

In multiple stepwise regression analysis muscle thickness was the only predictor of isometric F_{max}, whereas both muscle thickness and relative iEMG entered the models for RFD and time to reach 30%, 60% and 90% force levels of the maximum (Table 6). Age and fascicle length did not enter into the models of for isometric strength measures. Conversely, using stepwise regression, only age entered to the models of dynamic strength parameters, explaining 43%, 77% and 82% of the variance in squat 1-RM, squat jump and CMJ, respectively. When age was intentionally left out, 41%, 48% and 53% of the variance in squat 1-RM, squat jump and CMJ, respectively, was accounted for by the KE+PF thickness, while MyHC did not enter to these models.

TABLE 6 Stepwise multiple regression analysis for the skeletal muscle structural factors and relative agonist EMG activation predicting isometric and dynamic force production characteristics.

Dependent variable	Predictors	Cumulative r^2	p-value
Isom F_{max}	KE thickness	0.51	<0.001
Isom RFD	KE thickness	0.39	<0.001
	iEMG 0-100 ms	0.52	<0.001
t 30% F_{max}	iEMG 0-100 ms	0.27	<0.001
	KE thickness	0.44	<0.001
t 60% F_{max}	iEMG 0-500 ms	0.27	<0.001
	KE thickness	0.39	<0.001
t 90%F_{max}	iEMG 0-500 ms	0.18	<0.001
	KE thickness	0.27	<0.001
Squat-1RM	Age	0.43	<0.001
Squat Jump	Age	0.77	<0.001
CMJ	Age	0.82	<0.001

5.6.2 Relation of sprint performance to muscle structural and strength characteristics

When adjusted for age, KE+PF thickness was associated with V_{max} k_{vert}, k_{leg} and t_{60m} (Table 7) (II). No other morphological characteristics were related to running parameters (data not shown). Of the muscle strength, squat 1-RM, CMJ and maximal isometric leg extension force correlated with the sprint parameters, but no associations were found between isometric leg extension RFD and any sprint performance characteristics.

TABLE 7 Age-adjusted correlations between selected biomechanical variables and muscle structural and strength characteristics. Values are correlations (r). Significant correlations are in bold: *p<0.05, †p<0.01, ‡p<0.001.

	V_{max}	L_{str}	$Freq_{str}$	t_c	F_{brake}	F_{push}	k_{vert}	k_{leg}	t_{60m}
	Partial correlation adjusted for age								
KE+PF thickness	**0.28***	0.16	0.11	–0.08	0.22	0.10	**0.48‡**	**0.44‡**	**–0.26***
Squat 1-RM	**0.33†**	–0.04	**0.30***	**–0.28***	–0.00	0.14	**0.52‡**	**0.48‡**	**–0.34†**
CMJ	**0.34†**	0.04	0.22	**–0.23***	0.00	0.17	0.21	0.16	**–0.28***
Isom F_{max}	**0.24***	0.12	0.09	–0.04	0.13	0.16	**0.33†**	**0.27***	–0.18
Isom RFD	0.08	0.16	-0.08	0.11	0.07	-0.04	0.16	0.11	-0.06

The stepwise regression models of the muscle predictors for V_{max}, F_{brake} and F_{push} are shown in Table 8. The variables entered were age, KE+PF thickness, type II fiber percentage, type II-to-I fiber area ratio, maximal isometric leg extension force and CMJ (n=54). Age was the strongest predictor of V_{max} and explained 89% of the total variance, with maximal isometric force being the only other factor (1%) to appear in the model. When age was intentionally excluded from the model, CMJ and KE+PF thickness appeared in the model and together explained 80% of the variance in V_{max} (not shown).

In the GRFs, KE+PF thickness was the only variable to enter the model for F_{brake} (26%), whereas maximum CMJ height was the only significant predictor of F_{push} (34%). Age did not enter the models for F_{brake} and F_{push}.

TABLE 8 Stepwise multiple regression analysis for the skeletal muscle factors predicting maximum sprinting velocity (V_{max}), and the average net resultant GRF of the braking (F_{brake}) and push-off (F_{push}) contact phases.

Dependent variable	Predictors	Cumulative r^2	p-value
V_{max}	Age	0.88	<0.001
	Isom F_{max}	0.89	0.012
F_{brake}	KE+PF thickness	0.26	<0.001
F_{push}	CMJ	0.34	<0.001

5.7 Effects of combined strength and sprint training on running performance and muscle characteristics

5.7.1 Sprint running performance

The experimental group (EX) showed a significant increase in the average resultant GRF of the push-off phase (8%) and decreases in contact times of the braking (9%) and push-off (5%) phases with training (Fig. 30A) (VI). These changes led to an increase in the RFD in both the braking (12%) and push-off (14%) phases. Maximum 10-m running speed improved from 7.47±0.28 to 7.74±0.31 m/s (4%, $p<0.01$) and 60-m sprint time from 8.69±0.30 to 8.52±0.29 m/s (2%, $p<0.01$). Stride length of the maximum speed phase increased from 1.79±0.06 to 1.85±0.08 m (3%, $p<0.05$), but no significant change occurred in stride rate (from 4.19±0.10 to 4.22±0.13 Hz). Training led to an increase in absolute leg stiffness ($Fz_{max}/\Delta L$) from 11.8±2.7 to 13.5±3.5 kN/m ($p<0.05$) and body weight normalized leg stiffness from 0.151±0.030 to 0.157±0.028 $kN/m/kg_{bw}$ ($p<0.05$). Increased leg stiffness was accompanied by a significant increase in Fz_{max} from 2.13±0.22 kN to 2.34±0.32 kN ($p<0.05$), while there was a trend towards a decrease in ΔL (from 0.188±0.041 to 0.180±0.036 m, $p=0.059$). In the control group (CTRL), no changes were observed in either the GRF characteristics (Fig. 30B) or in any of the other studied parameters of running. When comparing percentage changes over the intervention period, the push-off phase RFD ($p<0.05$), Fz_{max} ($p<0.05$) stride length ($p<0.05$), maximum 10-m speed ($p<0.05$) and overall 60-m sprint time ($p<0.01$) differed between the two groups.

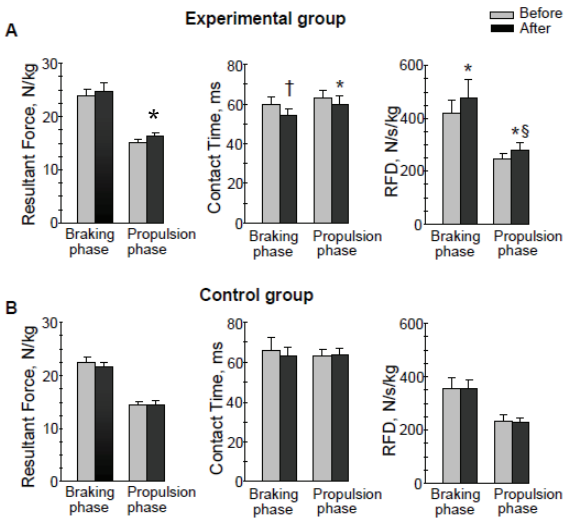

FIGURE 30 Selected ground reaction force characteristics of maximal running in the experimental (A) and control (B) groups. Values are mean ± SE. *$p<0.05$ for the comparison with the corresponding value before training; §$p<0.05$ for the comparison of the percentage change from before training with that in the control group.

5.7.2 Maximal and explosive muscle strength

Maximal isometric and dynamic strength before and after the 20-week period are shown in Figure 31. In EX, the maximal isometric torque during unilateral knee extension and knee flexion exercises increased by 21% and 40%, respectively. Maximum dynamic strength, as evaluated by bilateral concentric 1-RM squat, increased by 27% (n=5). Significant increases were also observed in the squat jump (10%), standing triple jump (4%), and the mechanical power of reactive jump test (29%). In CTRL, no significant changes were found in any of these parameters. When comparing percentage changes over the intervention period, all the jump test variables were increased significantly more for the EX than CTRL group.

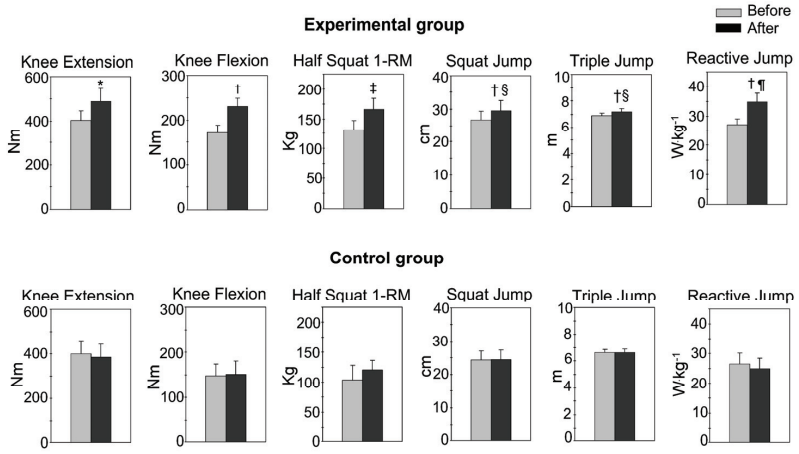

FIGURE 31 Isometric and dynamic force parameters in the experimental and control groups. Values are mean ± SE. †p<0.01, ‡ p<0.001 for the comparison with the corresponding value before training; §p<0.05, ¶p<0.01 for the comparison of the percentage change from before training with that in the control group.

5.7.3 Muscle composition

The histochemically determined fiber type distribution remained unaltered during the training period (VI: Table 2). In EX there was an increase in mean CSA of IIa fibers (20%, p<0.05), while the changes observed in type I (19%), IIAB (17%) and type IIB (33%) fibres were not statistically significant (p=0.3–0.1). In CTRL, no significant changes were observed in either muscle fiber type distribution or CSAs (p=0.6–0.3). The percentage changes over the experimental period in the CSA of type II and type IIA fibres were significantly (p<0.05) different between EX and CTRL. The relative content of type I, IIa and IIx MyHC isoforms in the muscle homogenates of the biopsies did not change over the 20-week period in either EX or CTRL.

5.7.4 Single-fiber properties

Single fiber CSA was measured in all the analyzed 470 fiber segments in EX (n=312) and CTRL (n=158), but statistical analyses were restricted to type I and IIa MyHC fibers due to the low number of type IIx MyHC fibers and fibres co-expressing the type I and IIa, and IIa and IIx MyHC isoforms. In line with histochemical analyses, EX showed a significant increase in CSA of the type IIa MyHC fibers (40%), while the change in CSA of type I fibers (22%) was not significant (VI: Fig. 5). The difference in change in type IIa fibres between EX and CTRL did not reach significance (p=0.097).

A total of 219 single membrane permeabilized muscle fiber segments out of 470 fibres fulfilled the criteria for acceptance for contractile measurements (EX 48%, CTRL 44%) (VI: Table 3). In EX, the increases in maximum force (P_o) in the cells expressing the type I (630±49 μN, n=45 vs. 800±155 μN, n=45 fibers) and IIa (640±137 μN, n=21 vs. 1010±187 μN, n=28) MyHC isoforms, respectively, did not reach statistical significance. In CTRL, P_o in fibres expressing the type I (630± 31 μN, n= 15 vs. 620±122 μN, n=12) and type IIa (740±77 μN, n=19 vs. 720±103 μN, n=15) MyHC isoform showed consistency before and after the 20-week period. The specific tension (ST), i.e., maximum force normalized to fiber CSA, in single type I and IIa MyHC fibres was not affected in either group. In maximum unloaded shortening velocity (V_o), EX showed no significant changes in type I (0.50±0.06 ML/s, n=45 vs. 0.61±0.05 ML/s, n=45) or type II 1.74±0.19 ML/s, n=21 vs. 1.83±0.26 ML/s, n=28) fibers after training. Similarly, no changes were present for the V_o of type I (0.57±0.10 ML/s, n=15 vs. 0.57±0.16 ML/s, n=12) or IIa (1.58±0.14 ML/s, n=19 vs. 1.66±0.33 ML/s, n=15) fibers in CTRL group.

5.7.5 Muscle electromyographic activity

In terms of absolute iEMG values of the agonist, EX showed a 9% increase ($p<0.05$) maximum iEMG of the VL+VM in the squat jump, but no significant changes were observed in the muscle activation concentric half squat 1-RM or during isometric knee extension (VI: Fig. 4A). The maximum iEMGs of the BF during the isometric knee flexion remained unaltered. The percentage increase in the squat jump iEMG by the EX group was significantly greater than the percentage change by the CTRL ($p<0.05$).

Statistically nonsignificant increases were observed when the iEMGs of the VL and VM obtained in the dynamic squat 1-RM and squat jump actions were normalized to their maximum isometric activity during knee extension (VI: Fig. 4A). In addition, no change was observed in the magnitude of antagonist BF iEMG coactivity (relative to maximum agonist values of the BF) during the isometric knee extension (28±8% before; 25±5% after), squat 1-RM (48±15% before; 47±10% after) or squat jump (55±8% before; 53±9% after) exercises. In CTRL, no significant alterations were observed in any of the muscle activity parameters (VI: 4B).

5.7.6 Relation between changes in muscle strength, fiber characteristics and electromyographic activity

The correlation analysis of pooled data showed that the increase in unilateral isometric knee extension torque was associated ($p<0.05$) with the increase in the cross-sectional area of histochemically determined type II fibers ($r=0.68$). No significant correlation between the changes in bilateral squat 1-RM strength and changes in fiber areas was observed. Of the explosive strength measures, the improvements in squat jump and triple jump correlated ($p<0.05$) with increases in type II fiber area ($r=0.71$; $r=0.66$, respectively), while improvement in reactive jump performance correlated with changes in the areas of both type I ($r= 0.68$, $p<0.05$) and II fibers ($r=0.62$, $p<0.05$). No significant association was found between changes in muscle strength and iEMG activity levels, although a trend for relationship between the changes in squat jump performance and in corresponding iEMG was noted ($r=0.51$, $p=0.11$).

6 DISCUSSION

This thesis examined young and master sprinters to gain knowledge about the effect of age and regular training on speed performance characteristics, a topic that has largely remained unstudied. There were four main components in this research. *First*, the effect of age on the biomechanical aspects of sprinting was examined by analyzing velocity curves, stride cycle parameters, ground reaction forces, lower extremity stiffness and the variability and symmetry of the performance measures. *Second*, post-competition blood lactate concentration was determined to obtain an insight into the effect of age on anaerobic lactacid energy production. *Third*, age-related differences in structural and functional characteristics of muscle and the role of these factors on maximal sprint performance were investigated. *Fourth*, the effects of a 20-week combined strength and sprint training program on neuromuscular and sprint performance characteristics were evaluated in older sprinters. The following discussion is organized in the above-mentioned order.

6.1 Sprint running performance

RUNNING TIMES AND VELOCITY CURVE. In accordance with the current age group world record data, our results (I, II) showed a nonlinear decline in 100-m and 60-m sprint performance with increasing age. Since many of the athletes had a life-long training and competition history, it was possible to estimate the validity of the cross-sectional performance data (II). The average rate of decline in 100-m sprint performance with advancing age in these athletes showed close similarity whether they were studied cross-sectionally (~0.5-0.6%/yr) or longitudinally (~0.6%/yr).

Age-related differences in the velocity curve over 100 m were described in paper I. With age, the time required to reach the peak velocity sequence remained unchanged, whereas the distance to the peak velocity phase declined significantly. This velocity curve pattern was confirmed in the 60-m trials using

more accurate laser radar analysis (II), that showed that distance to peak velocity decreased with age from about 40 m to 25 m, whereas time to peak velocity remained unchanged (range 4.1–4.7 s). Thus, it is the length of the acceleration phase that differs with age and ability of the sprinter. This is in line with the data on elite young sprinters who reach maximum speed at 60 m and more (Bruggemann and Glad 1990, Ae et al. 1994, Ritzdorf 1997, Ferro et al. 2001). We also found that during 100-m competitions, the relative loss in velocity from the peak velocity sequence to the end of the run became greater with age (from 5.4% to 10.6%). These values were somewhat greater when compared to the decreases of about 2-7% in velocity from peak velocity phase to the end of the run in young elite runners observed in major championships (Bruggemann and Glad 1990, Ae et al. 1994, Ritzdorf 1997, Ferro et al. 2001). In sum, it seems that the effect of age on overall sprint performance is characterized not only by decreased V_{max}, but also shorter distance to reach V_{max} and greater relative decrease in velocity toward the end of the run.

It is clear that sprint performance was preserved at a remarkably high level well into old age in these athletes. For example, sprint performance of the 70-84-yr-old sprinters was better than that of 64 untrained Finnish men with a mean age of 44 years (Surakka 2005). Nevertheless, data on speed performance in the untrained men from young adult to older age are lacking and thus it remains unknown whether the rate of loss may be less rapid in sprint-trained athletes than in the general population. To the best of my knowledge, the only evidence (indirect) comes from a sprint test up a staircase (4-5 s) that revealed a decrease in running speed of about 50% in men between 20-30 and 70 years of age, which is two-fold more than the decrease observed in the present study (Margaria et al. 1966).

STRIDE CYCLE PARAMETERS. The changes in stride length (L_{str}) and stride frequency ($Freq_{str}$) over 100-m competitive run were similar in runners of different age groups (I). In all groups, $Freq_{str}$ reached peak value rapidly in the run (10-20 m), whereas maximum L_{str} was achieved later. Further, the loss of velocity at the end of the run was explained by the decrease in $Freq_{str}$. The results on the maximum velocity phase indicated that the decrease in L_{str} contributed more to the age-related decline in V_{max} than $Freq_{str}$ (I, II). The decline in $Freq_{str}$ was related fully to increased contact time (t_c), since swing time (t_{sw}) did not differ between runners of different ages. Furthermore, when L_{str} and $Freq_{str}$ were normalized to leg length, the decline with age remained for L_{str} but not for $Freq_{str}$. This indicates that it was only the L_{str} aspect of velocity that was affected by age. These findings are supported by an earlier report on stride pattern in 83 elite-level male sprinters aged 30-94 years examined in competition conditions (Hamilton 1993). Information obtained from young experienced male sprinters (Ae et al. 1994, Ito et al. 2008) and male and female athlete comparisons (Mero 1987) have also provided evidence that L_{str} is a more important predictor of between-subject differences in maximum speed than $Freq_{str}$. For example, Ae et al. (1994) reported that at the World Championships

(Tokyo, 1991), the 100-m finalists (9.96 s, n=8) differed from the semifinalists (10.26 s, n=8) and runners who qualified for 2nd round (10.53 s, n=3) by longer absolute and body-height normalized L_{str}, whereas no group differences were observed in $Freq_{str}$.

GROUND REACTION FORCES. The magnitude of resultant GRFs of the braking (F_{brake}) and push-off (F_{push}) phases declined progressively with age and was reflected in changes in L_{str}, t_c and consequently in V_{max} (II). Along with the 27% age-related decline in V_{max} (from 9.7 to 7.1 m/s), F_{brake} and F_{push} decreased by 20% and 32%, respectively. These findings appears to be in general agreement with the previous study by Mero (1987) who found clear differences in F_{brake} and F_{push} between good and average young male sprinters and female sprinters.

As to the components of resultant GRF, our results showed a typical pattern, i.e., the average vertical force was about five times greater in magnitude than the horizontal anterior-posterior forces (III: Table 1). It is known that the impulse necessary to support body weight and to produce t_{aer} just long enough for repositioning the swing leg is a function of vertical force and time of contact. Considering this, differences in vertical force production are likely to have strong influence on the minimum time needed to be spent on the ground (Weyand et al. 2000), and may have been the mechanism behind the age-related increase in t_c. The importance of vertical GRF for sprinting was proposed by Weyand et al. (2000) who found that a small 1.26-fold inter-subject variation in vertical GRF was accompanied by a 1.8-fold difference in V_{max} (6.1-11.1 m/s) and reflected in t_c and L_{str}. This seems to be also consistent with the recent data suggesting that higher initial acceleration in earlier 100-m world record holder compared to that in national level runners was characterized by higher absolute and body mass-specific vertical GRF (Kobayashi et al. 2008).

On the other hand, studies on young elite sprinters have suggested that for the most efficient force production the sprinter must be able to transfer the body's center of gravity as fast as possible from vertical projection to horizontal movement (O'Conner 2000). Maximization of horizontal push-off force can decrease the angle of release and increase L_{str} while maintaining $Freq_{str}$. The present results showed a small, but significant age-related decrease in the mean angle of push-off resultant GRF (i.e., decrease in the push-off horizontal/vertical GRF ratio). To what extent this difference in GRF direction, even if minor, may impair the acceleration of the body in optimal horizontal direction and thus affect L_{str} and V_{max} sprint velocity is unknown at present.

Our subjects also demonstrated a decline in their t_{push}/t_{brake} and F_{push}/F_{brake} ratios with age, the decrements becoming evident in 70- to 82-year-old group (Fig. 32; II: Appendix 1). It could be hypothesized that high eccentric impact loads are less tolerated at older ages resulting in a longer braking phase. Moreover, it is likely that inability for fast eccentric force production at higher impact force conditions has a negative influence on the efficiency of the concentric push-off action. In this connection, it is interesting to note that F_{push} and L_{str} showed strong relationship with t_{brake}, while no significant association was seen with t_{push}.

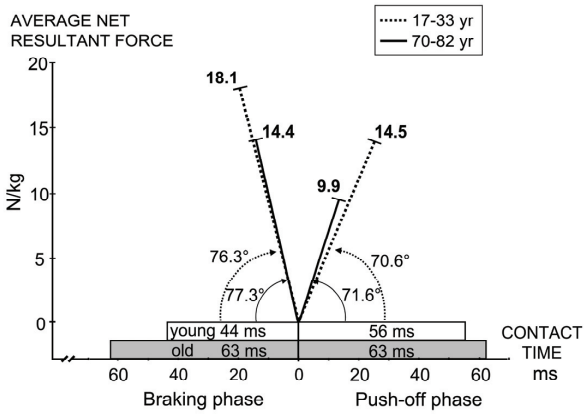

FIGURE 32 Resultant force production characteristics of maximum-speed running in the youngest and oldest athlete groups. There was a greater relative age-group difference in push-off than braking forces (decline in F_{push}/F_{brake} ratio) as well as greater age-related increase in braking than push-off ground contact time (decline in t_{push}/t_{brake} ratio). Further, the mean angle of F_{push} increased with age.

LOWER EXTREMITY STIFFNESS. In this study both leg stiffness (k_{leg}) and vertical stiffness (k_{vert}) decreased with increasing age (i.e., with decreasing velocity from ~9.7 m/s to ~7.1 m/s). Further, stiffness values were strongly negatively related to t_{brake}, suggesting that high stiffness is a prerequisite for tolerating higher impact loads and can lead to faster transition from the braking to the push-off phase. The finding of increased k_{vert} with increasing velocity is supported by most of the available investigations (Luhtanen and Komi 1980, Mero and Komi 1986, He et al. 1991, Farley and Gonzalez 1996, Arampatzis et al. 1999, Morin et al. 2005). Moreover, the result that the decline in k_{vert} is attributed to the combination of a decline in Fz_{max} and increase in Δy is in agreement with previous experiments (Farley and Gonzalez 1996, Arampatzis et al. 1999). In contrast, the data on k_{leg} is inconsistent. While many studies have indicated that k_{leg} does not change with increasing velocity (He et al. 1991, Farley and Gonzalez 1996, Morin et al. 2005), other studies have found a positive correlation between k_{leg} and running velocity (Arampatzis et al. 1999, Chelly and Denis 2001). It has been speculated that the lack of an association with velocity and k_{leg} in previous studies may be explained by submaximal velocity range (2-5 m/s) as well as the potential confounding influence of using non-athletic subjects (Brughelli and Cronin 2008). As to the spring-mass model components, our data indicated that the decrease in k_{leg} with increasing age was attributed to a decrease in Fz_{max}, while ΔL remained constant. This finding is in line with the previous data by Arampatzis et al. (1999). Moreover, in our training intervention significant increase in k_{leg} (14%) was due to an increase in Fz_{max} (10%), while no change occurred in leg compression.

It is currently unclear whether during contact aging affects limb posture (ankle, knee, hip angles) which in turn can affect stiffness. However, the

observation that the mean angle of F_{push} changed (i.e., decrease in horizontal push-off/vertical GRF ratio) may be an indication of an altered movement pattern and leg posture in runners of different ages. One possibility is that the younger (stronger) runners were more capable than the older runners of producing a similar level of leg spring compression with the knee joint more flexed. Such a leg posture has been found in young elite sprinters and is likely to enable more efficient transmission of the hip extension velocity to the foot backward velocity than if the driving leg is without knee flexion (Ito 1993). Differences in body position and landing angle could also explain the clear age-related differences in vertical displacement of the center of mass. However, in the absence of kinematic data, any conclusions on leg movement patterns remain speculative at best. Moreover, neural activity and muscle-tendon function should be examined in the future to fully understand the effect of aging on stiffness regulation during maximal running.

VARIABILITY AND SYMMETRY. The importance of variability as well as symmetry in data interpretation is obvious (Hamill and McNiven 1990). The total lack of knowledge about the effect of age on the reliability of biomechanical variables in sprinting led to the interest in the methodological study (III). The results showed low variability for most of the vertical and resultant GRFs as well as for of all the temporal-spatial stride parameters in young and older athlete groups (CV 1 to 6%) and agree with those of a previous study in young runners that indicated good stability of the average resultant GRFs during maximum-speed sprinting (CV 5.1-7.3% for two steps) (Mero and Komi 1986). These measures may be reliable enough to detect small changes in young and older sprinters' performance. Age-related increases in CVs were seen in the parameters describing vertical loading rate, horizontal GRFs and aerial time. However, the major age-related difference was in the horizontal GRF component, where three out of four of the examined parameters showed increased CV values with age. From a practical standpoint, it seems that in horizontal GRFs more steps are required to produce reliable values, especially for older runners, to ensure the biomechanical relevance of the differences observed. The study design does not allow us to draw any conclusions about the cause of age-related differences in variability. The exact mechanism responsible for the variability in locomotion is very difficult to determine as it may arise from deviation in one or multiple factors in the neuromuscular locomotor system, including the musculoskeletal dynamics, central pattern generator, supraspinal neural system and peripheral feedback mechanism (Masani et al. 2002). However, further research on these issues and the potential beneficial effect of movement variability for injury risk (Bartlett et al. 2007) might prove of value.

The symmetry analysis in the present study indicated no consistent age effect on the SI values between the dominant and non-dominant limbs. However, the SI equation is limited in that pooled data across participants can lead to a zero value, if some participants show superior measurements in the dominant leg and some in the non-dominant leg. When we did not take limb

dominance into account but examined only inter-limb differences (ASI %), the asymmetry of the parameters increased and were comparable in both the young (ASI 3-14%, mean 8% for all parameters) and older (2-19%, mean 8%) group. According to this result, clear variable-specific asymmetries between legs are present during sprinting, which would appear to support previous studies on submaximal-speed running (Williams et al. 1987, Belli et al. 1995) in young athletes. Therefore, testing runners bilaterally or using the average score of both sides is recommended.

6.2 Anaerobic lactacid energy production during sprinting

Changes in progressive sprinting speed correspond to the rate and magnitude of energy production in the anaerobic processes of PCr degradation and glycolysis, and oxidative metabolism in longer sprint distances. In the present investigation (IV), post-competition peak blood lactate concentration ($[La]_{b\ peak}$) was used as an indirect measure of anaerobic glycolytic energy production in the world-level master sprinters aged 40 to 88 years. The results showed that $[La]_{b\ peak}$ following 100- to 400-m races declined with age, the age-group differences becoming significant after 70 years of age. Moreover, the age-associated decline in $[La]_{b\ peak}$ was related to increases in running times. Therefore, these data support the view that reduced energy production via anaerobic glycolysis in older ages may be a factor in the curvilinear deterioration in overall sprint performance (Reaburn and Dascombe 2009). Our results also agree with the recent study by Benelli et al. (2007) who studied blood lactate response to competitive swimming in 52 male swimmers (40-79 yr) using the same analytical method. They found an age-related decline in $[La]_{b\ peak}$ following competitive 50-400-m events (from 14.2 to 8.2 mmol/l) the age-related difference becoming most salient after 70 years of age.

Total lactic acid production is highly correlated with the contractile muscle mass (Stainsby and Brooks 1990, Jensen-Urstad et al. 1994) and the amount of fast fibers (Essen and Haggmark 1975, Tesch et al. 1978) and therefore the age-related reduction in muscle mass via preferential atrophy of fast fibers could play major role in the observed changes in $[La]_{b\ peak}$. As far it is known, only one study has addressed the effect of aging on activities glycolytic regulatory enzymes in master sprinters. In his study, Reaburn (1993) found no decline in the activity of lactic acid-producing M-isozyme of lactate dehydrogenase (LDH), and total LDH in muscle homogenates in older sprinters compared with young sprinters, suggesting that glycolytic enzyme activity and thus muscle mass-specific capacity to generate lactate might be prevented by sprint-type training. In contrast, several studies in untrained people have reported a decline in activities of glycolytic enzymes (Larsson et al. 1978, Pastoris et al. 2000, Hunter et al. 2002, Kaczor et al. 2006). Further, well-controlled rat studies have suggested, that with normal aging the glycolytic function/lactate production is

decreased in muscles containing mainly fast glycolytic fibers as well as single fast type II fibers, but unchanged in muscles containing a large proportion of slow fibers (Campbell et al. 1991, Holloszy et al. 1991, Lowe et al. 2000, Hepple et al. 2004). Some of the animal studies have also provided evidence of impaired lactate transport capacity from muscle to blood with age, that could, in theory, lead to underestimation of the rate of anaerobic glycolysis (Juel et al. 1991, Hepple et al. 2004). Yet, it is believed that potential lactate trapping alone could not explain the lower age-related lactate efflux during contraction and that lactate production is really decreased in the old muscle (Hepple et al. 2004).

The present study does not allow drawing any firm conclusions about fatigue. The major concern related to the maintenance of the maximal speed towards the end of the sprinting is the depletion PCr and the increased contribution of glycolysis that leads to lactic-acid induced accumulation of H^+ ions (Hirvonen et al. 1987, Hirvonen et al. 1992). The reduced pH, in turn, may have direct negative effect on the contractile function and could also inhibit glycolytic rate (Fitts 2004). Nummela and coworkers (1992) have reported that in young runners the impairment in the force generating capacity (estimated from drop jumping height after 100-400 m runs), starts when the blood lactate concentration approaches the level of about 10 mmol/l. In our study, $[La]_{b\ peak}$ following 100- to 400-m races was at a high level (100m: 10.9-14.6 mmol/l, 200m: 15.4-17.4mmol/l, 400m: 14.1-17.5 mmol/l), suggesting muscle acidosis might have played a role in the fatigue response among these runners in all distances. Nevertheless, the finding that $[La]_{b\ peak}$ was not related to the loss of speed from the fastest race sequence to the final phase in these races suggests that fatigue could be explained also by other factors (levels of intramuscular PCr, ATP and glycogen, buffering capacity, neural fatigue etc.). Age-related increase in relative loss in velocity from the peak velocity sequence to the end of the run was observed in 100 m (from -5.4% to -10.6%) but not in 200 m (range -12.3% to -14.9%) or 400 m (range -13.8 to -18.4%). However, the interpretation of the 100-m event is complicated by the fact that older runners reach their maximum speed earlier and have thus longer fatigue-inducing speed-endurance phase. On the other hand, one could speculate that a similar loss of speed in longer sprint events may reflect age-related differences in race tactics. For example, a usual tactics for 400 m in older runners is to begin at lower relative pace than that in young runners and to rely more on aerobic metabolism throughout the race. Age-related reduction in high-intensity anaerobic exercises with age supports such race pacing.

6.3 Skeletal muscle structure and contractile function

MUSCLE STRUCTURE. In previous studies on master athletes it has become apparent that the muscle is capable of adapting to chronic training of different types. However, these studies have focused on strength, and particularly

endurance-trained, athletes and skeletal muscle adaptations to long-term sprint training remain largely unstudied. The present results (II) indicated that the sprint-trained subjects experienced a gradual age-related decline in muscle thickness both in the knee extensors (KE) (~0.6%/yr) and ankle plantar flexors (PF) (0.3%/yr). The rate of loss of thickness in KE agrees generally with previous results on KE (VL+VI) muscle thickness in untrained (0.8%/yr) and strength-trained master throwers (0.6%/yr) aged 40-75 years. Similarly, in the study by Pearson et al. (2002) the age-related loss of lean lower limb volume in elite level master weight lifters (0.8%/yr) compares well with our data.

The reason for the decline in muscle mass could be a decrease in the size of individual muscle fibers, a reduction in muscle fiber number, or a combination of the two. Our results from m. vastus lateralis indicated an age-related reduction in type II fiber area, whereas type I fiber area remained mostly unaffected (Fig. 22). This led to a decline in the type II-to-type I area ratio, which is the most probable explanation for the increase in type I MyHC isoform content with age, since the major fiber types showed no age-related differences. The maintenance of histochemically-determined fiber type percentage is in line with some (Saltin 1986) but not all (Trappe et al. 1995) findings in well-trained endurance athletes. Further, the greater reduction in the size of fast fibers with age confirms earlier studies in sprinters (Reaburn 1993), strength athletes (Brown and Coggan 1990, Klitgaard et al. 1990a), endurance runners (Reaburn 1993, Proctor et al. 1995, Widrick et al. 1996a) and untrained men (Tomonaga 1977, Larsson et al. 1979, Lexell et al. 1988). Nonetheless, on the basis of these studies, strength-trained athletes could maintain their type II-to-type I area ratio better than sprint or endurance athletes. It is possible that while the stimulus of sprint training exercises may be sufficient to ensure that all muscle fiber types are recruited, the overall duration of activation is too short or overload stimulus too small to cause hypertrophy of the fast fibers. Nevertheless, in older sprinters the fast fibers showed an up to 30 year "advantage" when compared to those in untrained men (V: Fig. 7A).

As to the other determinant of muscle mass, attempts have not yet been made to estimate the number of motor units affecting fiber number and loss of muscle mass in aging athletes. However, in sprinters muscle did not display the dominance of histochemically-determined slow-type fibers, or the fiber type grouping or increased coexpression of MyHC isoforms in the same fibers, that might reflect the denervation-reinnervation process of muscle fibers and which are typically found in untrained sarcopenic muscle (Klitgaard et al. 1990b, Andersen et al. 1999). A question arises as to whether systematic training is able to reduce the magnitude of the age-related loss of muscle fibers and/or motor units in the vastus lateralis muscle. Although apparently no attempts have been made to study this issue in aging elite athletes from any sports, some authors have hypothesized that systematic training may not protect skeletal muscles from the fiber and motor unit losses in the course of aging (Alway et al. 1996, Faulkner et al. 2008). On the other hand, it is notable that some data from a well-controlled animal experiment have indicated that elderly strength-trained

(plantar flexion, 6h/wk) male Wistar rats had a higher number of muscle fibers in trained soleus muscle compared to that in untrained older animals (Klitgaard et al. 1989b). This suggests that very intensive strength training could have some delaying effect on the age-related reduction of fiber numbers. Interesting, there seems to be large inter-individual variation in the total number of muscle fibers between individuals of the same-age (Lexell et al. 1988). In a recent paper, Faulkner et al. (2008) suggested that this variability may relate to polymorphisms in genes that control fiber number during embryonic development, such as myostatin and insulin-like growth factor 1 (IGF-I), and that such polymorphisms could play a role in athletic performance with age.

It is known that the muscle architecture affects the functional properties of muscle (Narici and Maganaris 2006). Our results on pennate-architecture muscles VL, GM and GL showed no age-related differences in fascicle length, suggesting the type of stress and strain on muscle in sprint training can attenuate the age-related loss of sarcomeres in series. This is line with findings of VL and GM on well-trained endurance runners (Karamanidis and Arampatzis 2006) but in conflict with other data showing shorter fascicle length of GM in older compared to young healthy sedentary men (Narici et al. 2003). Therefore, it can be assumed that regular exercise training in general can prevent the aging/disuse-related reduction in fiber length, thus maintaining the shortening velocity potential of the muscles. The present results, further, indicated that older age was associated with a decrease (30%) in the pennation angle of VL that may relate to reduced fiber size. However, the significance of the decline in the pennation angle in VL is not clear. While this change in the direction of fibers towards the distal tendon would imply lower contractile force potential (decline in physiological cross-sectional area), it may have a positive influence on contractile speed by increasing muscle fiber length in relation to whole muscle length.

SINGLE-FIBER CONTRACTILE FUNCTION. The few studies conducted on the effects of age and long-term training on single fiber contractile function have focused on endurance/strength athletes (Larsson et al. 1997, D'Antona et al. 2007), while it remains unknown what age changes in fiber contractility, if any, occur in sprint athletes. The present results indicated that the maximal shortening velocity, V_o, of slow type I and fast IIa MyHC fibers in older (53-77 yr) sprinters was similar or only slightly lower than that in young (18-33 yr) sprinters (Fig. 24D). This result differs from most previous findings of a significant decline in V_o in type I and/or type II fibers in untrained men (D'Antona et al. 2007, Ochala et al. 2007, Yu et al. 2007) and endurance/strength-trained men (Larsson et al. 1997, D'Antona et al. 2007). On the other hand, Widrick et al. (1996a) reported a higher V_o in type I fibers in middle-aged endurance runners compared with untrained men of the same age. This difference was thought to be related to the faster MyLC profile in runners. The electrophoretic analyses in the present study, however, demonstrated no age-related differences in MyLC isoform composition in either type I or II MyHC fibers (not reported).

Our data also showed that fiber-size adjusted force-generating capacity, i.e., specific tension (ST=P_o/CSA) of single fibers was similar (type IIa fibers) or even slightly higher (type I fibers) in older than in young runners (Fig. 24C). The maintenance of ST with age is in accordance with some (D'Antona et al. 2007) but not all results in endurance/strength athletes (Larsson et al. 1997) and in conflict with those of most studies in untrained men (Larsson et al. 1997, D'Antona et al. 2003, Ochala et al. 2007, Hvid et al. 2009). Our findings of increased type I fiber ST in older runners finds some support in the recent 9-year longitudinal study by Frontera et al. (2008) who reported about 28% increase in ST of type I fibers (p=0.07) in m. vastus lateralis in post-measurements in five elderly untrained men. Taken together, our ST results provide evidence that sprint-trained athletes show no significant age-related deterioration in the intrinsic quality (number of cross-bridges acting in parallel and the tension generated by each cross-bridge) of slow-type I and fast-type IIa MyHC fibers and that the properties of these fibers may be changed by a quantitative mechanism, i.e. loss of CSA. It should noted that the average age of the older subject group was 67.5 years and thus further investigations are needed to elucidate whether this protective effect of sprint training also applies to the elderly runners over 70 years of age.

MUSCLE ACTIVATION. In the present study the relative agonist (VL, VM) and antagonist (BF) surface EMG in the early phases of isometric leg extension was compared to that at maximum activation (Figs. 28-29). This EMG normalization allowed a more reliable inter-subject comparison, but confined the analysis to the early phases of muscular action. With respect to the agonist muscles, there was an age-related decline in neural activation; however a significant age effect was seen only in the first 100 ms for VL and VL+VM. Our results also indicated that the early iEMG values were lower than maximal iEMG in the older but not in the younger age groups. These results confirm those of a previous study on master throwers showing decreases in rapid neural activation of the agonist VL muscle (Ojanen et al. 2007).

What could be the mechanism underlying the loss of rapid neural activation of muscle? In a recent study in untrained subjects, intramuscular EMG data from the tibialis anterior muscle indicated lower motor unit discharge frequency along with a reduced number of doublet discharges (brief interspike intervals of ~5 ms) in older (71-84 yr, n=5) than young (18-22 yr, n=5) subjects during fast isometric dorsiflexion (Klass et al. 2008). Once again, however, it remains unclear whether these mechanisms seen in sedentary people apply to well-trained older athletes. For example, Leong et al. (1999) reported greater motor unit discharge rates in the rectus femoris muscle during slow maximal isometric knee extension in older weight-lifters (67-79 yr, n=7) compared with age-matched untrained men (n=5).

It is noteworthy that training may reduce the neural drive to antagonist muscles enhancing the net force production of the agonists (Häkkinen et al. 1998b). We found no significant age-related differences in the amount of BF

coactivity during the maximal force phase of isometric leg extension, that support the results of the study in elite weightlifters by Pearson et al. (2002). Furthermore, our EMG data expand the previous findings on master athletes that hamstring coactivation was unaffected by age not only in the late maximal force phase but also in the early rapid-force phase (0-100 ms) of isometric muscular action.

WHOLE-MUSCLE STRENGTH. Maximal and explosive strength, measured by both isometric and dynamic actions, declined progressively with age. The observation that strength had decreased already by around age 40 is in accordance with the findings of studies on master strength/power athletes (Grassi et al. 1991, Anton et al. 2004) and some larger studies of untrained men (Lindle et al. 1997, Lauretani et al. 2003). Previous studies indicate that sprint/power and strength athletes have an advantage of about 20-40 years in maximal and rapid force over untrained men (Grassi et al. 1991, Pearson et al. 2002, Ojanen et al. 2007).

We found that when the force data were normalized with respect to the values of young athletes values, the relative effect of age resembled that for maximal (~8-9%/decade) and explosive (10-11%/decade) strength qualities (II: Fig. 8). Interestingly, maximum sprinting speed was the least affected by age (5%/decade) in our athletes. Our observations differ from those of untrained subjects, where a much larger decline has been found in rapid than maximal force capacity (Skelton et al. 1994, Lauretani et al. 2003). Other beneficial adaptations could be that the relative differences in CMJ and SJ height (prestretch augmentation) (Fig. 25) as well as the isometric knee flexion/extension ratio were not affected by age. While the first observation seems to conflict with age-related decrease in elasticity found in untrained people (Bosco and Komi 1980), the second is in contrast to the greater loss observed in knee flexor vs. extensor function with age in recreational (≥20 km/wk) runners (Savelberg and Meijer 2004) and in master throwers (Ojanen et al. 2007). One could assume that the differential effect of age on the changes in force-production characteristics (speed, explosive strength, elasticity, knee flexor/extensor function) in the present sprinters may partially reflect training specificity for high-power stretch-shortening cycle actions that impose great demands on tendo-muscular elasticity and the hamstring muscles.

RELATIVE IMPORTANCE OF MUSCULAR AND NEURAL FACTORS. The foregoing data does not allow us to draw conclusions of the exact contribution of muscular and neural factors to the loss of mechanical performance. However, the findings of no age-related differences in single-fiber specific force and antagonist coactivation along with small non-significant decline isometric strength/muscle thickness ratio (Fig. 26) suggest that maximal isometric strength can be largely attributed to the loss of muscle mass. This possibility is also supported by the significant correlation between leg muscle thickness and maximal isometric strength ($r=0.71$) in the overall sample. However, the finding that a large portion of the variability in maximal isometric force was not explained by the

differences in muscle thickness may be related to the use of ultrasound muscle thickness as an indirect estimate of muscle size. It could also indicate that besides muscle size, other factors contributed to the age-related difference in maximal strength. For example, maximality of agonist muscle activation and synergist coactivation were not investigated in the present study and may thus not be excluded as contributing factors in determining maximal strength in these athletes.

Deterioration in the rapid isometric force production of the leg extensors in the sprinters studied here seemed to be explained not only by decreased muscle mass but also by some alteration of the muscle's capacity to produce force. This was indicated by reduced absolute isometric RFD/muscle thickness (Fig 26) as well as a decline in the slope of the normalized force-time curve (Fig. 27). Regarding potential neuromuscular contributors, the results indicated that the times required to reach force levels of 10-70% of relative isometric loads (V) and time to 30% of F_{max} when controlled for age (Table 5) correlated negatively with MyHC II content. A further reason for the loss of rapid isometric force capacity could be a decline in the rate of neural activation. We found that the relative iEMG value of the agonists was together with muscle thickness the most important predictors of fast isometric force production (Table 6). However, in contrast to previous studies in untrained men (Häkkinen et al. 1998a, Izquierdo et al. 1999), our data suggest no effect of age on antagonist activity during rapid isometric action (Fig. 29).

Vertical jump height correlated with MyHC II when controlled for age. In agreement with this, studies in older non-trained (Harridge et al. 1995, Sipilä et al. 2004) and young athletic subjects (Mero et al. 1981, Viitasalo et al. 1981) have indicated that fast dynamic actions are related to fast MyHC composition or percentage of type II fibers. The role of muscle activation capacity was not examined in dynamic performance for the sample overall. However, on the basis of the results of slowing of relative neural activation in the isometric strength test it can be suggested that age-related changes in neural activation may have taken place. Moreover, it is known that maximum strength has a great effect on power output at both light and high resistances (Stone et al. 2003) and therefore in older age deterioration of jumping ability may become increasingly more dependent on loss of maximal strength.

6.4 Skeletal muscle characteristics as determinants of sprint running performance

Traditionally, a large percentage of hypertrophied fast-type II fibers or high MyHC II isoform content, has been viewed as prerequisite for successful sprint performance (Andersen et al. 2000). For example, significant correlations between the relative number/area of type II fibers in the VL muscle and resultant GRF of the propulsive phase (Mero 1987) and maximum running

velocity (Mero et al. 1981, Mero 1987) have been found in young experienced sprinters. A somewhat new observation is that the fascicle length of the quadriceps (VL) and triceps surae (GM, GL) muscles could predict overall 100-m performance (Kumagai et al. 2000, Abe et al. 2001). In the present study (II, V) VL muscle fiber distribution, MyHC II isoform content or fascicle length (VL, GM, GL) were not significantly associated with sprint performance. Thus, it would appear that the contribution of muscle fiber-type composition and fascicle length to sprint performance decreases as a function of age while the role of some other factors increases. One can however not exclude that these factors could have been important if we had studied similarly aged older distance runners or non-athletes as well.

The overall loss of muscle size could become increasingly important for limiting sprint performance in master athletes. The results indicated that V_{max} and 60-m running times were significantly associated with combined KE+PF muscle thickness, when controlled for the effect of age. Moreover, both leg and vertical stiffness correlated with KE+PF muscle thickness and in the stepwise regression analysis muscle thickness was the strongest predictor of F_{brake}. In other words, it seems that the larger the leg muscles, the higher the stiffness (elasticity) and ability to produce/tolerate high eccentric forces, which can be as much as 5–fold greater than body weight. Although not having independent predictive value, it is possible that the selective reduction of fast fiber size and MyHC isoform content has a negative effect on the minimum ground contact time that the older runner is able to use. It should be noted that in older sprinters lower body weight is partially due to the age-related loss of muscle mass, and thus our method of controlling for average net GRFs for body mass [$N\text{-}N_{body\ mass}$)/$kg_{body\ mass}$] may underestimate the "true" significance of muscle thickness with respect to GRFs. In fact, when the average net GRFs ($N\text{-}N_{bw}$) were used in the stepwise regression analysis, KE+PF muscle thickness showed increased predictive value for F_{brake} (50%), and was also a primary predictor for F_{push} (54%), with CMJ playing a secondary role (5%) (data not shown). These findings are in line with Weyand et al. (2005) who estimated the vertical force requirement for different velocities in young athletes and concluded that the greater body masses of faster sprint specialists are directly associated with the greater ground support forces required to reach faster running speeds.

In terms of muscle strength, the stepwise regression analysis showed that CMJ height was a significant predictor of GRFs, explaining 34% of the variance in F_{push} (II). On the other hand, the correlation analysis indicated that maximal strength is significantly, but weakly, related to performance when controlled for age. These data do not allow safe conclusions to be drawn about the relative importance of different strength qualities (dynamic vs. isometric, maximal vs. explosive-strength) for performance in master sprinters. A perennial difficulty in interpreting data in this area is a lack of specificity in testing the strength required for sprinting. For example, the fact that sprint performance requires good force-generating capacity from a combination of muscle groups may affect the level of the association between sprint and simple strength measures.

Furthermore, very high-speed cyclic sprinting movements (e.g., even 600°/s for hip and ankle extensions), which involve continuous changes in muscle activation, length and velocity using stretch-shortening action, are impossible to simulate closely by means of strength tests. Ideally, strength measurements would identify the weakest links that set the limits to sprint-specific force production. Given the different strength and metabolic requirements of different phases of the run (acceleration, maximum speed, speed endurance), it is obvious that testing different aspects of sprinting itself can be a valuable tool in monitoring changes in overall performance.

As a whole, the mechanism underlying the age-related decline in maximum running speed is complex, and comprises a vast number of determinants. Figure 33 presents a simplified model of this phenomenon based on the biomechanical and skeletal muscle changes observed in this study.

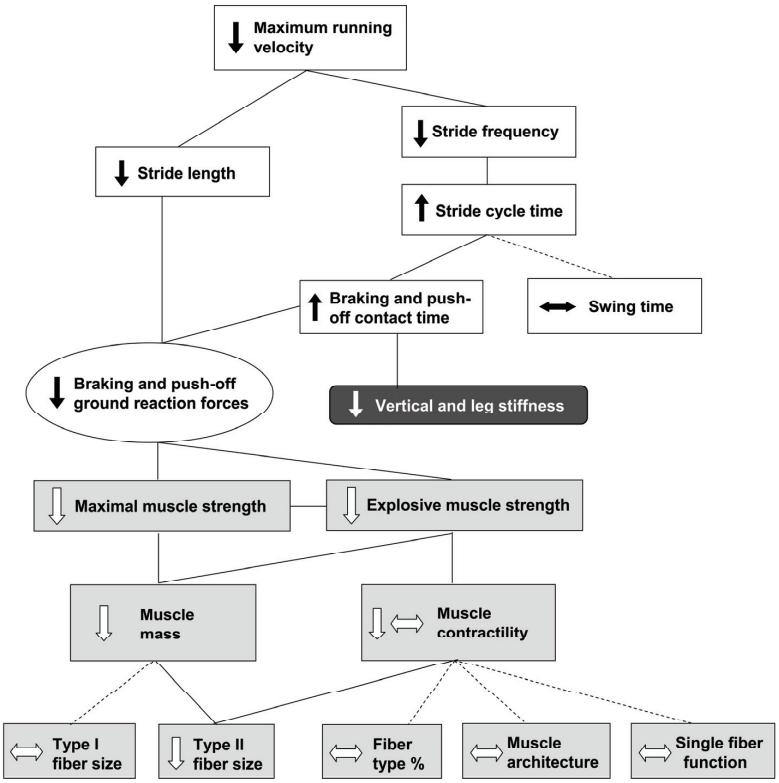

FIGURE 33 Simplified model of some of the biomechanical and skeletal muscle factors that may determine maximum running velocity with aging. Arrows represent significant age-related decrease (↓), increase (↑) and no alteration (↔) in the variables as indicated by the regression analyses.

6.5 Effects of combined strength and sprint training

Modified 20-weeks training, incorporating heavy-resistance and high-power strength exercises to running training, led to significant improvements in selected neuromuscular and performance characteristics in already highly-trained older sprinters (Fig. 34). With respect to running mechanics, training was associated with increases in $V_{max,}$ L_{step}, F_{push}, RFD, k_{leg}, and shortening of t_{brake} and t_{push} in the subjects in the EX group. This suggests that the improvements were targeted in particular at the biomechanical factors that were the most affected by age. Our results support previous findings of improvements in the 100-m (4%) and 300-m (2%) performance of male sprinters (55±6 yr) when hypertrophy-based strength training is included as part of overall training (Reaburn and Mackinnon 1995). For high-performance sprinters such improvements are marked and can be very meaningful for competitive success, as differences between the best athletes are usually very small.

WHOLE-MUSCLE STRENGTH. In agreement with the sprint performance results, the training led to significant improvements in both maximal and explosive strength (Fig. 31). The pre-post measurements showed an average improvement in maximal strength of 29% (21-40%) while the improvement in the explosive strength parameters was about 14% (4-29%). The observation of smaller increases in rapid force capacity may be explicable by the training background of the athletes, which already consisted of sprint running and jumping exercises. It is also noteworthy, that maximal isometric strength of knee flexors showed the greatest improvement (40%). It may indicate that the athletes in their previous training had paid limited attention to hamstring muscles, critical determinants of force production during sprinting.

The main objective of the strength training was to increase sprinting ability. Therefore, to optimize the transfer of strength gains to sprint performance, we carefully selected strength exercises that were sport-specific (muscle groups stressed, mechanical specificity). Nevertheless, it can be seen that the strength gains were much greater than the improvements in force production during running, suggesting that the transfer effect was not closely matched. This limited carry-over effect is in agreement with studies in young athletes showing about 1.5-2.5% improvements in sprint performance (30-m, 40-m) despite about 20-30% gain in muscle strength (Wilson et al. 1996, Harris et al. 2000). It is possible, that although the improvement in strength level can be achieved in a relatively short period, it requires longer time and large number of repetitions running-specific movements before the muscle groups collectively can utilize the improvements in strength in actual sprint performance.

MUSCLE ADAPTATIONS. Significant hypertrophy was observed in fast type IIa fibers in both histochemical (20%) and single fiber (40%) experiments. The magnitude of the increase in fiber size seems to correspond well with the magnitude of the change in maximal strength (21-40%). The increase in fast-fiber size supports the effectiveness of this training regimen and also demonstrates that there is a considerable adaptational potential for increases in myofibrillar protein in older athletes. However, although the fast muscle fibers responded to the training program, the type IIa-to-I fiber area ratio remained smaller than that observed in young athletes (V). More detailed investigations over longer experimental periods, with control groups, could yield further information about upper limits of fast fiber hypertrophy and whether the older athletes experience typical decline in protein synthesis rate of myofibrillar proteins (Welle et al. 1995) and other hypertrophy-related mechanisms.

No significant changes were found in MyHC isoform composition. That is in disagreement with the results of a previous experiment on young elite sprinters utilizing a similar training intervention (Andersen et al. 1994). In their study, Andersen and coworkers examined muscle biopsies from six elite male sprinters (20-27 yr) before and after a 12-week period of combined heavy-resistance and short interval-running and found a bi-directional switch in MyHC expression towards the IIa isoform (MyHC I→IIa ←IIx). However, this pattern is in contrast to the study of Cadefau and coworkers (1990) who found significant, approximately 8% increase in histochemically-determined type I fiber percentage along with insignificant decrease in type IIa, IIc, and IIb after an 8-month sprint training in 16 young athletes (16-17 yr). The explanation for the differential muscle fiber adaptations between these studies remains unclear, but could reflect differences in total training duration, and type, intensity and relative amount of strength and sprint exercises in the training programs. However, the MyHC regulation is complex interplay of mechanical and physiological factors and might be influenced, for example, age- and inter-individual differences in thyroid hormone (Caiozzo and Haddad 1996), growth hormone (Lange et al. 2002) and MyHC mRNA (Welle et al. 2000, Balagopal et al. 2001) profiles.

Improvement in muscle performance could also be related to increases in single fiber contractile function. We found insignificant increases in the maximum force (P_o) of type I (28%) and IIa (58%) MyHC fibers. These changes were mainly a result of fiber hypertrophy since no changes in specific tension ($ST=P_o/CSA$) were seen. Moreover, maximal unloaded shortening velocity remained unaffected. This lack of significant adaptation in specific tension and shortening velocity agrees with the findings of some sprint (Harridge et al. 1998) and strength (Widrick et al. 2002, Shoepe et al. 2003) training studies in young men, suggesting that mechanisms such as the maximum rate of cross-bridge cycling, number of active cross-bridges and the tension generated by each cross-bridge remain unchanged with training. In contrast, previous studies in sedentary older people have presented evidence of significant improvements in single fiber force (Trappe et al. 2000, Frontera et al. 2003) and shortening

velocity (Trappe et al. 2000) in response to strength training. In the sedentary persons the improvement in single fiber function could, however, reflect adaptation from the detraining state to the active usage of muscles, and therefore these findings should be compared with caution.

NEURAL ADAPTATIONS. The training-induced increases in maximal isometric knee extension and knee flexion torque as well as dynamic 1-RM squat in the EX group were accompanied by no significant change in the maximum iEMGs of the agonist muscles or antagonist co-activation (VI: Fig. 4). This observation suggests that the modified training stimulus did not result in increases in neural drive during maximal slow concentric, and especially isometric contractions, and that improvements in strength occurred via hypertrophic rather than intramuscular mechanisms. No change in the iEMG of the maximal strength test is contrast with many previous results in untrained older people (Moritani and deVries 1980, Häkkinen et al. 1996), suggesting that the magnitude of neural adaptations were also partially related to previous training status. Nevertheless, it is possible that that the largest increases in maximum iEMG take place during the earlier weeks of training reaching thereafter possibly plateau or even a periodical decrease during more prolonged training (Häkkinen and Komi 1983, Deschenes and Kraemer 2002). The iEMG results may be influenced to some degree by the design of this study. For example, the absence of iEMG adaptation in unilateral isometric knee extension and flexion could be attributed to the specificity of the training program, which consisted only of dynamic exercises. In support of this idea, Häkkinen et al. (1986) reported that the maximal iEMG response was higher when tested with the same exercise mode as that used in training (Häkkinen et al. 1996).

The 10% improvement in squat jump performance in EX was accompanied by a significant 9% increase in the iEMG of the agonist leg extensor muscles, and this was significantly greater than the change in the CTRL group. In theory, increased iEMG response in the initial movement phase could reflect increased rapid neural activation of motor units. Van Cutsem and coworkers (1998) studied a 12-week (5x/wk) training program consisting of fast ballistic contractions of the ankle dorsiflexors. The results indicated that the training led to more rapid onset of EMG that involved earlier motor unit activation along with increases in firing frequency and brief interspike intervals (doublets) in the EMG burst at the beginning of ballistic contractions (Van Cutsem et al. 1998). It has been suggested that even young athletes who are unfamiliar with strength training are not able to recruit the high-threshold fast motor units (Sale 1991). Accordingly, another possibility is that as a result of strength training the master athletes could recruit more and/or earlier the high-threshold motor units.

In this study, no EMG measurements were taken during the sprint tests and it thus remains unknown whether any changes in neuronal mechanisms occurred in response to training. However, our result of decreased contact time with an increase in GRF and a clear improvement in the reactive jump test

(29%) could be indicative of improved tolerance to high stretch loads. On the basis of previous studies on plyometric (Häkkinen et al. 1985, Schmidtbleicher et al. 1988) and strength training studies (Hortobagyi et al. 1996, Aagaard et al. 2000, Duclay et al. 2008), such improvements in reactive (stiffness) characteristics could be partially related to changes in inhibitory and/or facilitatory reflex mechanisms related to lengthening contractions; however, this explanation remains speculative.

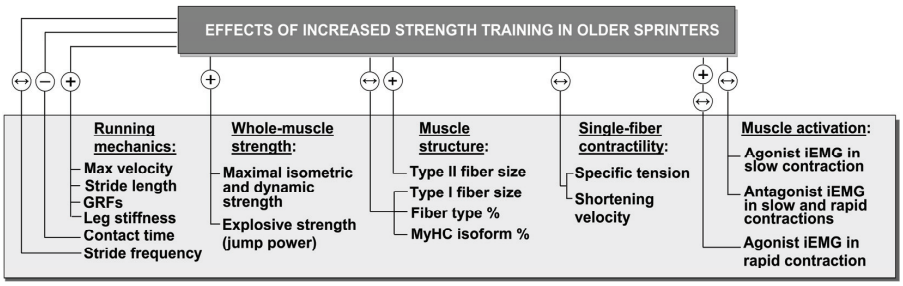

FIGURE 34 Schematic summary of effects of increased strength training on sprint performance, strength and neuromuscular characteristics in older athletes. ↔, no influence; -, decrease; + increase with training.

TRAINABILITY OF MASTER SPRINTERS. The observed aging of function reflects not only physiological changes but perhaps also a decrease in the volume and intensity of training. For the maintenance of sprint/power performance, one critical change in training could be a reduction in the amount of near-maximum or maximum workouts. For instance, according to the size principle of motor unit recruitment, the highest threshold motor units and fast fibers may not be recruited until the force exerted exceeds 90% of the maximum or when low-load high-velocity movements are performed (Sale 1991). Thus, an emphasis on less intensive exercises provides limited stimulus for structural and metabolic adaptations on the part of the fast contracting type II fibers. Moreover, it is only through regular practice of maximum-intensity exercises that the more advanced neural adaptations, such as motor unit synchronization, can take place (Semmler and Nordstrom 1998).

The factors that contribute to reduction in training intensity and volume with age in master athletes are unclear. However, it is conceivable that while many elite master athletes remain well motivated, they are forced to train at a lower level due to aging-related physiological changes. In other words, despite an overall reduction in training, aging athletes continue to train to the upper limit in terms of their physiological capacity. One factor contributing to a reduction in training level could be a longer recovery time. This may partially be related to greater exercise-induced damage and/or slower rate of repair from this damage (Fell and Williams 2008). Studies conducted on untrained people have suggested that with aging, high eccentric loads, in particular, can

cause greater cellular damage (Manfredi et al. 1991). Such age-related change can reduce the number of hard workout days per week that an older sprint athlete is able to undertake.

Many hormones (e.g., testosterone, growth hormone, IGF-I) are known to mediate acute and long-term responses and adaptations to training (Kraemer and Ratamess 2005). In this thesis only a few basic hormonal and nutritional aspects were examined as background factors. However, as far we know, our data are the first to demonstrate, that even in selected group of healthy athletes, aging leads to a decline in serum free testosterone level (T/SHBG), a factor that could play a role in trainability. This decline was mainly due to an increase in SHBG, since T showed only a trend towards a decline with age (Table 1). T concentrations were within normal reference values (ref. values for 51-70 yr: 8-29 nmol/L; for 70+yr: 6-25 nmol/L) and comparable with earlier study in older athletes of different sports (Suominen and Rahkila 1991). Yet, none of the sprint athletes were hypogonadal, defined as having a T/SHBG ratio below 0.153 nmol/nmol (Harman et al. 2001). This is in contrast, e.g., to the results of a large study (n=890, 23-91-yr-old) indicating that 9%, 34%, 68% and 91% of Caucasian men in their 50s, 60s, 70s, and 80s, respectively, could be classified as hypogonadal (Harman et al. 2001).

Nutrition could influence concentrations of hormones (Wang et al. 2005) and promote anabolism and recovery in training. A recent study found that serum T/SHBG ratio correlated significantly with daily intakes of energy, protein and fat in 38 healthy men aged 49-73 (Sallinen et al. 2007). In the present athletes (40-84 yr, n=50; Table 1), no clear age-related differences were seen in macro-nutrient intake. However, our data indicated a positive relationship between T/SHBG and energy intake ($p<0.05$), while the direct associations of T/SHBG with protein intake ($p\leq0.1$) and fat intake ($p<0.2$) were at trend level (not reported). Further, in line with some previous data (Key et al. 1990, Allen et al. 2002), saturated fatty acids correlated negatively with SHBG ($p<0.05$) and positively with T/SHBG ($p<0.01$), whether adjusted or unadjusted for age. Muscle adaptations may also be related to micronutrient intake. In particular, vitamin D deficiency, as observed in the present subjects, could affect negatively the IGF-I signaling pathway that play a major role in muscle hypertrophy (Grounds 2002). Given the limitations inherent with both food diaries [e.g. underreporting (Buzzard 1998)] and basal serum hormones [e.g. temporal variations in hormone levels (Rowe et al. 1974)], these results must be interpreted with caution.

Periodization and variation of training stimuli can promote recovery-adaptation. The results of the progressive periodized training program (VI) are promising, suggesting that even elite-level older sprinters can successfully undertake higher intensity training than they may have been accustomed to. A tempting explanation is that the sprint and strength exercises performed in the program (four times per week) provided an adequate overload but, owing to the systematic variation in intensities and type of work, it remained within the normal physiological range without overtraining or injuries. Taken together,

these data suggest that strength training (both heavy-resistance and high-power exercises) should become an essential component of the overall training of master sprint athletes to maximize long-term adaptations in neuromuscular and speed performance characteristics. Furthermore, the principles of the progression and periodization of training as well as nutrition are likely to contribute to the trainability of older athletes and need to be taken into account when planning training regimes.

6.6 Limitations and perspectives

Some limitations of the present thesis should be emphasized. The present study was based on a cross-sectional design. The methodological weakness of this approach is that differences between the groups may have been affected not only by aging *per se*, but also by genetic and constitutional factors. Longitudinal studies conducted over a span of several years with athletes who train at a high level would be ideal for the purpose of determining the causality between physiological changes and aging. It should be noted, however, that in this study the estimates of the average rate of decline in competitive performance were comparable with those obtained from cross-sectional and longitudinal approaches.

Muscle mass was estimated by ultrasound measurements. The accuracy of the ultrasound technique can be called into question because it is unable to distinguish between muscle and intramuscular fat. However, this may not significantly influence our results as the amount of intramuscular fat in the calf muscles of these master sprinters was minimal when examined by computer tomography. In addition, previous studies have shown that muscle thickness, as determined by ultrasonography, provides a good estimate of muscle volume (Miyatani et al. 2004) and muscle mass (Sanada et al. 2006). A cause of concern is that only the quadriceps femoris and gastrocnemius muscles were analyzed. This may have limited the prediction of muscle mass for sprint performance.

In the present thesis, the blood lactate response to competitive sprinting was the only measurement that provided an insight into anaerobic energy production ability with age. Since the lactate concentration in the blood represents the balance between the production of lactic acid in muscle and the appearance and removal of lactate from the blood according to the physiology of the individual, $[La]_{b\ peak}$ as a indicator of the rate of anaerobic glycolytic energy release must be interpreted cautiously. Moreover, the limited evidence available suggests that the age-related decline in the release of lactate from muscle to blood might underestimate the rate of anaerobic glycolysis and lactate production (Tzankoff and Norris 1979, Hepple et al. 2004).

In the training intervention (VI) an obvious limitation was the small sample size since only the twelve athletes who participated in the single fiber measurements were included. This limited the statistical power for detecting

differences in the changes between age groups. Further, it was not possible in this study to supervise the training, as the athletes arrived from all over the country. This was the result of an attempt to recruit sprinters representative of the very best national or international levels. On the other hand, one could argue that since master athletes typically do not take part in supervised training by coaches, the non-supervised training intervention provided a more realistic picture of what can be expected to be achieved in normal training conditions. However, considerable effort was expended on making the training intervention as successful as possible. The athletes were carefully instructed with both written and video materials about the program and exercises to be performed. Training logs were collected every four weeks during the field testing sessions (two visits to the athletes' home towns and one in Jyväskylä) and the athletes contacted the researchers during the experimental period by phone if any queries about training arose. According to the completed training logs and the fact that the athletes had a long training history, we have no reason to believe that they were unable to adhere to the relatively long, 20-week, program. Perhaps the most important factor contributing to the success of the training was that all the athletes were very motivated to participate in the study and improve their performance.

Some researchers have criticized studies on elite human performance on the grounds that the results are applicable to only a few persons. At the same time many aging experts stress that much can be learnt by examining physiological and performance adaptations in older athletes because these individuals optimize their physical well-being and thus reveal what is possible in human performance during aging. An important future goal is that we can design and deliver optimal training programs not only to athletes but also to ordinary aging people. Additional, well-controlled basic and applied studies on neuromuscular and performance characteristics in athletes and untrained people might help in reaching this goal. For example, much could be learnt by examining: (1) How do master athletes in various sports events differ from young athletes and untrained people with respect to specific neurological (motor unit number, firing frequency and recruitment), tendon (elasticity, collagens), and bone characteristics? (2) Does systematic training of master athletes have a protective effect against muscle sarcopenic processes at the molecular and nuclear levels (posttranslational modifications, oxidative changes, myonuclear domain size, myonuclear organization)? (3) Does training background influence the utilization of different energy systems and fatigue response during brief anaerobic exercise? And (4) Would it be possible to adapt the training principles and modify the exercises of athletes for used by previously untrained persons, and what would be the optimal and minimal amount of such exercise to elicit improvements in the neuromuscular system? Finally, given the evidence that loss of rapid force production and anaerobic performance becomes already evident by the fourth decade, studies should also focus on early middle-aged persons as a means of effectively promoting speed and strength qualities and mobility in the later years of life.

7 MAIN FINDINGS AND CONCLUSIONS

On the basis of the results and within the limitations of the study, the research questions presented in the study aims (page 39) are answered as follows:

(1a) With age athletic sprint performance (60 m, 100 m) declined in a curvilinear fashion (5-6%/decade) from the peak levels attained at age 20-35 until ~85 years of age (I, II). The decline with age in maximum speed was mainly related to a reduction in stride length and an increase in contact time, while stride frequency showed a minor decline and swing time remained unaffected (I, II). The age-related reductions in force production during running, defined here as average net resultant GRFs, seem to be primarily responsible for the changes in stride length, contact time, and, consequently, in maximum velocity with age (II). Furthermore, lower maximal leg and vertical stiffness in older runners may limit the ability to resist high impact loads and contribute to the increase in contact time in the braking phase.

(1b) Methodological study (III) indicated that the older runners have increased variability in horizontal braking and push-off GRFs, maximal vertical loading rate and aerial time of maximum-speed running, whereas the symmetry of the biomechanical measures was not affected by age. However, variability in the older runners was comparable to that in the younger runners in all the parameters that were characterized by good repeatability (CV<6%).

(2) There was an age-related decline in $[La]_{b\ peak}$ following races over 100-400 m, the decrease becoming more evident from age 70 (IV). $[La]_{b\ peak}$ correlated negatively and significantly with running times in all sprint events in the overall sample, and with the 400-m sprint when controlled for age

(3a) The athletes showed a typical age-associated reduction in fast-fiber size, a shift toward a slower MyHC isoform profile and a loss of leg muscle thickness (II, V). On the other hand, the data suggest that sprint training can maintain fiber size above normal levels and counteract the age-related

reduction in fascicle length (II, V). Furthermore, the qualitative aspects of muscle contraction, such as single fiber shortening velocity and specific force, remained unchanged with age in these athletes (V).

(3b) Older age was associated with a reduction in maximal (8-9%) and explosive (10-11%) muscle strength. The mechanism underlying the decline in maximal strength can be largely attributed to loss of muscle mass, whereas rapid strength may also relate to a reduction in the fast fiber area and rapid neural activation (V). Yet, in the sprint athletes the rate of the age-related decline in strength in rapid and in maximal muscle strength was similar, which may reflect the specificity of training and positive effect of sprint training on the maintenance of rapid muscle strength (II).

(4) Age was the strongest predictor of V_{max} (88%). When age was excluded from the model, CMJ height and muscle thickness (KE+PF) appeared in the model and together explained 80% of the variance in V_{max}. Muscle thickness was the best determinant of F_{brake} (26%) while CMJ explained most of the variance in F_{push} (34%). The finding that a large part of the total variability of the data remained unexplained suggests that force production during running is affected by complex interaction of neuromuscular and biomechanical factors.

(5a) The 20-week periodized sprint training program with increased emphasis on weight-training resulted in improvements in 60-m sprint time, V_{max}, stride length, rate of propulsive force development and leg stiffness in the older sprinters. Significant increases were also noted in maximal dynamic and isometric strength and in jumping exercises.

(5b) Training increased type II fiber size by 17-20%. However, single fiber specific force and shortening velocity remained unchanged, suggesting that the qualitative mechanism of contraction had no clear training effect. Changes in the neural activation of the agonists were minor, with increases noted only in iEMG of the vertical jump.

The present studies in high-performance master athletes provided a new insight into the effect of aging and training on speed ability. The results indicate that with systematic training, sprint performance and its physiological determinants are preserved at an extraordinary high level into old age. However, age group comparisons showed a progressive decline both in performance and in most of the neuromuscular characteristics studied. This may, in part, be associated with lack of strength training in the master sprinters. The information obtained from this study, in addition to contributing to present knowledge about the effects of aging *per se*, can be applied in the planning of training for athletes and non-athletes alike. Modified speed/explosive types of exercises combined with heavy resistance training can be recommended as part of overall physical training for middle-aged and older people to prevent fast fiber atrophy and loss of explosive strength both of which are critical changes in the aging process as they contribute substantially to mobility impairment, falls and fractures.

TIIVISTELMÄ (FINNISH SUMMARY)

Ikääntymisen ja harjoittelun vaikutukset nopeussuorituskykyyn, lihasten rakenteeseen ja voimantuotto-ominaisuuksiin urheilijoilla

Ikääntyvät urheilijat, jotka ovat harjoitelleet vuosikymmenien ajan, tarjoavat tutkijoille vanhenemismallin, jossa fysiologisten muutosten ajatellaan olevan yhteydessä itse biologiseen vanhenemiseen eikä tyypilliseen elimistön vajaakäyttöön ja ikääntymiseen liittyviin sairauksiin. Aikaisemmissa veteraaniurheilijatutkimuksissa on selvitetty ikääntymisen ja säännöllisen harjoittelun yhteyttä maksimivoiman ja erityisesti kestävyyssuorituskyvyn säilymiseen. Nopeusominaisuuksia, jotka myös vaikuttavat olennaisesti ikääntyvän henkilön liikuntakykyyn, on tutkittu erittäin vähän. Tämän tutkimuksen tavoitteena olikin selvittää iän myötä tapahtuvia nopeussuorituskyvyn ja siihen liittyvien anaerobisen energiantuoton, voimantuotto-ominaisuuksien ja lihasrakenteen muutoksia aktiivisesti harjoittelevilla miespikajuoksijoilla. Lisäksi tutkittiin, voidaanko nuorten urheilijoiden harjoittelumenetelmiä soveltamalla saada aikaan positiivisia muutoksia hermolihasjärjestelmässä ja edelleen parantaa nopeussuorituskykyä iäkkäillä urheilijoilla.

Tutkimus oli kolmivaiheinen. Ensimmäinen osavaihe toteutettiin veteraanien yleisurheilun Euroopanmestaruuskisojen yhteydessä. Siinä selvitettiin kansainvälisen tason veteraanipikajuoksijoiden (40-88 v, n=37) 100 metrin kilpailusuoritusten avulla ikään liittyviä eroja nopeussuorituskyvyssä analysoimalla videonauhoilta nopeutta ja kinemaattisia askelparametrejä (askeltiheys, askelpituus, kontaktiaika, lentoaika) juoksun eri vaiheissa (osajulkaisu I). Kilpailututkimuksessa arvioitiin myös finaaleihin selviytyneiden urheilijoiden (40-89 v, n=128) anaerobista energiantuottoa 100, 200 ja 400 metrin juoksumatkoilla veren laktaattimääritysten avulla (IV).

Toisessa tutkimusvaiheessa syvennettiin ikään liittyvien erojen tarkastelua juoksumekaniikan osalta ja tutkittiin juoksurataan upotetun voimalevyjonon (9.4 m) avulla maksimijuoksun reaktiovoimia, kinemaattisia askelparametrejä, tukijalan jäykkyyttä sekä muuttujien toistettavuutta ja symmetriaa jalkojen välillä kansallisen tason pikajuoksijoilla (17-84 v, n=93) (II, III). Juoksuanalyysien ohella tutkittiin alaraajojen lihaksiston maksimi- ja nopeusvoimaominaisuuksia, sähköistä aktiivisuutta (EMG) suorituskykytesteissä, lihasarkkitehtuuria, eri lihassolutyyppien suhteellista osuutta, poikkipinta-alaa ja supistumisominaisuuksia, myosiinin raskasketjukoostumusta (MyHC) sekä näiden keskinäisiä yhteyksiä (II, V).

Kolmannessa osavaiheessa toteutettiin harjoittelututkimus, jossa selvitettiin yhdistetyn voima- ja nopeusharjoittelun vaikutuksia edellä mainittuihin lajisuorituskykyä ja lihaksiston toimintaa ja rakennetta kuvaaviin muuttujiin iäkkäillä urheilijoilla (VI). Tutkittavat satunnaistettiin koe- ja kontrolliryhmiin. Koeryhmä toteutti jaksotetun, nousujohteisen 20 viikkoa kestävän harjoitteluohjelman, joka lajinomaisten juoksu- ja hyppyharjoitteiden lisäksi sisälsi kuntosalilla tehtäviä kesto-, hypertrofia-, maksimi- ja nopeusvoimaharjoitteita. La-

jispesifisen voimaharjoittelun tavoitteena oli vaikuttaa nopeisiin motorisiin yksiköihin ja nostaa maksimi- ja nopeusvoimatasoja vastaamaan paremmin pikajuoksun, ja erityisesti sen lyhyen kontaktivaiheen, voimantuotolle asettamia vaatimuksia. Kontrolliryhmä jatkoi aiempaa lajinomaista nopeusharjoittelua. Tutkimukseen liittyvät laboratoriomittaukset ja suorituskykytestit tehtiin harjoittelujakson alussa, puolivälissä ja lopussa.

Tulokset osoittivat, että pikajuoksun lajisuorituskyky (60-400 m) heikkenee keskimäärin 0.5-0.6 % vuodessa 35-40. ikävuoden jälkeen (I ,II, IV). Ikään liittyvät nopeuserot olivat samansuuruisia 100 metrin juoksun kiihdytyksen, vakionopeuden ja nopeuskestävyyden vaiheissa. Maksimaalisen juoksunopeuden hidastuminen iän lisääntyessä oli yhteydessä askelpituuden lyhenemiseen ja kontaktiajan pitenemiseen, sekä askelkontaktin jarrutus- ja työntövaiheen reaktiovoimien pienenemiseen. Askeltiheys ja heilahdusaika säilyivät lähes muuttumattomina. Tämä selittyy ainakin osaksi askelpituuden lyhenemisen kautta, sillä lyhyttä askelta pystyy tuottamaan nopeasti. Jousimallien avulla laskettu tukijalan jäykkyys (Δpystyvoima/Δ jalan pituus) ja pystyjäykkyys (Δpystyvoima/Δkehon painopisteen pystyheilahtelu) alenivat iän myötä. Jäykkyyden aleneminen oli vahvimmin yhteydessä kontaktin jarrutusvaiheen hidastumiseen.

Aikaisemman tutkimustiedon puuttumisen vuoksi tässä työssä (III) selvitettiin myös ikään liittyviä eroja maksimivauhtisen juoksun biomekaanisten muuttujien toistettavuudessa ja symmetriassa jalkojen välillä. Kinemaattisen parametrien ja reaktiovoimamuuttujien variaatio (CV) ja epäsymmetria olivat samansuuruisia nuorten ja iäkkäiden ryhmissä, poikkeuksena pystyvoiman maksimaalista voimantuottonopeutta (Δvoima/Δaika: kontaktin alkuvaihe) sekä vaakavoimia kuvaavat muuttujat, joissa variaatiokertoimet olivat korkeampia iäkkäillä. Hyvin toistettavia (CV: 1-6 %) ja harjoittelun seurantaan soveltuvia olivat kinemaattiset parametrit (askeltiheys ja -pituus, kontaktiaika, lentoaika) sekä maksimaalista ja keskimääräistä pysty- ja resultanttivoimaa kuvaavat muuttujat. Epäsymmetria muuttujissa oli kuitenkin yleisesti sitä suuruusluokkaa (2-10 %), että se tulisi ottaa huomioon analyyseissa.

100-400 metrin kilpailusuoritusten jälkeiset veren maksimaaliset laktaattipitoisuudet alenivat 40 vuoden iästä alkaen, mutta muutokset olivat selvempiä vasta 70. ikävuoden jälkeen (IV). Eri juoksumatkojen lopputulokset korreloivat laktaattivasteisiin siten, että paremmat juoksuajat olivat yhteydessä korkeampiin laktaattipitoisuuksiin. Nämä havainnot viittaavat siihen, että nopeussuorituskyvyn heikkeneminen iän myötä johtuu osittain kyvystä tuottaa energiaa anaerobisen glykolyysin kautta. Tuloksia täytyy kuitenkin tulkita tietyllä varauksella, koska vielä ei tiedetä, missä määrin esim. ikään liittyvät erot lihasmassassa, solujakaumassa ja juoksuajoissa heijastuvat laktaattivasteeseen. Jatkossa tulisikin tarkemmilla analysointimenetelmillä pyrkiä yksityiskohtaisemmin selvittämään ikääntymisen ja systemaattisen harjoittelun vaikutuksia glykolyysin ja muiden energiantuottoreittien tehokkuuteen.

Ultraäänitekniikalla mitatut etureiden ja pohjelihasten paksuudet ja ulomman reisilihaksen (m. vastus lateralis) lihaskimpun pennaatiokulma pienenivät iän lisääntyessä. Reiden tai pohkeen lihaskimppujen pituudessa ei kui-

tenkaan havaittu tyypillistä ikään liittyvää lyhenemistä. Vastus lateralis-lihaksen neulabiopsianäytteistä määritettyjen nopeiden (IIA, IIAB, IIB) lihassolujen keskimääräinen poikkipinta-ala ja pinta-alaosuus sekä nopeiden myosiinin raskasketjujen (MyHC IIa+IIx) suhteellinen osuus pienenivät iän lisääntyessä. Hitaiden lihassolujen koolla ja solutyyppien suhteellisella lukumäärällä ei ollut tilastollisesti merkitsevää yhteyttä ikään. Vaikka ikään liittyvä nopeiden II-tyypin lihassolujen koon pieneneminen ja siihen liittyvä MyHC II:n väheneminen oli samansuuntaista kuin aikaisemmissa tutkimuksissa harjoittelemattomilla henkilöillä, juoksijoiden nopeiden lihassolujen koot olivat kuitenkin huomattavasti suurempia. Esim. 70-vuotiaiden juoksijoiden II-tyypin solukoossa havaittiin olevan ”ikäetua” jopa 30 vuotta, vastaten nuorten keski-ikäisten miesten tasoa.

Biopsianäytteistä (m. vastus lateralis) eristettyjen yksittäisten hitaiden (MyHC I) ja nopeiden (MyHC IIa) lihassolujen absoluuttinen supistumisvoima oli pienempi iäkkäiden kuin nuorten ryhmässä. Sen sijaan solujen pinta-alaan suhteutettu voima ja supistumisnopeus olivat samaa suuruusluokkaa molemmissa ikäryhmissä poiketen aiemmista harjoittelemattomilla henkilöillä tehdyistä havainnoista, joiden mukaan lihassolun supistuminen hidastuu ja heikkenee vanhetessa. Nopeusurheilijoilta saadut uudet tutkimustulokset ovat merkittäviä, sillä ne viittaavat siihen, että säännöllisellä harjoittelulla voidaan ehkäistä ikääntymiseen ja inaktiivisuuteen liittyviä haitallisia muutoksia lihassolujen (kokoon suhteutetussa) supistumismekaniikassa.

Konsentrisen jalkakyykyn yhden toiston maksimivoima ja erityyppisten hyppysuoritusten (vertikaalihyppy ilman kevennystä ja esikevennyksellä, 5 s:n reaktiivisuushyppely, vauhditon 3-loikka) avulla mitattu nopeusvoima vähenivät asteittain nuorimmasta ikäryhmästä alkaen. Samoin alaraajojen lihasten isometrinen maksimivoima pieneni ja voimantuottonopeus hidastui iän myötä. Maksimivoiman lasku liittyi pääasiassa lihaksen koon pienenemiseen. Nopean voimantuoton heikkeneminen kytkeytyi lihasatrofian ohella myosiinin nopeiden raskasketjujen (MyHC IIa+IIx) osuuden vähenemiseen. Edelleen havaittiin, että isometrisessa jalkojen ojennuksessa voimantuoton hidastuminen iän mukana oli yhteydessä agonistilihasten aktivointinopeuden hidastumiseen. Veteraanijuoksijoiden voimantuotto-ominaisuudet olivat yleisesti ottaen huomattavasti paremmat kuin aikaisemmin tutkittujen harjoittelemattomien miesten. Vastoin aikaisempia havaintoja juoksijoilla ikäryhmien väliset erot olivat lähes samansuuruisia nopeaa (1.0-1.1 %/v) ja maksimaalista (0.8-0.9 %/v) voimantuottoa kuvaavissa parametreissä. Pikajuoksijoilla alaraajojen nopean voimantuoton hyvä säilyminen suhteessa maksimivoimaan selittynee harjoittelun spefisyydellä eli juoksu- ja hyppyharjoitteluun liittyvillä nopeusharjoitusärsykkeillä.

Koko tutkimusjoukossa maksimaalinen juoksunopeus, 60 metrin juoksuaika sekä jarrutus- ja työntövaiheen resultanttivoimat korreloivat vahvimmin jalkakyykkytulokseen, isometriseen maksimivoimaan, esikevennyshypyn nousukorkeuteen ja jalkalihasten paksuuteen. Juoksun voimantuotto oli lisäksi yhteydessä lihaksiston ominaisuuksiin siten, että mitä nopeampi oli maakontakti, sitä suuremmat olivat maksimivoima- ja lihaspaksuusarvot. Lihasvoima ja -

massa näyttäisivat vaikuttavan erityisesti tukijalan jäykkyyteen, mikä puolestaan heijastuu kontaktiaikaan. Juoksun maksiminopeuteen ja voimantuottoon vaikuttavien lihasominaisuuksien itsenäistä selitysosuutta tutkittiin askeltavalla regressioanalyysillä. Merkittävin yksilöiden välistä eroa maksiminopeudessa selittävä tekijä oli kronologinen ikä. Muista muuttujista vain isometrisella maksimivoimalla oli pieni itsenäinen selitysosuus. Kun ikä jätettiin mallista pois, nousivat esikevennyshyppy ja lihaspaksuus merkitseviksi. Juoksun jarrutusvaiheen voimantuottoa parhaiten selitti lihaspaksuus, kun taas esikevennyshyppy määräsi eniten työntövaiheen voimantuoton eroista. Ikä tai muut lihasominaisuudet eivät nousseet merkitseviksi selittäjiksi näissä malleissa.

Kahdenkymmenen viikon yhdistetty nopeus- ja voimaharjoittelu lisäsi koeryhmässä konsentrisen jalkakyykyn yhden toiston maksimivoimaa (27%), polven ojentajien (21%) ja koukistajien (40%) unilateraalista isometristä maksimivoimaa, sekä erityyppisillä hypyillä mitattua dynaamista nopeusvoimaa (4-29%) verrattuna lähtötilanteeseen. Lisäksi juoksun maksiminopeus (4%), 60 metrin juoksuaika (2%), askelpituus (3%), työntövaiheen resultanttivoima (8%) ja voimantuoton nopeus (12-14%) paranivat, jarrutus- ja työntövaiheen askelkontaktiajat (5-9%) lyhenivät ja tukijalan jäykkyys kasvoi (4-14%). Muutokset nopeissa hyppysuorituksissa ja useimmissa juoksusuorituskykyä kuvaavissa muuttujissa olivat koeryhmässä merkitsevästi suurempia kuin kontrolliryhmässä. Lihassolujen prosenttiosuus ja myosiinin raskasketjujen suhteellinen osuus eivät muuttuneet harjoittelun seurauksena, mutta nopeiden solujen poikkipinta-ala kasvoi koeryhmässä (20-40 %) ja muutos oli merkitsevä verrattuna kontrolliryhmässä tapahtuneeseen muutokseen. Staattisessa vertikaalihypyssä agonisteina toimivien polven ojentajalihasten sähköiset aktiivisuudet lisääntyivät koeryhmässä (9 %) muutoksen ollessa merkitsevästi suurempi kuin vertailuryhmässä. Interventiotutkimuksen mukaan yhdistämällä voimaharjoitusärsykkeitä lajinomaiseen pikajuoksuharjoitteluun voidaan edelleen tehostaa harjoitusvaikutusta voima- ja nopeusominaisuuksien, nopeitten lihassolujen koon sekä mahdollisesti myös lihaksen nopean aktivointikyvyn säilyttämiseksi. Täten näyttäisi tarkoituksenmukaiselta sisällyttää intensiivisiä voimaharjoitteita veteraaniurheilijoiden harjoitusohjelmiin, ottaen kuitenkin esim. turvallisuuteen liittyvät tekijät huomioon vanhimmilla henkilöillä.

Yhteenvetona voidaan todeta, että ikääntymisen myötä tapahtuvan nopeussuorituskyvyn heikkenemisen taustalla on useita juoksun biomekaniikkaan ja lihaksiston rakenteeseen, voimantuottoon ja energiametaboliaan liittyviä iän tuomia muutoksia. Keskeinen mekanismi näyttäisi olevan lihasmassan väheneminen, mikä ilmenee erityisesti nopeiden solujen koon pienenemisenä. Pikajuoksussa tämä voi vaikuttaa kykyyn vastustaa suuria eksentrisiä törmäysvoimia pidentäen jarrutusvaiheen kestoa sekä rajoittaa työntövaiheen voimantuottoa ja sitä kautta askelpituutta. Normaalin fysiologisen vanhenemisen lisäksi nopeusominaisuuksien heikkenemistä näyttäisi selittävän muutokset harjoittelutottumuksissa, erityisesti voimaharjoittelun väheneminen iän mukana. Huomionarvoista on kuitenkin, että veteraanipikajuoksijoilla lihaksistoon ja varsinkin sen nopeaan voimantuottoon liittyvät ominaisuudet ovat selvästi paremmalla tasolla kuin vastaavanikäisillä harjoittelemattomattomilla miehillä aikai-

semmissa tutkimuksissa. Täten spesifisellä harjoittelulla on hyvä mahdollisuus vaikuttaa hermolihasjärjestelmään siten, että nopeussuorituskyvyn heikkeneminen vanhetessa hidastuu. Useat tutkimukset ovat osoittaneet, että heikentynyt kyky nopeisiin koordinoituihin liikesuorituksiin on tärkeä liikkumis- ja tasapainovaikeuksien taustatekijä ja että pienilläkin harjoitusvaikutuksilla saattaa olla merkitystä näiden ominaisuuksien säilymisessä. Tämän tutkimuksen perusteella voidaan suositella, että keski-ikäisten ja sitä vanhempien henkilöiden liikuntaohjelmiin liitetään myös nopeusvoimatyyppisiä harjoitusärsykkeitä hermolihasjärjestelmän nopean voimantuottokyvyn ylläpitämiseksi ja kehittämiseksi.

REFERENCES

Aagaard P, Magnusson PS, Larsson B, Kjaer M, Krustrup P (2007). Mechanical muscle function, morphology, and fiber type in lifelong trained elderly. Med Sci Sports Exerc 39 (11): 1989-96.

Aagaard P, Simonsen EB, Andersen JL, Magnusson SP, Halkjaer-Kristensen J, Dyhre-Poulsen P (2000). Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. J Appl Physiol 89 (6): 2249-57.

Aagaard P, Simonsen EB, Magnusson SP, Larsson B, Dyhre-Poulsen P (1998). A new concept for isokinetic hamstring: quadriceps muscle strength ratio. Am J Sports Med 26 (2): 231-7.

Abe T, Fukashiro S, Harada Y, Kawamoto K (2001). Relationship between sprint performance and muscle fascicle length in female sprinters. J Physiol Anthropol Appl Human Sci 20 (2): 141-7.

Ae M, Ito A, Suzuki M (1992). The men's 100 meters. New Stud Athlet 7 (1): 47-52.

Ae M, Suzuki M, Miyanishi H, Okada M, Hirano T (1994). Analysis of racing patterns of the world's best top sprinters.Biomechanical research groups for track and fields. The biomechanical reports on 3rd IAAF World Championships in Athletics. Tokyo: Baseball Magazine Sha (Co.).

Akima H, Kano Y, Enomoto Y, Ishizu M, Okada M, Oishi Y, Katsuta S, Kuno S (2001). Muscle function in 164 men and women aged 20-84 yr. Med Sci Sports Exerc 33 (2): 220-6.

Allen D, Westerblad H (2004). Physiology. Lactic acid--the latest performance-enhancing drug. Science 305 (5687): 1112-3.

Allen NE, Appleby PN, Davey GK, Key TJ (2002). Lifestyle and nutritional determinants of bioavailable androgens and related hormones in British men. Cancer Causes Control 13 (4): 353-63.

Alway SE, Coggan AR, Sproul MS, Abduljalil AM, Robitaille PM (1996). Muscle torque in young and older untrained and endurance-trained men. J Gerontol A Biol Sci Med Sci 51 (3): B195-201.

Andersen JL (2001). Muscle fiber type characteristics of the runner. In Bangsbo J, Larsen HB (eds.), Running & Science. Copenhagen: Munksgaard, pp. 49-65.

Andersen JL (2003). Muscle fibre type adaptation in the elderly human muscle. Scand J Med Sci Sports 13 (1): 40-7.

Andersen JL, Aagaard P (2000). Myosin heavy chain IIX overshoot in human skeletal muscle. Muscle Nerve 23 (7): 1095-104.

Andersen JL, Klitgaard H, Saltin B (1994). Myosin heavy chain isoforms in single fibres from m. vastus lateralis of sprinters: influence of training. Acta Physiol Scand 151 (2): 135-42.

Andersen JL, Schjerling P, Saltin B (2000). Muscle, genes and athletic performance. Sci Am 283 (3): 48-55.

Andersen JL, Terzis G, Kryger A (1999). Increase in the degree of coexpression of myosin heavy chain isoforms in skeletal muscle fibers of the very old. Muscle Nerve 22 (4): 449-54.

Andersson H, Alnes LO (1996). Analys av sprintlöpare med olika kapacitet. Raport 4 - Utvecklingsprojekt. Farsta: Sveriges Olympiska Kommitte.

Aniansson A, Grimby G, Hedberg M (1992). Compensatory muscle fiber hypertrophy in elderly men. J Appl Physiol 73 (3): 812-6.

Aniansson A, Hedberg M, Henning GB, Grimby G (1986). Muscle morphology, enzymatic activity, and muscle strength in elderly men: a follow-up study. Muscle Nerve 9 (7): 585-91.

Anton MM, Spirduso WW, Tanaka H (2004). Age-related declines in anaerobic muscular performance: weightlifting and powerlifting. Med Sci Sports Exerc 36 (1): 143-7.

Arampatzis A, Bruggemann GP, Metzler V (1999). The effect of speed on leg stiffness and joint kinetics in human running. J Biomech 32 (12): 1349-53.

Asmussen E, Bonde-Petersen F (1974). Storage of elastic energy in skeletal muscles in man. Acta Physiol Scand 91 (3): 385-92.

Atwater A (1982). Kinematic analyses of sprinting. Track Field Q Rev 82: 12-6.

Balagopal P, Schimke JC, Ades P, Adey D, Nair KS (2001). Age effect on transcript levels and synthesis rate of muscle MHC and response to resistance exercise. Am J Physiol Endocrinol Metab 280 (2): E203-8.

Bamman MM, Ingram SG, Caruso JF, Greenisen MC (1997). Evaluation of surface electromyography during maximal voluntary contraction. J Strength Cond Res 11 (2): 68-72.

Barany M (1967). ATPase activity of myosin correlated with speed of muscle shortening. J Gen Physiol 50 (6): Suppl:197-218.

Bartlett R, Wheat J, Robins M (2007). Is movement variability important for sports biomechanists? Sports Biomech 6 (2): 224-43.

Bassey EJ, Fiatarone MA, O'Neill EF, Kelly M, Evans WJ, Lipsitz LA (1992). Leg extensor power and functional performance in very old men and women. Clin Sci (Lond) 82 (3): 321-7.

Bean JF, Leveille SG, Kiely DK, Bandinelli S, Guralnik JM, Ferrucci L (2003). A comparison of leg power and leg strength within the InCHIANTI study: which influences mobility more? J Gerontol A Biol Sci Med Sci 58 (8): 728-33.

Belli A, Lacour JR, Komi PV, Candau R, Denis C (1995). Mechanical step variability during treadmill running. Eur J Appl Physiol 70 (6): 510-7.

Bemben MG, Massey BH, Bemben DA, Misner JE, Boileau RA (1991). Isometric muscle force production as a function of age in healthy 20- to 74-yr-old men. Med Sci Sports Exerc 23 (11): 1302-10.

Benelli P, Ditroilo M, Forte R, De Vito G, Stocchi V (2007). Assessment of post-competition peak blood lactate in male and female master swimmers aged 40-79 years and its relationship with swimming performance. Eur J Appl Physiol 99 (6): 685-93.

Bergström J, Harris RC, Hultman E, Nordesjö LO (1971). Energy rich phosphagens in dynamic and static work. In Pernow B, Saltin B (eds.), Muscle metabolism during exercise. New York: Plenum Press, pp. 87-95.

Biewener AA, Konieczynski DD, Baudinette RV (1998). In vivo muscle force-length behavior during steady-speed hopping in tammar wallabies. J Exp Biol 201 (Pt 11): 1681-94.

Blazevich AJ, Jenkins DG (2002). Effect of the movement speed of resistance training exercises on sprint and strength performance in concurrently training elite junior sprinters. J Sports Sci 20 (12): 981-90.

Boobis LH, Williams C, Wootton S (1982). Human muscle metabolism during brief maximal exercise. J Physiol 338: 21P-22P.

Bortz WMt, Bortz WM, 2nd (1996). How fast do we age? Exercise performance over time as a biomarker. J Gerontol A Biol Sci Med Sci 51 (5): M223-5.

Bosco C, Komi PV (1980). Influence of aging on the mechanical behavior of leg extensor muscles. Eur J Appl Physiol Occup Physiol 45 (2-3): 209-19.

Bottinelli R, Canepari M, Pellegrino MA, Reggiani C (1996). Force-velocity properties of human skeletal muscle fibres: myosin heavy chain isoform and temperature dependence. J Physiol 495 (Pt 2) 573-86.

Bradshaw EJ, Maulder PS, Keogh JW (2007). Biological movement variability during the sprint start: performance enhancement or hindrance? Sports Biomech 6 (3): 246-60.

Bret C, Rahmani A, Dufour AB, Messonnier L, Lacour JR (2002). Leg strength and stiffness as ability factors in 100 m sprint running. J Sports Med Phys Fitness 42 (3): 274-81.

Brooke MH, Kaiser KK (1970). Muscle fiber types: how many and what kind? Arch Neurol 23 (4): 369-79.

Brooks GA (1987). Amino acid and protein metabolism during exercise and recovery. Med Sci Sports Exerc 19 (5 Suppl): S150-6.

Brown MB, Coggan A (1990). Is muscle wasting inevitable with aging? Med Sci Sports Exerc 22 (2): S73.

Bruce SA, Newton D, Woledge RC (1989). Effect of age on voluntary force and cross-sectional area of human adductor pollicis muscle. Q J Exp Physiol 74 (3): 359-62.

Bruggemann G-P, Glad B (1990). Time analysis of the sprint events. In Bruggemann G-P, Glad B (eds.), Scientific research project at the games of the XXIVth Olympiad-Seoul 1988, Final report. International Athletic Foundation/International Amateur Athletic Federation, pp. 11-89.

Brughelli M, Cronin J (2008). Influence of running velocity on vertical, leg and joint stiffness : modelling and recommendations for future research. Sports Med 38 (8): 647-57.

Butler RJ, Crowell HP, 3rd, Davis IM (2003). Lower extremity stiffness: implications for performance and injury. Clin Biomech (Bristol, Avon) 18 (6): 511-7.

Buzzard M (1998). 24-hour dietary recall and food record methods. In Willett W (ed.), Nutritional epidemiology. New York: Oxford University Press, pp. 51-73.

Cadefau J, Casademont J, Grau JM, Fernandez J, Balaguer A, Vernet M, Cusso R, Urbano-Marquez A (1990). Biochemical and histochemical adaptation to sprint training in young athletes. Acta Physiol Scand 140 (3): 341-51.

Caiozzo VJ, Haddad F (1996). Thyroid hormone: modulation of muscle structure, function, and adaptive responses to mechanical loading. Exerc Sport Sci Rev 24: 321-61.

Campbell CB, Marsh DR, Spriet LL (1991). Anaerobic energy provision in aged skeletal muscle during tetanic stimulation. J Appl Physiol 70 (4): 1787-95.

Campbell MJ, McComas AJ, Petito F (1973). Physiological changes in ageing muscles. J Neurol Neurosurg Psychiatry 36 (2): 174-82.

Campbell WW, Geik RA (2004). Nutritional considerations for the older athlete. Nutrition 20 (7-8): 603-8.

Cavagna GA, Komarek L, Mazzoleni S (1971). The mechanics of sprint running. J Physiol 217 (3): 709-21.

Chapman AE, Caldwell GE (1983). Factors determining changes in lower limb energy during swing in treadmill running. J Biomech 16 (1): 69-77.

Cheetham ME, Boobis LH, Brooks S, Williams C (1986). Human muscle metabolism during sprint running. J Appl Physiol 61 (1): 54-60.

Chelly SM, Denis C (2001). Leg power and hopping stiffness: relationship with sprint running performance. Med Sci Sports Exerc 33 (2): 326-33.

Clarkson PM, Kroll W, Melchionda AM (1981). Age, isometric strength, rate of tension development and fiber type composition. J Gerontol 36 (6): 648-53.

Coggan AR, Abduljalil AM, Swanson SC, Earle MS, Farris JW, Mendenhall LA, Robitaille PM (1993). Muscle metabolism during exercise in young and older untrained and endurance-trained men. J Appl Physiol 75 (5): 2125-33.

Connelly DM, Rice CL, Roos MR, Vandervoort AA (1999). Motor unit firing rates and contractile properties in tibialis anterior of young and old men. J Appl Physiol 87 (2): 843-52.

Conzelmann A (1997). The plasticity of courses of physical performance in the second part of life. In Huber G (ed.), Healthy Aging, Activity, and Sports (Proceedings, PAAS IV). Health Promotion Publications, Gamburg, Germany, pp. 435-445.

D'Antona G, Pellegrino MA, Adami R, Rossi R, Carlizzi CN, Canepari M, Saltin B, Bottinelli R (2003). The effect of ageing and immobilization on structure and function of human skeletal muscle fibres. J Physiol 552 (Pt 2): 499-511.

D'Antona G, Pellegrino MA, Carlizzi CN, Bottinelli R (2007). Deterioration of contractile properties of muscle fibres in elderly subjects is modulated by the level of physical activity. Eur J Appl Physiol 100 (5): 603-11.

Davis JA, Rozenek R, Decicco DM, Carizzi MT, Pham PH (2007). Effect of plasma volume loss during graded exercise testing on blood lactate concentration. J Physiol Sci 57 (2): 95-9.

Delbono O, O'Rourke KS, Ettinger WH (1995). Excitation-calcium release uncoupling in aged single human skeletal muscle fibers. J Membr Biol 148 (3): 211-22.

Delecluse C (1997). Influence of strength training on sprint running performance. Current findings and implications for training. Sports Med 24 (3): 147-56.

Deschenes MR, Kraemer WJ (2002). Performance and physiologic adaptations to resistance training. Am J Phys Med Rehabil 81 (11 Suppl): S3-16.

Dietz V, Schmidtbleicher D, Noth J (1979). Neuronal mechanisms of human locomotion. Journal of Neurophysiology 42 (5): 1212-22.

Dinenno FA, Seals DR, DeSouza CA, Tanaka H (2001). Age-related decreases in basal limb blood flow in humans: time course, determinants and habitual exercise effects. J Physiol 531 (Pt 2): 573-9.

Doherty TJ (2003). Invited review: Aging and sarcopenia. J Appl Physiol 95 (4): 1717-27.

Doherty TJ, Vandervoort AA, Brown WF (1993). Effects of ageing on the motor unit: a brief review. Can J Appl Physiol 18 (4): 331-58.

Dreyer HC, Schroeder ET, Hawkins SA, Marcell TJ, Tarpenning KM, Vallejo AF, Jensky NE, Shaibi GQ, Spears S, Yamada R, Wiswell RA (2006). Chronic exercise and skeletal muscle power in older men. Appl Physiol Nutr Metab 31 (3): 190-5.

Duclay J, Martin A, Robbe A, Pousson M (2008). Spinal reflex plasticity during maximal dynamic contractions after eccentric training. Med Sci Sports Exerc 40 (4): 722-34.

Ennion S, Sant'ana Pereira J, Sargeant AJ, Young A, Goldspink G (1995). Characterization of human skeletal muscle fibres according to the myosin heavy chains they express. J Muscle Res Cell Motil 16 (1): 35-43.

Eriksen HK, Kristiansen JR, Langangen O, Wehus IK (2008). Velocity dispersions in a cluster of stars: how fast could Usain Bolt have run ? arXiv:0809.0209v2 [physics.pop-ph].

Essen-Gustavsson B, Borges O (1986). Histochemical and metabolic characteristics of human skeletal muscle in relation to age. Acta Physiol Scand 126 (1): 107-14.

Essen B, Haggmark T (1975). Lactate concentration in type I and II muscle fibres during muscular contraction in man. Acta Physiol Scand 95 (3): 344-6.

Farley CT, Gonzalez O (1996). Leg stiffness and stride frequency in human running. J Biomech 29 (2): 181-6.

Faulkner JA, Davis CS, Mendias CL, Brooks SV (2008). The aging of elite male athletes: age-related changes in performance and skeletal muscle structure and function. Clin J Sport Med 18 (6): 501-7.

Fell J, Williams D (2008). The effect of aging on skeletal-muscle recovery from exercise: possible implications for aging athletes. J Aging Phys Act 16 (1): 97-115.

Ferro A, Rivera A, Pagola I (2001). Biomechanical analysis of the 7th World Championships in Athletics Seville 1999. New Stud Athlet 16: 25-60.

Fitts RH (2004). Mechanisms of muscular fatigue. In Poortmans JR (ed.), Principles of exercise biochemistry. Brussels: Karger, pp. 279-300.

Fitts RH, Widrick JJ (1996). Muscle mechanics: adaptations with exercise-training. Exerc Sport Sci Rev 24: 427-73.

Frontera WR, Hughes VA, Fielding RA, Fiatarone MA, Evans WJ, Roubenoff R (2000a). Aging of skeletal muscle: a 12-yr longitudinal study. J Appl Physiol 88 (4): 1321-6.

Frontera WR, Hughes VA, Krivickas LS, Kim SK, Foldvari M, Roubenoff R (2003). Strength training in older women: early and late changes in whole muscle and single cells. Muscle Nerve 28 (5): 601-8.

Frontera WR, Hughes VA, Lutz KJ, Evans WJ (1991). A cross-sectional study of muscle strength and mass in 45- to 78-yr-old men and women. J Appl Physiol 71 (2): 644-50.

Frontera WR, Reid KF, Phillips EM, Krivickas LS, Hughes VA, Roubenoff R, Fielding RA (2008). Muscle fiber size and function in elderly humans: a longitudinal study. J Appl Physiol 105 (2): 637-42.

Frontera WR, Suh D, Krivickas LS, Hughes VA, Goldstein R, Roubenoff R (2000b). Skeletal muscle fiber quality in older men and women. Am J Physiol Cell Physiol 279 (3): C611-8.

Fujitsuka N, Yamamoto T, Ohkuwa T, Saito M, Miyamura M (1982). Peak blood lactate after short periods of maximal treadmill running. Eur J Appl Physiol Occup Physiol 48 (3): 289-96.

Gabaldon AM, Nelson FE, Roberts TJ (2008). Relative shortening velocity in locomotor muscles: turkey ankle extensors operate at low V/Vmax. Am J Physiol Regul Integr Comp Physiol 294 R200-R10.

Gaitanos GC, Williams C, Boobis LH, Brooks S (1993). Human muscle metabolism during intermittent maximal exercise. J Appl Physiol 75 (2): 712-9.

Gajer B, Thepaut-Mathieu C, Lehenaff D (1999). Evolution of stride and amplitude during course of the 100 m event in athletics. New Stud Athlet 14 (1): 43-50.

Galloway MT, Jokl P (1996). Age and sports participation: The effect of aging on muscle function and athletic performance. Sports Med Arthroscopy Rev 4: 221-34.

Gastin PB (2001). Energy system interaction and relative contribution during maximal exercise. Sports Med 31 (10): 725-41.

Giakas G, Baltzopoulos V (1997). Time and frequency domain analysis of ground reaction forces during walking: an investigation of variability and symmetry. Gait Posture 5 189-97.

Godt RE, Maughan DW (1981). Influence of osmotic compression on calcium activation and tension in skinned muscle fibers of the rabbit. Pflugers Arch 391 (4): 334-7.

Gollhofer A, Schmidtbleicher D, Dietz V (1984). Regulation of muscle stiffness in human locomotion. Int J Sports Med 5 (1): 19-22.

Goodpaster BH, Park SW, Harris TB, Kritchevsky SB, Nevitt M, Schwartz AV, Simonsick EM, Tylavsky FA, Visser M, Newman AB (2006). The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. J Gerontol A Biol Sci Med Sci 61 (10): 1059-64.

Goodwin PC, Koorts K, Mack R, Mai S, Morrissey MC, Hooper DM (1999). Reliability of leg muscle electromyography in vertical jumping. Eur J Appl Physiol Occup Physiol 79 (4): 374-8.

Grassi B, Cerretelli P, Narici MV, Marconi C (1991). Peak anaerobic power in master athletes. Eur J Appl Physiol Occup Physiol 62 (6): 394-9.

Grimby G, Aniansson A, Zetterberg C, Saltin B (1984). Is there a change in relative muscle fibre composition with age? Clin Physiol 4 (2): 189-94.

Grimby G, Danneskiold-Samsoe B, Hvid K, Saltin B (1982). Morphology and enzymatic capacity in arm and leg muscles in 78-81 year old men and women. Acta Physiol Scand 115 (1): 125-34.

Grounds MD (2002). Reasons for the degeneration of ageing skeletal muscle: a central role for IGF-1 signalling. Biogerontology 3 (1-2): 19-24.

Häkkinen K, Alen M, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Mälkiä E, Kraemer WJ, Newton RU (1998a). Muscle CSA, force production, and activation of leg extensors during isometric and dynamic actions in middle-aged and elderly men and women. JAPA 8: 232-47.

Häkkinen K, Häkkinen A (1991). Muscle cross-sectional area, force production and relaxation characteristics in women at different ages. Eur J Appl Physiol Occup Physiol 62 (6): 410-4.

Häkkinen K, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Mälkiä E, Kraemer WJ, Newton RU, Alen M (1998b). Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. J Appl Physiol 84 (4): 1341-9.

Häkkinen K, Kallinen M, Linnamo V, Pastinen UM, Newton RU, Kraemer WJ (1996). Neuromuscular adaptations during bilateral versus unilateral strength training in middle-aged and elderly men and women. Acta Physiol Scand 158 (1): 77-88.

Häkkinen K, Komi PV (1983). Electromyographic changes during strength training and detraining. Med Sci Sports Exerc 15 (6): 455-60.

Häkkinen K, Komi PV, Alen M (1985). Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. Acta Physiol Scand 125 (4): 587-600.

Häkkinen K, Kraemer WJ, Pakarinen A, Triplett-McBride T, McBride JM, Häkkinen A, Alen M, McGuigan MR, Bronks R, Newton RU (2002). Effects of heavy resistance/power training on maximal strength, muscle morphology, and hormonal response patterns in 60-75-year-old men and women. Can J Appl Physiol 27 (3): 213-31.

Hameed M, Orrell RW, Cobbold M, Goldspink G, Harridge SD (2003). Expression of IGF-I splice variants in young and old human skeletal muscle after high resistance exercise. J Physiol 547 (Pt 1): 247-54.

Hamill J, McNiven SL (1990). Reliability of selected ground reaction force parameters during walking. Hum Mov Sci 9: 117-31.

Hamilton N (1993). Changes in sprint stride kinematics with age in master's athletes. J Appl Biomech 9: 15-26.

Harman SM, Metter EJ, Tobin JD, Pearson J, Blackman MR (2001). Longitudinal effects of aging on serum total and free testosterone levels in healthy men. Baltimore Longitudinal Study of Aging. J Clin Endocrinol Metab 86 (2): 724-31.

Harridge S, Magnusson G, Saltin B (1997). Life-long endurance-trained elderly men have high aerobic power, but have similar muscle strength to non-active elderly men. Aging (Milano) 9 (1-2): 80-7.

Harridge SD, Bottinelli R, Canepari M, Pellegrino M, Reggiani C, Esbjornsson M, Balsom PD, Saltin B (1998). Sprint training, in vitro and in vivo muscle function, and myosin heavy chain expression. J Appl Physiol 84 (2): 442-9.

Harridge SD, Bottinelli R, Canepari M, Pellegrino MA, Reggiani C, Esbjornsson M, Saltin B (1996). Whole-muscle and single-fibre contractile properties and myosin heavy chain isoforms in humans. Pflugers Arch 432 (5): 913-20.

Harridge SD, Kryger A, Stensgaard A (1999). Knee extensor strength, activation, and size in very elderly people following strength training. Muscle Nerve 22 (7): 831-9.

Harridge SD, White MJ, Carrington CA, Goodman M, Cummins P (1995). Electrically evoked torque-velocity characteristics and isomyosin composition of the triceps surae in young and elderly men. Acta Physiol Scand 154 (4): 469-77.

Harris GR, Stone MH, O'Bryant HS, Proulx CM, Johnson RL (2000). Short-term performance effects of high power, high force, or combined weight-training methods. J Strength Cond Res 14 (1): 14-20.

Hautier CA, Wouassi D, Arsac LM, Bitanga E, Thiriet P, Lacour JR (1994). Relationships between postcompetition blood lactate concentration and average running velocity over 100-m and 200-m races. Eur J Appl Physiol Occup Physiol 68 (6): 508-13.

Hawkins SA, Wiswell RA, Marcell TJ (2003). Exercise and the master athlete--a model of successful aging? J Gerontol A Biol Sci Med Sci 58 (11): 1009-11.

He JP, Kram R, McMahon TA (1991). Mechanics of running under simulated low gravity. J Appl Physiol 71 (3): 863-70.

Heinonen A, Sievanen H, Viitasalo J, Pasanen M, Oja P, Vuori I (1994). Reproducibility of computer measurement of maximal isometric strength and electromyography in sedentary middle-aged women. Eur J Appl Physiol Occup Physiol 68 (4): 310-4.

Hepple RT, Hagen JL, Krause DJ, Baker DJ (2004). Skeletal muscle aging in F344BN F1-hybrid rats: II. Improved contractile economy in senescence helps compensate for reduced ATP-generating capacity. J Gerontol A Biol Sci Med Sci 59 (11): 1111-9.

Herzog W, Nigg BM, Read LJ, Olsson E (1989). Asymmetries in ground reaction force patterns in normal human gait. Med Sci Sports Exerc 21 (1): 110-4.

Hill DW (1999). Energy system contributions in middle-distance running events. J Sports Sci 17 (6): 477-83.

Hirvonen J (1984). Physiology of anaerobic performance. University of Jyväskylä, Finland. (unpublished report).

Hirvonen J, Nummela A, Rusko H, Rehunen S, Harkonen M (1992). Fatigue and changes of ATP, creatine phosphate, and lactate during the 400-m sprint. Can J Sport Sci 17 (2): 141-4.

Hirvonen J, Rehunen S, Rusko H, Harkonen M (1987). Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. Eur J Appl Physiol Occup Physiol 56 (3): 253-9.

Hoffren M, Ishikawa M, Komi PV (2007). Age-related neuromuscular function during drop jumps. J Appl Physiol 103 (4): 1276-83.

Holloszy JO, Chen M, Cartee GD, Young JC (1991). Skeletal muscle atrophy in old rats: differential changes in the three fiber types. Mech Ageing Dev 60 (2): 199-213.

Höök P, Sriramoju V, Larsson L (2001). Effects of aging on actin sliding speed on myosin from single skeletal muscle cells of mice, rats, and humans. Am J Physiol Cell Physiol 280 (4): C782-8.

Hortobagyi T, Hill JP, Houmard JA, Fraser DD, Lambert NJ, Israel RG (1996). Adaptive responses to muscle lengthening and shortening in humans. J Appl Physiol 80 (3): 765-72.

Hortobagyi T, Zheng D, Weidner M, Lambert NJ, Westbrook S, Houmard JA (1995). The influence of aging on muscle strength and muscle fiber characteristics with special reference to eccentric strength. J Gerontol A Biol Sci Med Sci 50 (6): B399-406.

Hughes VA, Frontera WR, Wood M, Evans WJ, Dallal GE, Roubenoff R, Fiatarone Singh MA (2001). Longitudinal muscle strength changes in older adults: influence of muscle mass, physical activity, and health. J Gerontol A Biol Sci Med Sci 56 (5): B209-17.

Hultman E, Sjöholm H (1983). Substrate availability. In Knuttgen HG, Vogel JA, Poortmans JR (eds.), Biochemistry of exercise. Champaign, Ill: Human Kinetics, pp. 63-75.

Hunter GR, Newcomer BR, Weinsier RL, Karapondo DL, Larson-Meyer DE, Joanisse DR, Bamman MM (2002). Age is independently related to muscle metabolic capacity in premenopausal women. J Appl Physiol 93 (1): 70-6.

Hunter JP, Marshall RN, McNair P (2004). Reliability of biomechanical variables of sprint running. Med Sci Sports Exerc 36 (5): 850-61.

Hvid L, Suetta C, Aagaard P, Kjaer M, Ortenblad N (2009). Changes in single fibre contractility with immobilization in old and young. Med Sci Sports Exerc 41 (5 Suppl): s97.

Ito A 1993. Leg movement analysis of gold and silver medalists in men's 100m at the III world championships in athletics. XIVth ISB Congress Paris, France. pp. 624-625.

Ito A, Fukuda K, Kijima K (2008). Mid-phase movements of Tyson Gay and Asafa Powell in the 100 metres at the 2007 World Championships in Athletics. New Stud Athlet 23 (2): 39-43.

Iwaoka K, Funato K, Takatoh S, Mutoh Y, Miyashita M (1989). Characteristics of leg extensor muscle in a world champion masters jumper. A case study. J Sports Med Phys Fitness 29 (4): 394-7.

Izquierdo M, Ibanez J, Gorostiaga E, Garrues M, Zuniga A, Anton A, Larrion JL, Hakkinen K (1999). Maximal strength and power characteristics in isometric and dynamic actions of the upper and lower extremities in middle-aged and older men. Acta Physiol Scand 167 (1): 57-68.

Jakobsson F, Borg K, Edstrom L (1990). Fibre-type composition, structure and cytoskeletal protein location of fibres in anterior tibial muscle. Comparison between young adults and physically active aged humans. Acta Neuropathol (Berl) 80 (5): 459-68.

Jansson E (1994). Age-related fiber type changes in human skeletal muscle. Biochemistry of Exercise IX. In Maughan RJ, Shireffs SM (eds.), Champaign, Ill; Human Kinetics, pp. 297-307

Jensen-Urstad M, Svedenhag J, Sahlin K (1994). Effect of muscle mass on lactate formation during exercise in humans. Eur J Appl Physiol Occup Physiol 69 (3): 189-95.

Joch W (ed.) 1992. Rahmentrainingsplan für das Aufbautraining Sprint. Aachen, Germany: Meyer & Meyer Verlag.

Jones PP, Davy KP, DeSouza CA, van Pelt RE, Seals DR (1997). Absence of age-related decline in total blood volume in physically active females. Am J Physiol 272 (6 Pt 2): H2534-40.

Jönhagen S, Nemeth G, Eriksson E (1994). Hamstring injuries in sprinters. The role of concentric and eccentric hamstring muscle strength and flexibility. Am J Sports Med 22 (2): 262-6.

Jubrias SA, Odderson IR, Esselman PC, Conley KE (1997). Decline in isokinetic force with age: muscle cross-sectional area and specific force. Pflugers Arch 434 (3): 246-53.

Juel C (1988). Intracellular pH recovery and lactate efflux in mouse soleus muscles stimulated in vitro: the involvement of sodium/proton exchange and a lactate carrier. Acta Physiol Scand 132 (3): 363-71.

Juel C, Honig A, Pilegaard H (1991). Muscle lactate transport studied in sarcolemmal giant vesicles: dependence on fibre type and age. Acta Physiol Scand 143 (4): 361-5.

Kaczor JJ, Ziolkowski W, Antosiewicz J, Hac S, Tarnopolsky MA, Popinigis J (2006). The effect of aging on anaerobic and aerobic enzyme activities in human skeletal muscle. J Gerontol A Biol Sci Med Sci 61 (4): 339-44.

Kallinen M, Alen M (1994). Sports-related injuries in elderly men still active in sports. Br J Sports Med 28 (1): 52-5.

Kamen G, Knight CA (2004). Training-related adaptations in motor unit discharge rate in young and older adults. J Gerontol A Biol Sci Med Sci 59 (12): 1334-8.

Kamen G, Roy A (2000). Motor unit synchronization in young and elderly adults. Eur J Appl Physiol 81 (5): 403-10.

Kamen G, Sison SV, Du CC, Patten C (1995). Motor unit discharge behavior in older adults during maximal-effort contractions. J Appl Physiol 79 (6): 1908-13.

Karamanidis K, Arampatzis A (2006). Mechanical and morphological properties of human quadriceps femoris and triceps surae muscle-tendon unit in relation to aging and running. J Biomech 39 (3): 406-17.

Kent-Braun JA (2009). Skeletal muscle fatigue in old age: whose advantage ? Exerc Sport Sci Rev 37 (1): 3-9.

Kent-Braun JA, Ng AV (1999). Specific strength and voluntary muscle activation in young and elderly women and men. J Appl Physiol 87 (1): 22-9.

Kent-Braun JA, Ng AV, Young K (2000). Skeletal muscle contractile and noncontractile components in young and older women and men. J Appl Physiol 88 (2): 662-8.

Kersting UG (1999). Biomechanical analysis of the sprinting events. In Bruggemann GP, Koszewski D, Muller H (eds.), Biomechanical research project Athens 1997, Final report. Oxford: Meyer & Meyer, pp. 12-61.

Key TJ, Roe L, Thorogood M, Moore JW, Clark GM, Wang DY (1990). Testosterone, sex hormone-binding globulin, calculated free testosterone, and oestradiol in male vegans and omnivores. Br J Nutr 64 (1): 111-9.

Kindermann W, Keul J (1977). Anaerobe Energiebereitstellung im Hochleistungssport [Anaerobic capacity in elite sports]. Schondorf: Verlag Karl Hofmann.

Klass M, Baudry S, Duchateau J (2005). Aging does not affect voluntary activation of the ankle dorsiflexors during isometric, concentric, and eccentric contractions. J Appl Physiol 99 (1): 31-8.

Klass M, Baudry S, Duchateau J (2008). Age-related decline in rate of torque development is accompanied by lower maximal motor unit discharge frequency during fast contractions. J Appl Physiol 104 (3): 739-46.

Klitgaard H, Ausoni S, Damiani E (1989a). Sarcoplasmic reticulum of human skeletal muscle: age-related changes and effect of training. Acta Physiol Scand 137 (1): 23-31.

Klitgaard H, Brunet A, Maton B, Lamaziere C, Lesty C, Monod H (1989b). Morphological and biochemical changes in old rat muscles: effect of increased use. J Appl Physiol 67 (4): 1409-17.

Klitgaard H, Mantoni M, Schiaffino S, Ausoni S, Gorza L, Laurent-Winter C, Schnohr P, Saltin B (1990a). Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. Acta Physiol Scand 140 (1): 41-54.

Klitgaard H, Zhou M, Schiaffino S, Betto R, Salviati G, Saltin B (1990b). Ageing alters the myosin heavy chain composition of single fibres from human skeletal muscle. Acta Physiol Scand 140 (1): 55-62.

Kobayashi K, Tsuchie H, Matsuo A, Fukunaga T, Kawakami Y (2008). Changes in sprint performance and kinetics during acceleration phase of running of a world record holder. The 13th Annual Congress of the European College of Sport Science; July 9-12. Estoril (Portugal), p. 591.

Kraemer WJ, Häkkinen K (eds.) 2002. Strength Training for Sport. Oxford, England: Blackwell Science.

Kraemer WJ, Ratamess NA (2005). Hormonal responses and adaptations to resistance exercise and training. Sports Med 35 (4): 339-61.

Krivickas LS, Suh D, Wilkins J, Hughes VA, Roubenoff R, Frontera WR (2001). Age- and gender-related differences in maximum shortening velocity of skeletal muscle fibers. Am J Phys Med Rehabil 80 (6): 447-55; quiz 56-7.

Kubo K, Kanehisa H, Azuma K, Ishizu M, Kuno SY, Okada M, Fukunaga T (2003a). Muscle architectural characteristics in women aged 20-79 years. Med Sci Sports Exerc 35 (1): 39-44.

Kubo K, Kanehisa H, Azuma K, Ishizu M, Kuno SY, Okada M, Fukunaga T (2003b). Muscle architectural characteristics in young and elderly men and women. Int J Sports Med 24 (2): 125-30.

Kubo K, Morimoto M, Komuro T, Tsunoda N, Kanehisa H, Fukunaga T (2007). Age-related differences in the properties of the plantar flexor muscles and tendons. Med Sci Sports Exerc 39 (3): 541-7.

Kumagai K, Abe T, Brechue WF, Ryushi T, Takano S, Mizuno M (2000). Sprint performance is related to muscle fascicle length in male 100-m sprinters. J Appl Physiol 88 (3): 811-6.

Lacour JR, Bouvat E, Barthelemy JC (1990). Post-competition blood lactate concentrations as indicators of anaerobic energy expenditure during 400-m and 800-m races. Eur J Appl Physiol Occup Physiol 61 (3-4): 172-6.

Lange KH, Andersen JL, Beyer N, Isaksson F, Larsson B, Rasmussen MH, Juul A, Bulow J, Kjaer M (2002). GH administration changes myosin heavy chain isoforms in skeletal muscle but does not augment muscle strength or hypertrophy, either alone or combined with resistance exercise training in healthy elderly men. J Clin Endocrinol Metab 87 (2): 513-23.

Lanza IR, Towse TF, Caldwell GE, Wigmore DM, Kent-Braun JA (2003). Effects of age on human muscle torque, velocity, and power in two muscle groups. J Appl Physiol 95 (6): 2361-9.

Larsen AH, Puggaard L, Hamalainen U, Aagaard P (2008). Comparison of ground reaction forces and antagonist muscle coactivation during stair walking with ageing. J Electromyogr Kinesiol 18 (4): 568-80.

Larsson L, Grimby G, Karlsson J (1979). Muscle strength and speed of movement in relation to age and muscle morphology. J Appl Physiol 46 (3): 451-6.

Larsson L, Li X, Berg HE, Frontera WR (1996). Effects of removal of weight-bearing function on contractility and myosin isoform composition in single human skeletal muscle cells. Pflugers Arch 432 (2): 320-8.

Larsson L, Li X, Frontera WR (1997). Effects of aging on shortening velocity and myosin isoform composition in single human skeletal muscle cells. Am J Physiol 272 (2 Pt 1): C638-49.

Larsson L, Moss RL (1993). Maximum velocity of shortening in relation to myosin isoform composition in single fibres from human skeletal muscles. J Physiol 472 595-614.

Larsson L, Sjodin B, Karlsson J (1978). Histochemical and biochemical changes in human skeletal muscle with age in sedentary males, age 22-65 years. Acta Physiol Scand 103 (1): 31-9.

Lauretani F, Russo CR, Bandinelli S, Bartali B, Cavazzini C, Di Iorio A, Corsi AM, Rantanen T, Guralnik JM, Ferrucci L (2003). Age-associated changes in skeletal muscles and their effect on mobility: an operational diagnosis of sarcopenia. J Appl Physiol 95 (5): 1851-60.

Lazarus NR, Harridge SD (2007). Inherent ageing in humans: the case for studying master athletes. Scand J Med Sci Sports 17 (5): 461-3.

Lehman SL, Thomson LV (2009). Skeletal muscle fatigue: the tortoise's advantage. Exerc Sport Sci Rev 37 (1): 2.

Leong B, Kamen G, Patten C, Burke JR (1999). Maximal motor unit discharge rates in the quadriceps muscles of older weight lifters. Med Sci Sports Exerc 31 (11): 1638-44.

Lexell J, Downham D (1992a). What determines the muscle cross-sectional area? J Neurol Sci 111 (1): 113-4.

Lexell J, Downham D (1992b). What is the effect of ageing on type 2 muscle fibres? J Neurol Sci 107 (2): 250-1.

Lexell J, Downham DY (1991). The occurrence of fibre-type grouping in healthy human muscle: a quantitative study of cross-sections of whole vastus lateralis from men between 15 and 83 years. Acta Neuropathol (Berl) 81 (4): 377-81.

Lexell J, Taylor CC, Sjostrom M (1988). What is the cause of the ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year-old men. J Neurol Sci 84 (2-3): 275-94.

Li C (1991). Chinese sprinters, why the wide gap? New Stud Athlet 6 (2): 10-4.

Lindle RS, Metter EJ, Lynch NA, Fleg JL, Fozard JL, Tobin J, Roy TA, Hurley BF (1997). Age and gender comparisons of muscle strength in 654 women and men aged 20-93 yr. J Appl Physiol 83 (5): 1581-7.

Lionikas A, Li M, Larsson L (2006). Human skeletal muscle myosin function at physiological and non-physiological temperatures. Acta Physiol 186 151-8.

Liu Y, Peng CH, Wei SH, Chi JC, Tsai FR, Chen JY (2006). Active leg stiffness and energy stored in the muscles during maximal counter movement jump in the aged. J Electromyogr Kinesiol 16 (4): 342-51.

Locatelli E (1996). The importance of anaerobic glycolysis and stiffness in the sprints (60, 100 and 200 metres). New Stud Athlet 11 (3): 121-5.

Locatelli E, Arsac LM (1995). The mechanics and energetics of the 100m sprint. New Stud Athlet 10 (1): 81-7.

Lowe DA, Degens H, Chen KD, Alway SE (2000). Glyceraldehyde-3-phosphate dehydrogenase varies with age in glycolytic muscles of rats. J Gerontol A Biol Sci Med Sci 55 (3): B160-4.

Lowe DA, Surek JT, Thomas DD, Thompson LV (2001). Electron paramagnetic resonance reveals age-related myosin structural changes in rat skeletal muscle fibers. Am J Physiol Cell Physiol 280 (3): C540-7.

Luhtanen P, Komi PV (1980). Force-, power-, and elasticity-velocity relationships in walking, running, and jumping. Eur J Appl Physiol Occup Physiol 44 (3): 279-89.

Macaluso A, Nimmo MA, Foster JE, Cockburn M, McMillan NC, De Vito G (2002). Contractile muscle volume and agonist-antagonist coactivation account for differences in torque between young and older women. Muscle Nerve 25 (6): 858-63.

Makrides L, Heigenhauser GJ, Jones NL (1990). High-intensity endurance training in 20- to 30- and 60- to 70-yr-old healthy men. J Appl Physiol 69 (5): 1792-8.

Manfredi TG, Fielding RA, O'Reilly KP, Meredith CN, Lee HY, Evans WJ (1991). Plasma creatine kinase activity and exercise-induced muscle damage in older men. Med Sci Sports Exerc 23 (9): 1028-34.

Mann RA (1998). Methods of sprint training from biomechanical point of view. New Stud Athlet 13 (1): 89-90.

Mann RA, Hagy J (1980). Biomechanics of walking, running, and sprinting. Am J Sports Med 8 (5): 345-50.

Mann RA, Moran GT, Dougherty SE (1986). Comparative electromyography of the lower extremity in jogging, running, and sprinting. Am J Sports Med 14 (6): 501-10.

Margaria R, Aghemo P, Rovelli E (1966). Measurement of muscular power (anaerobic) in man. J Appl Physiol 21 (5): 1662-4.

Marsh GD, Paterson DH, Govindasamy D, Cunningham DA (1999). Anaerobic power of the arms and legs of young and older men. Exp Physiol 84 (3): 589-97.

Martin JC, Farrar RP, Wagner BM, Spirduso WW (2000). Maximal power across the lifespan. J Gerontol A Biol Sci Med Sci 55 (6): M311-6.

Masani K, Kouzaki M, Fukunaga T (2002). Variability of ground reaction forces during treadmill walking. J Appl Physiol 92 (5): 1885-90.

McMahon TA, Cheng GC (1990). The mechanics of running: how does stiffness couple with speed? J Biomech 23 Suppl 1 65-78.

Meltzer DE (1994). Age dependence of Olympic weightlifting ability. Med Sci Sports Exerc 26 (8): 1053-67.

Mero A (1987). Electromyographic activity, force and anaerobic energy production in sprint running with special reference to different constant speeds ranging from submaximal to supramaximal. PhD thesis, University of Jyväskylä, Finland, No. 24.

Mero A, Komi PV (1986). Force-, EMG-, and elasticity-velocity relationships at submaximal, maximal and supramaximal running speeds in sprinters. Eur J Appl Physiol 55 (5): 553-61.

Mero A, Komi PV (1987). Electromyographic activity in sprinting at speeds ranging from sub-maximal to supra-maximal. Med Sci Sports Exerc 19 (3): 266-74.

Mero A, Komi PV (1990). Reaction time and electromyographic activity during a sprint start. Eur J Appl Physiol Occup Physiol 61 (1-2): 73-80.

Mero A, Komi PV (1994). EMG, force, and power analysis of sprint-specific strength exercises. J Appl Biomech 10: 1-13.

Mero A, Komi PV, Gregor RJ (1992). Biomechanics of sprint running. A review. Sports Med 13 (6): 376-92.

Mero A, Kuitunen S, Harland M, Kyrolainen H, Komi PV (2006). Effects of muscle-tendon length on joint moment and power during sprint starts. J Sports Sci 24 (2): 165-73.

Mero A, Luhtanen P, Viitasalo JT, Komi PV (1981). Relationships between the maximal running velocity, muscle fiber characteristics, force production and force relaxation in sprinters. Scand J Sports Sci 3 (1): 16-22.

Mero A, Peltola E (1989). Neural activation fatigued and non-fatigued conditions of short and long sprint running. Biol Sports 6 (1): 43-58.

Mero A, Peltola E, Saarela J (1987). Nopeus- ja nopeuskestävyysharjoittelu [Speed and speed endurance training]. Jyväskylä: Gummerus OY.

Michaelis I, Kwiet A, Gast U, Boshof A, Antvorskov T, Jung T, Rittweger J, Felsenberg D (2008). Decline of specific peak jumping power with age in master runners. J Musculoskelet Neuronal Interact 8 (1): 64-70.

Miyatani M, Kanehisa H, Ito M, Kawakami Y, Fukunaga T (2004). The accuracy of volume estimates using ultrasound muscle thickness measurements in different muscle groups. Eur J Appl Physiol 91 (2-3): 264-72.

Moore DH (1975). A study of age group track and field records to relate to age and running speed. Nature 253: 264-5.

Morin JB, Dalleau G, Kyrolainen H, Jeannin T, Belli A (2005). A simple method for measuring stiffness during running. J Appl Biomech 21 (2): 167-80.

Moritani T, deVries HA (1980). Potential for gross muscle hypertrophy in older men. J Gerontol 35 (5): 672-82.

Morley JE (2000). The aging athlete. J Gerontol A Biol Sci Med Sci 55 (11): M627-9.

Morse CI, Thom JM, Birch KM, Narici MV (2005). Changes in triceps surae muscle architecture with sarcopenia. Acta Physiol Scand 183 (3): 291-8.

Morse CI, Thom JM, Davis MG, Fox KR, Birch KM, Narici MV (2004). Reduced plantarflexor specific torque in the elderly is associated with a lower activation capacity. Eur J Appl Physiol 92 (1-2): 219-26.

Moss RL (1979). Sarcomere length-tension relations of frog skinned muscle fibres during calcium activation at short lengths. J Physiol 292 177-92.

Narici M, Maganaris C (2006). Muscle architecture and adaptations to functional requirements. In Bottinelli R, Reggiani C (eds.), Skeletal muscle plasticity in health and disease: from genes to whole muscle. The Netherlands, Dordrecht; Springer, pp. 265-288.

Narici MV, Maganaris CN, Reeves ND, Capodaglio P (2003). Effect of aging on human muscle architecture. J Appl Physiol 95 (6): 2229-34.

Nevill ME, Boobis LH, Brooks S, Williams C (1989). Effect of training on muscle metabolism during treadmill sprinting. J Appl Physiol 67 (6): 2376-82.

Newsholme EA, Blomstrand E, MacAndrew E, Parry-Billings M (1992). Biochemical causes of fatigue and overtraining. In Shephard RJ, Åstrand P-O (eds.), Endurance in sport. Oxford: Blackwell Scientific, pp. 351-364.

Nummela A, Mero A, Stray-Gundersen J, Rusko H (1996). Important determinants of anaerobic running performance in male athletes and non-athletes. Int J Sports Med 17 Suppl 2: S91-6.

Nummela A, Rusko H (1995). Time course of anaerobic and aerobic energy expenditure during short-term exhaustive running in athletes. Int J Sports Med 16 (8): 522-7.

Nummela A, Rusko H, Mero A (1994). EMG activities and ground reaction forces during fatigued and nonfatigued sprinting. Med Sci Sports Exerc 26 (5): 605-9.

Nummela A, Vuorimaa T, Rusko H (1992). Changes in force production, blood lactate and EMG activity in the 400-m sprint. J Sports Sci 10 (3): 217-28.

O'Conner F 2000. An analysis of the sprinting stride. In Jarver J (ed.), Sprints & relays. Mountain View, USA: Tafnews Press, pp. 29-30.

Ochala J, Frontera WR, Dorer DJ, Van Hoecke J, Krivickas LS (2007). Single skeletal muscle fiber elastic and contractile characteristics in young and older men. J Gerontol A Biol Sci Med Sci 62 (4): 375-81.

Ohkuwa T, Kato Y, Katsumata K, Nakao T, Miyamura M (1984). Blood lactate and glycerol after 400-m and 3,000-m runs in sprint and long distance runners. Eur J Appl Physiol Occup Physiol 53 (3): 213-8.

Ojanen T, Rauhala T, Häkkinen K (2007). Strength and power profiles of the lower and upper extremities in master throwers at different ages. J Strength Cond Res 21 (1): 216-22.

Ono M, Miyashita M, Asami T 1976. Inhibitory effect of lond distance running training on the vertical jump and other performances among aged males. In Komi PV (ed.), Biomechanics V-B. Baltimore, Maryland, USA: University park press, pp. 94-100.

Overend TJ, Cunningham DA, Paterson DH, Lefcoe MS (1992). Thigh composition in young and elderly men determined by computed tomography. Clin Physiol 12 (6): 629-40.

Pastoris O, Boschi F, Verri M, Baiardi P, Felzani G, Vecchiet J, Dossena M, Catapano M (2000). The effects of aging on enzyme activities and metabolite concentrations in skeletal muscle from sedentary male and female subjects. Exp Gerontol 35 (1): 95-104.

Payne AM, Delbono O (2004). Neurogenesis of excitation-contraction uncoupling in aging skeletal muscle. Exerc Sport Sci Rev 32 (1): 36-40.

Pearson SJ, Young A, Macaluso A, Devito G, Nimmo MA, Cobbold M, Harridge SD (2002). Muscle function in elite master weightlifters. Med Sci Sports Exerc 34 (7): 1199-206.

Poggi P, Marchetti C, Scelsi R (1987). Automatic morphometric analysis of skeletal muscle fibers in the aging man. Anat Rec 217 (1): 30-4.

Porter MM, Vandervoort AA, Kramer JF (1997). Eccentric peak torque of the plantar and dorsiflexors is maintained in older women. J Gerontol A Biol Sci Med Sci 52 (2): B125-31.

Poulin MJ, Vandervoort AA, Paterson DH, Kramer JF, Cunningham DA (1992). Eccentric and concentric torques of knee and elbow extension in young and older men. Can J Sport Sci 17 (1): 3-7.

Proctor DN, Sinning WE, Walro JM, Sieck GC, Lemon PW (1995). Oxidative capacity of human muscle fiber types: effects of age and training status. J Appl Physiol 78 (6): 2033-8.

Pyne DB, Boston T, Martin DT, Logan A (2000). Evaluation of the Lactate Pro blood lactate analyser. Eur J Appl Physiol 82 (1-2): 112-6.

Quetelet LAJ (1835). Sur l'homme et le developpement de ses facultes. L. Hauman and Cie. Paris: Bachelier, Imprimeur-Libraire.

Radford PF, Upton ARM (1976). Trends in speed of alternated movement during development and among elite sprinters. In Komi PV (ed.), Biomechanics V-B. Baltimore, pp. 188-193.

Reaburn P, Dascombe B (2009). Anaerobic performance in master athletes. Eur Rev Aging Phys Act 6 (1): 39-53.

Reaburn P, Logan P, Mackinnon L (1997). Serum testosterone response to high-intensity resistance training in male veteran sprint runners. J Strength Cond Res 11 (4): 256-60.

Reaburn P, Mackinnon L (1995). The effect of high-intensity isotonic training on anaerobic work capacity in veteran sprint runners. Report for coaches - Australian Sports Commision Applied Sports Research Program (unpublished).

Reaburn PRJ 1993. The lifetime athlete - Physical work capacities and skeletal muscle characteristics. Unpublished PhD thesis. The University of Queensland, Australia.

Reed RL, Pearlmutter L, Yochum K, Meredith KE, Mooradian AD (1991). The relationship between muscle mass and muscle strength in the elderly. J Am Geriatr Soc 39 (6): 555-61.

Reeves ND, Maganaris CN, Narici MV (2003). Effect of strength training on human patella tendon mechanical properties of older individuals. J Physiol 548 (Pt 3): 971-81.

Rittweger J, di Prampero PE, Maffulli N, Narici MV (2009). Sprint and endurance power and ageing: an analysis of master athletic world records. Proc Biol Sci 22; 276(1657): 683-9.

Ritzdorf W (1997). 100 meters - In: Biomechanical Research Project at the VIth World Championships in Athletics, Athens 1997: preliminary report. New Stud Athlet 12: 43-73.

Roberts EM, Cheung TK, Abdel Hafez AM, Hong D (1997a). Swing limb biomechanics of 60- to 65-year-old male runners. Gait and Posture 5 42-53.

Roberts TJ, Marsh RL, Weyand PG, Taylor CR (1997b). Muscular force in running turkeys: the economy of minimizing work. Science 275 (5303): 1113-5.

Rome LC, Sosnicki AA, Goble DO (1990). Maximum velocity of shortening of three fibre types from horse soleus muscle: implications for scaling with body size. J Physiol 431 173-85.

Rowe PH, Lincoln GA, Racey PA, Lehane J, Stephenson MJ, Shenton JC, Glover TD (1974). Temporal variations of testosterone levels in the peripheral blood plasma of men. J Endocrinol 61 (1): 63-73.

Runge M, Rittweger J, Russo CR, Schiessl H, Felsenberg D (2004). Is muscle power output a key factor in the age-related decline in physical performance? A comparison of muscle cross section, chair-rising test and jumping power. Clin Physiol Funct Imaging 24 (6): 335-40.

Sahlin K, Alvestrand A, Brandt R, Hultman E (1978). Intracellular pH and bicarbonate concentration in human muscle during recovery from exercise. J Appl Physiol 45 (3): 474-80.

Sale DG 1991. Neural adaptation to strength training. In Komi PV (ed.), Strength and Power in Sport. Boston: Blackwell Science, pp. 249-265.

Sallinen J 2007. Dietary intake and strength training adaptations in 50-70 -year old men and women. PhD thesis, University of Jyväskylä, Finland, No. 123.

Sallinen J, Pakarinen A, Fogelholm M, Alen M, Volek JS, Kraemer WJ, Hakkinen K (2007). Dietary intake, serum hormones, muscle mass and strength during strength training in 49 - 73-year-old men. Int J Sports Med 28 (12): 1070-6.

Salthouse TA (1976). Speed and age: multiple rates of age decline. Exp Aging Res 2 (4): 349-59.

Saltin B 1986. The aging endurance athlete. In Sutton JR, Brock RM (eds.), Sports medicine for the mature athlete. Indianapolis, Indiana : Benchmark Press, Inc, pp. 59-80.

Samson MM, Meeuwsen IB, Crowe A, Dessens JA, Duursma SA, Verhaar HJ (2000). Relationships between physical performance measures, age, height and body weight in healthy adults. Age Ageing 29 (3): 235-42.

Sanada K, Kearns CF, Midorikawa T, Abe T (2006). Prediction and validation of total and regional skeletal muscle mass by ultrasound in Japanese adults. Eur J Appl Physiol 96 (1): 24-31.

Savelberg HH, Meijer K (2004). The effect of age and joint angle on the proportionality of extensor and flexor strength at the knee joint. J Gerontol A Biol Sci Med Sci 59 (11): 1120-8.

Schmidtbleicher D, Gollhofer A, Frick U (1988). Effects of stretch-shortening typed training on the performance capability and innervation characteristics of leg extensor muscles. In De Groot G, Hollander A, Huijing P, Van Ingen Schenau GJ (eds.), Biomechanics XI-A. Amsterdam: Free University Press, pp.185-189.

Schultz AB (1995). Muscle function and mobility biomechanics in the elderly: an overview of some recent research. J Gerontol A Biol Sci Med Sci 50: 60-3.

Semmler JG, Nordstrom MA (1998). Motor unit discharge and force tremor in skill- and strength-trained individuals. Exp Brain Res 119 (1): 27-38.

Shimojo N, Naka K, Uenoyama H, Hamamoto K, Yoshioka K, Okuda K (1993). Electrochemical assay system with single-use electrode strip for measuring lactate in whole blood. Clin Chem 39 (11 Pt 1): 2312-4.

Shoepe TC, Stelzer JE, Garner DP, Widrick JJ (2003). Functional adaptability of muscle fibers to long-term resistance exercise. Med Sci Sports Exerc 35 (6): 944-51.

Short KR, Vittone JL, Bigelow ML, Proctor DN, Coenen-Schimke JM, Rys P, Nair KS (2005). Changes in myosin heavy chain mRNA and protein expression in human skeletal muscle with age and endurance exercise training. J Appl Physiol 99 (1): 95-102.

Simonsen EB, Thomsen L, Klausen K (1985). Activity of mono- and biarticular leg muscles during sprint running. Eur J Appl Physiol Occup Physiol 54 (5): 524-32.

Sipilä S, Koskinen SO, Taaffe DR, Takala TE, Cheng S, Rantanen T, Toivanen J, Suominen H (2004). Determinants of lower-body muscle power in early postmenopausal women. J Am Geriatr Soc 52 (6): 939-44.

Sipilä S, Suominen H (1991). Ultrasound imaging of the quadriceps muscle in elderly athletes and untrained men. Muscle Nerve 14 (6): 527-33.

Sipilä S, Viitasalo J, Era P, Suominen H (1991). Muscle strength in male athletes aged 70-81 years and a population sample. Eur J Appl Physiol Occup Physiol 63 (5): 399-403.

Skelton DA, Greig CA, Davies JM, Young A (1994). Strength, power and related functional ability of healthy people aged 65-89 years. Age Ageing 23 (5): 371-7.

Smerdu V, Karsch-Mizrachi I, Campione M, Leinwand L, Schiaffino S (1994). Type IIx myosin heavy chain transcripts are expressed in type IIb fibers of human skeletal muscle. Am J Physiol 267 (6 Pt 1): C1723-8.

Söderlund K, Hultman E (1986). Effects of delayed freezing on content of phosphagens in human skeletal muscle biopsy samples. J Appl Physiol 61 (3): 832-5.

Sosnoff JJ, Vaillancourt DE, Newell KM (2004). Aging and rhythmical force output: loss of adaptive control of multiple neural oscillators. J Neurophysiol 91 (1): 172-81.

Spirduso WW (2005). Physically elite older adults. In: Spirduso, WW, Francis KL, MacRae PG (eds.), Physical dimensions of aging. Champaign, Ill, Human Kinetics, pp. 287-316.

Spirduso WW, Francis KL, MacRae PG (2005). Physical dimensions of aging. Human Kinetics.

Stackhouse SK, Stevens JE, Lee SC, Pearce KM, Snyder-Mackler L, Binder-Macleod SA (2001). Maximum voluntary activation in nonfatigued and fatigued muscle of young and elderly individuals. Phys Ther 81: 1102-9.

Stafilidis S, Arampatzis A (2007). Track compliance does not affect sprinting performance. J Sports Sci 25 (13): 1479-90.

Stainsby WN, Brooks GA (1990). Control of lactic acid metabolism in contracting muscles and during exercise. Exerc Sport Sci Rev 18: 29-63.

Staron RS, Hagerman FC, Hikida RS, Murray TF, Hostler DP, Crill MT, Ragg KE, Toma K (2000). Fiber type composition of the vastus lateralis muscle of young men and women. J Histochem Cytochem 48 (5): 623-9.

Stevens JE, Stackhouse SK, Binder-Macleod SA, Snyder-Mackler L (2003). Are voluntary muscle activation deficits in older adults meaningful? Muscle Nerve 27 (1): 99-101.

Stone M, Plisk S, Collins D (2002). Training principles: evaluation of modes and methods of resistance training--a coaching perspective. Sports Biomech 1 (1): 79-103.

Stone MH, O'Bryant HS, McCoy L, Coglianese R, Lehmkuhl M, Schilling B (2003). Power and maximum strength relationships during performance of dynamic and static weighted jumps. J Strength Cond Res 17 (1): 140-7.

Sugiura Y, Saito T, Sakuraba K, Sakuma K, Suzuki E (2008). Strength deficits identified with concentric action of the hip extensors and eccentric action of the hamstrings predispose to hamstring injury in elite sprinters. J Orthop Sports Phys Ther 38 (8): 457-64.

Suominen H (1978). Effects of physical training in middle-aged and elderly people. With special regard to skeletal muscle, connective tissue, and functional aging. PhD thesis, University of Jyväskylä, Finland, No. 11.

Suominen H, Rahkila P (1991). Bone mineral density of the calcaneus in 70- to 81-yr-old male athletes and a population sample. Med Sci Sports Exerc 23 (11): 1227-33.

Surakka J 2005. Power-type strength training in middle-aged men and women. PhD thesis, National Public Health Institute, Turku, Finland.

Tanaka H, Seals DR (2003). Invited Review: Dynamic exercise performance in Masters athletes: insight into the effects of primary human aging on physiological functional capacity. J Appl Physiol 95 (5): 2152-62.

Tesch P, Sjodin B, Karlsson J (1978). Relationship between lactate accumulation, LDH activity, LDH isozyme and fibre type distribution in human skeletal muscle. Acta Physiol Scand 103 (1): 40-6.

Thom JM, Morse CI, Birch KM, Narici MV (2005). Triceps surae muscle power, volume, and quality in older versus younger healthy men. J Gerontol A Biol Sci Med Sci 60 (9): 1111-7.

Thom JM, Morse CI, Birch KM, Narici MV (2007). Influence of muscle architecture on the torque and power-velocity characteristics of young and elderly men. Eur J Appl Physiol 100 (5): 613-9.

Thorstensson A (1976). Muscle strength, fiber types and enzyme activities in man. Acta Physiol Scand (Suppl. 443).

Tomonaga M (1977). Histochemical and ultrastructural changes in senile human skeletal muscle. J Am Geriatr Soc 25 (3): 125-31.

Trappe S, Gallagher P, Harber M, Carrithers J, Fluckey J, Trappe T (2003). Single muscle fibre contractile properties in young and old men and women. J Physiol 552 (Pt 1): 47-58.

Trappe S, Williamson D, Godard M, Porter D, Rowden G, Costill D (2000). Effect of resistance training on single muscle fiber contractile function in older men. J Appl Physiol 89 (1): 143-52.

Trappe SW, Costill DL, Fink WJ, Pearson DR (1995). Skeletal muscle characteristics among distance runners: a 20-yr follow-up study. J Appl Physiol 78 (3): 823-9.

Trappe SW, Costill DL, Goodpaster BH, Pearson DR (1996). Calf muscle strength in former elite distance runners. Scand J Med Sci Sports 6 : 205-10.

Tucker R, Dugas J 2008. Science of sports - Beijing 2008: Men 100m race analysis [http://www.sportsscientists.com/2008/08/beijing-2008-men-100m-race-analysis.html].

Tzankoff SP, Norris AH (1979). Age-related differences in lactate distribution kinetics following maximal exercise. Eur J Appl Physiol Occup Physiol 42 (1): 35-40.

Van Cutsem M, Duchateau J, Hainaut K (1998). Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. J Physiol 513 (Pt 1): 295-305.

Viitasalo J, Häkkinen K, Komi PV (1981). Isometric and dynamic force production and muscle fiber composition in man. J Hum Mov Stud 7: 199-209.

Viitasalo JH, Komi PV (1975). Signal characteristics of EMG with special reference to reproducibility of measurements. Acta Physiol Scand 93 (4): 531-9.

Viitasalo JT 1985. Measurement of force-velocity characteristics for sportmen in field conditions. In Winter DA, Norman RW, Wells RP, Hayes KC, Patla AE. (ed.), Biomechanics IX-A. Champaign, Illinois: Human Kinetics Publishers, pp. 96-101.

Viitasalo JT, Komi PV (1978). Force-time characteristics and fiber composition in human leg extensor muscles. Eur J Appl Physiol 40 (1): 7-15.

Viitasalo JT, Saukkonen S, Komi PV (1980). Reproducibility of measurements of selected neuromuscular performance variables in man. Electromyogr Clin Neurophysiol 20 (6): 487-501.

Walshe AD, Wilson GJ, Murphy AJ (1996). The validity and reliability of a test of lower body musculotendinous stiffness. Eur J Appl Physiol Occup Physiol 73 (3-4): 332-9.

Wang C, Catlin DH, Starcevic B, Heber D, Ambler C, Berman N, Lucas G, Leung A, Schramm K, Lee PW, Hull L, Swerdloff RS (2005). Low-fat high-fiber diet decreased serum and urine androgens in men. J Clin Endocrinol Metab 90 (6): 3550-9.

Wang FC, de Pasqua V, Delwaide PJ (1999). Age-related changes in fastest and slowest conducting axons of thenar motor units. Muscle Nerve 22 (8): 1022-9.

Welle S, Bhatt K, Thornton CA (2000). High-abundance mRNAs in human muscle: comparison between young and old. J Appl Physiol 89: 297-304.

Welle S, Thornton C, Statt M (1995). Myofibrillar protein synthesis in young and old human subjects after three months of resistance training. Am J Physiol 268 (3 Pt 1): E422-7.

Westerblad H, Allen DG, Lannergren J (2002). Muscle fatigue: lactic acid or inorganic phosphate the major cause? News Physiol Sci 17 17-21.

Weyand PG, Cureton KJ, Conley DS, Sloniger MA, Liu YL (1994). Peak oxygen deficit predicts sprint and middle-distance track performance. Med Sci Sports Exerc 26 (9): 1174-80.

Weyand PG, Davis JA (2005). Running performance has a structural basis. J Exp Biol 208 (Pt 14): 2625-31.

Weyand PG, Sternlight DB, Bellizzi MJ, Wright S (2000). Faster top running speeds are achieved with greater ground forces not more rapid leg movements. J Appl Physiol 89 (5): 1991-9.

Widrick JJ, Stelzer JE, Shoepe TC, Garner DP (2002). Functional properties of human muscle fibers after short-term resistance exercise training. Am J Physiol Regul Integr Comp Physiol 283 (2): R408-16.

Widrick JJ, Trappe SW, Blaser CA, Costill DL, Fitts RH (1996a). Isometric force and maximal shortening velocity of single muscle fibers from elite master runners. Am J Physiol 271 (2 Pt 1): C666-75.

Widrick JJ, Trappe SW, Costill DL, Fitts RH (1996b). Force-velocity and force-power properties of single muscle fibers from elite master runners and sedentary men. Am J Physiol 271 (2 Pt 1): C676-83.

Wiemann K, Tidow G (1995). Relative activity of hip and knee extensors in sprinting - implications for training. Natl Strength Cond Assoc J 10: 29-49.

Williams KR, Cavanagh PR, Ziff JL (1987). Biomechanical studies of elite female distance runners. Int J Sports Med 8: s107-18.

Wilson GJ, Murphy AJ, Walshe A (1996). The specificity of strength training: the effect of posture. Eur J Appl Physiol Occup Physiol 73 (3-4): 346-52.

Winegard KJ, Hicks AL, Sale DG, Vandervoort AA (1996). A 12-year follow-up study of ankle muscle function in older adults. J Gerontol A Biol Sci Med Sci 51 (3): B202-7.

Young A, Stokes M, Crowe M (1985). The size and strength of the quadriceps muscles of old and young men. Clin Physiol 5 (2): 145-54.

Young W, Benton D, Duthie G, Pryor J (2001). Resistance training for shorts sprints and maximum-speed sprints. Strength Cond. J. 23 (2): 7-13.

Yu F, Hedstrom M, Cristea A, Dalen N, Larsson L (2007). Effects of ageing and gender on contractile properties in human skeletal muscle and single fibres. Acta Physiol (Oxf) 190 (3): 229-41.

Yue GH, Ranganathan VK, Siemionow V, Liu JZ, Sahgal V (1999). Older adults exhibit a reduced ability to fully activate their biceps brachii muscle. J Gerontol A Biol Sci Med Sci 54 (5): M249-53.

PAPER I

AGE-RELATED DIFFERENCES IN 100-M SPRINT PERFORMANCE IN MALE AND FEMALE MASTER RUNNERS

by

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I

Age-Related Differences in 100-m Sprint Performance in Male and Female Master Runners

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ABSTRACT

KORHONEN, M. T., A. MERO, and H. SUOMINEN. Age-Related Differences in 100-m Sprint Performance in Male and Female Master Runners. *Med. Sci. Sports Exerc*., Vol. 35, No. 8, pp. 1419–1428, 2003. **Purpose:** This study was undertaken to investigate age-related differences in the velocity and selected stride parameters in male and female master sprinters and to determine which stride characteristics were related to the overall decline in the performance of the 100 m with age. **Methods:** The performances of 70 finalists (males 40–88 yr, females 35–87 yr) at the European Veterans Athletics Championships were recorded using two high-speed cameras (200 Hz) with a panning video technique and distance markers at 10-m intervals. Velocity, stride length (SL), stride rate (SR), ground contact time (CT), and flight time (FT) during the acceleration, peak velocity, and deceleration phases of the 100-m race were determined from the video records with the aid of the Peak Performance analysis system. **Results:** There was a general decline in sprint performances with age, the decrease becoming more evident around 65–70 yr of age. The velocity during the different phases of the run declined on average from 5 to 6% per decade in males and from 5 to 7% per decade in females. Similarly, SL showed clear reductions with increasing age, whereas SR remained unchanged until the oldest age groups in both genders. Furthermore, the CT, which correlated with velocity, was significantly longer, and FT, which correlated with both velocity and SL, was shorter in older age groups. **Conclusion:** Our findings indicated that age-associated differences in velocity in elite master sprinters were similar in each phase of the 100-m run. The deterioration of the overall performance with age was primarily related to reduction in SL and increase in CT. **Key Words:** AGING, BIOMECHANICS, LOCOMOTION, MASTER ATHLETES, RUNNING

Aging results in a gradual decline in physical performance. One of the prominent features of aging is a decrease in the ability to produce speed and force in whole-body movements that involve a number of different muscle groups working together (see 29 for review). Sprint running represents a form of complex whole-body movements in which the stretch-shortening cycle performance is largely dependent on the ability of the neuromuscular system to generate explosive force during the ground contact phase (23,24). With increasing age, both male and female athletes experience a rather linear decline in the ability to run fast until about 75–80 yr, followed by progressively greater decline. The comparison of the 100-m world records of master athletes over time shows that from the age of 40 to 80 the deterioration of maximal sprinting velocity is ~7% per decade for males and ~9% per decade for females (27).

Our present knowledge about the age-related changes in sprint running is based primarily on the magnitude of the deterioration in performance with age. However, there is a paucity of information regarding the effect of age on the parameters that determine the level and nature of the running performance. Because running velocity is the product of the stride rate (SR) and stride length (SL), these direct performance measures have frequently been analyzed in major championships to provide fundamental information about sprint performances (1,3,12,13,22,25). We are aware of only one study that has focused on the influence of aging on the relationship between sprint stride parameters and velocity. In that study by Hamilton (11), the nature of age-related decline in sprinting performance was examined by analyzing selected performance measures during the peak velocity phase of 100-m and 200-m events in master runners aged 30–94 yr (male and female groups, and the two sprint distances from different competitions were combined for the analysis). The results of that study demonstrated that the peak running velocity declined from 8.9 $m \cdot s^{-1}$ at age 30–39 to 4.9 $m \cdot s^{-1}$ in runners over 90. The

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TABLE 1. Physical characteristics and training history of male and female athletes in different age groups (mean, SE).

	Males					Females					
	40–49 yr (*N* = 7)	50–59 yr (*N* = 8)	60–69 yr (*N* = 8)	70–79 yr (*N* = 8)	80–89 yr (*N* = 6)	35–39 yr (*N* = 3)	40–49 yr (*N* = 8)	50–59 yr (*N* = 8)	60–69 yr (*N* = 8)	70–79 yr (*N* = 4)	80–89 yr (*N* = 2)
Age (yr)	43.6 (1.2)	54.9 (1.2)	63.3 (0.9)	73.6 (3.6)	84.4 (2.8)	37.3 (0.6)	43.6 (0.7)	54.8 (1.2)	64.4 (1.1)	71.8 (1.2)	83.5 (3.5)
Height (m)	1.77 (0.01)	1.77 (0.03)	1.73 (0.02)	1.73 (0.01)	1.67 (0.01)	NA[c]	1.70 (0.02)	1.66 (0.03)	1.67 (0.02)	1.68 (0.02)	1.53 (0.01)
Weight (kg)	80.2 (2.6)	77.5 (3.2)	69.3 (1.3)	71.0 (2.5)	63.2 (4.1)	NA	63.5 (2.3)	58.8 (3.0)	61.3 (3.1)	59.8 (6.2)	55.8 (5.4)
Training frequency (sessions per week)	3.3 (0.6)	3.5 (0.5)	4.5 (0.7)	4.8 (0.7)	2.8 (0.6)	NA	5.0 (0.5)	4.2 (0.2)	2.6 (0.6)	2.2 (1.8)	4.8 (2.3)
Training hours ($h \cdot wk^{-1}$)	5.8 (0.5)	6.0 (0.8)	7.6 (1.2)	9.0 (1.8)	6.1 (2.0)	NA	7.6 (2.6)	8.1 (2.7)	4.4 (2.0)	2.8 (1.8)	5.0 (2.1)
Sprint training of total training[a] (%)	92.2 (5.9)	80.6 (11.1)	81.7 (8.7)	71.8 (14.3)	80.2 (4.4)	NA	85.3 (7.3)	75.9 (11.2)	71.5 (12.1)	84.2 (15.8)	59.6 (36.8)
Years of sprint training	21.7 (4.7)	22.9 (8.5)	38.3 (10.0)	30.8 (8.3)	40.5 (13.3)	NA	21.5 (2.1)	30.8 (4.5)	24.7 (6.4)	12.5 (7.5)	42.5 (22.5)
All time personal best 100 m[b] (s)	10.87 (0.09)	10.96 (0.06)	11.07 (0.28)	11.00 (0.34)	11.35 (0.15)	NA	11.99 (0.31)	11.93 (0.18)	12.17 (0.12)	NA	NA

[a] Sprint training includes speed, speed endurance, resistance, and plyometric training.
[b] Males *N* = 22, females *N* = 10.
[c] Nonapplicable.

decline in peak velocity with age was attributed primarily to a decrease in SL.

Regarding the overall performance of sprint running, the interrelationships between SL, SR, and velocity changes over the entire course of a 100-m run have been investigated in several studies (1,3,8,10,25,26). Reports from major athletic competitions have shown that in young male and female sprinters the initial acceleration up to around 20 m (~9–10 $m \cdot s^{-1}$) is achieved by a combined increase in SR and SL, after which the velocity increase is attributed mainly to changes in SL up to about 50–80 m (~10–12 $m \cdot s^{-1}$) (1,25,28). As running velocity approaches the maximum, the contact time (CT) decreases, and the sprint stride becomes increasingly dependent on the runner's ability to produce force at a fast rate (24). An important factor for overall sprint performance is also the ability to maintain proper sprint mechanics and subsequently a high percentage of maximum velocity at the end of the run. Studies in world-class sprinters have indicated that during the last 10–20 m of the 100-m race the velocity declines by ~2–7% in males and by ~3–8% in females (1,3,7,25,28). This gradual loss of velocity after the peak velocity phase seems to be explained to a great extent by the decrease in SR (1,4,10,25).

The available knowledge of the effect of age on sprint running ability is based only on age-group world records and on one previous study (11) examining the peak velocity phase of master athletes. However, because not only maximum running velocity but also acceleration and speed endurance are critical factors in the overall sprint performance, it is important to assess all the velocity phases in the same study. Therefore, the aim of the present study was to investigate the velocity and selected stride parameters during the acceleration, peak velocity, and deceleration phases of the run, and to determine which stride characteristics were related to the decline in the overall 100-m performance with age. The present analysis was limited to the very best male and female sprinters in order to minimize the effect of age-associated confounding factors such as heterogeneity in performance level in different age categories.

METHODS

Subject selection. This study was part of a larger sprint running research project conducted during the XII European Veterans Athletics Championships held in Jyväskylä, Finland, in July 2000. During the championships, the highest ranked (according to the 100-m performance of the previous year) male (40–88 yr) and female (35–87 yr) sprinters were invited by personal letter to participate in the study. Besides these athletes, other sprinters who qualified for the semifinals in 100-m sprint events were contacted after the preliminary heats and informed about the study. Fifty-six males and 44 females representing 12 countries participated and gave their written consent for the study. Of the athletes, 37 males and 33 females (the fastest 1–4 finalists in each 5-yr age category) were selected for the final analyses. Approval for this study was granted by the Ethics Committee of the University of Jyväskylä. The physical characteristics of the selected athletes are shown in Table 1.

Training and competition history. The subjects of the study completed a detailed questionnaire (translated into eight languages) about current and former training, competition performance, and injuries. In addition, the athletes participated in a brief interview lasting about 30 min during which specific information on training methods and competition background was obtained. On this occasion, the measurements of body weight and height, and reaction speed were carried out. This information could not be obtained from 10 males and 7 females (including all 35-yr-old females). On the basis of the questionnaire and personal interview, most of the subjects had in their youth competed in sprint running events and maintained regular year-round training. There were slight age-related differences in the type and volume of the physical training. However, the race times showed that the subjects were rather similar with regard to the relative level of competitive performance and represented the world-class level in each age class. The training and competition history of the athletes are given in Table 1.

Collection of video data. The research project was carried out in close cooperation with the local organizing committee. One year before the championships, the research plan was presented to the competition organizers and arrangements for videotaping were made. For the use of the present study, three camera locations for videotaping procedures were agreed upon. All data were collected in a manner so as not to interfere with the competition.

During the championships 51 100-m sprint races, including 32 heats and 19 finals, were recorded by a four-person crew using two Peak Performance (HSC 200, Peak Performance Technologies, Inc.) high-speed cameras (200 Hz) with the panning video technique. The use of the panning video technique, in which the camera rotates on a single axis, enables one to cover large portions of the race by keeping runner's image size sufficiently large for accurate analysis (4,5). The cameras were mounted on specialized tripods with pan and tilt decoders and genlocked to each other to synchronize the video frames. Before and after measurements calibration was done by calibration rods (height 3.7 m) placed in both ends of the track. The cameras were equipped with Tamron (f: 1:2.5/20–80 mm) and Rank Taylor Hobson Monital (f: 1:2.1/20–100 mm) zoom lenses. For the recordings, S-VHS tapes (Basf SE 60 min) were utilized.

During the 100-m events, the cameras were located parallel to the track at a distance of 93 m away from the first lane (behind back straight) and at the point of 32 m and 72 m from a starting line. The cameras were placed on videotaping racks 5 m above the ground level and the optical axes of the cameras focused downward toward the track at an angle of approximately 4°. The first camera (at the 32-m point) covered the first 60 m of the race (including the smoke from the starter's pistol) and the second camera the latter half of the race (40–110 m). The angular motion of both of the cameras involved about 30° of rotation to capture the respective portion of the race. When the optical axes of the cameras intersected the plane of motion at right angles, the field of view for the lane one was approximately 7 m. To obtain horizontal velocity and stride parameters in different phases of the run, the distance markers were placed at 10-m intervals along both sides of the track. The position of markers was determined by placing the calibration rods in the middle of lanes of the track, and with the aid of cameras projecting the line from the calibration rods to both sides of the tracks indicating the 10-m points for analysis. The foreground markers (white plastic plates of 12 × 15 cm with black tape marks on) were placed on the curbs of track, and the background markers (black tapes of 5 cm wide) were taped to the track fences at the height of chest and head.

Reduction and analysis of video data. For the purpose of the present study, the performances of the four fastest runners in the finals in each 5-yr age category of both genders were to be analyzed. In some of the races, however, there was such a large heterogeneity in performance that the videotaped view required for accurate time analysis was not wide enough to capture more than three runners. The small number of female competitors over 70 yr (one or two in each age class) further reduced the analysis to a total of 70 running performances.

The video records were analyzed by two researchers with the aid of a Panasonic VCR (AG-7355) and a Hewlett Packard (Pentium II) computer with a 17-inch monitor (ViewSonic GA 771). The computer was operated with a Motus 32 workstation (Peak Performance Technologies, Inc.). This software enabled one to sequentially encode every frame of the recorded tape with a number and thus provided an actual frame rate of 200 Hz. The progressive running velocity was determined from the time required to run between consecutive 10-m sequences. The sequence times were measured by capturing the video frames when the runner crossed the 10-m distance markers and by dividing the number of frames elapsed between adjacent 10-m lines by the frame rate. Stride length (from one foot contact to other foot contact) was determined by dividing the sequence distance by the number of strides within each sequence. Stride rate (Hz) was calculated by dividing number of strides within each sequence by the sequence time. Ground contact times during peak velocity phase (the fastest 10-m sequence) and deceleration phase (90 m) of the run were measured by determining the number of frames elapsed from foot contact to toe-off in the same foot, and the flight times (during peak velocity and deceleration phases) were measured from the frames elapsed from one foot toe-off to opposite foot ground contact. Contact and flight time results were averaged over four consecutive strides.

Identification of the direct performance measures from video data was clear. In all of the sprint races, there were slight differences between the performance level of the fastest runners, and thus there were few view obstructions due to the fellow competitors that could prevent precise definition of the instant of foot contact, toe-off, and crossing the distance markers. Also, the officials and stands in the field area were situated so that they did not obstruct the camera views. The beginning of the race could be detected from the smoke of the starting pistol, and the exact moment of crossing the finish line could be verified with the aid of the official competition results. With the frame rate of 200 Hz, the margin of error in temporal measurements due to accuracy of the analyzing system was 0.005 s. Possible sources of error related to spatial information were lens distortions and photographic perspective error due to the distance of the camera to the track. The effect of lens distortion on stride parameters is expected to be small and primarily random in nature because the results were averaged over several consecutive strides in each 10-m sequence. The perspective errors were minimized by positioning the cameras sufficiently far away (93 m) from the runners and are also considered to be small for the purpose of the present study. In this study, a potential source of variation is air resistance. Despite that during the competition the wind readings of all the 100-m sprint races were within the legal wind speed limit of $+2.0\ m \cdot s^{-1}$, there were small differences in wind readings between the races (range from $-1.8\ m \cdot s^{-1}$ to $+0.9\ m \cdot s^{-1}$ in females and from $-1.2\ m \cdot s^{-1}$ to $+1.0\ m \cdot s^{-1}$ in males) and is thus expected to have

TABLE 2. Comparison of selected performance parameters of the 100-m run in 10-yr age groups of male runners (mean,SE).

Parameters	Age group				
	40–49 yr (*N* = 7)	50–59 yr (*N* = 8)	60–69 yr (*N* = 8)	70–79 yr (*N* = 8)	80–89 yr (*N* = 6)
Velocity ($m \cdot s^{-1}$)					
Average over 100 m	8.83[b,c,d,e] (0.10)	8.13[a,d,e] (0.07)	7.78[a,b,e] (0.07)	6.95[a,b,c,e] (0.14)	5.89[a,b,c,d] (0.13)
During acceleration (0–10 m)	4.76[b,c,d,e] (0.04)	4.45[a,d,e] (0.05)	4.35[a,d,e] (0.07)	3.99[a,b,c,e] (0.06)	3.45[a,b,c,d] (0.08)
During peak velocity phase	10.20[b,c,d,e] (0.15)	9.32[a,d,e] (0.09)	8.90[a,d,e] (0.09)	7.89[a,b,c,e] (0.16)	6.74[a,b,c,d] (0.15)
During final phase (90–100 m)	9.67[b,c,d,e] (0.16)	8.58[a,d,e] (0.11)	8.14[a,d,e] (0.10)	7.28[a,b,c,e] (0.17)	6.02[a,b,c,d] (0.17)
Stride rate (Hz)					
Average over 100 m	4.39[e] (0.06)	4.35[e] (0.09)	4.32[e] (0.04)	4.19 (0.06)	3.94[a,b,c] (0.09)
During acceleration (0–10 m)	3.70[e] (0.08)	3.70[e] (0.08)	3.75[e] (0.06)	3.50[e] (0.09)	3.11[a,b,c,d] (0.09)
During peak velocity phase	4.66[e] (0.06)	4.64[e] (0.09)	4.54 (0.05)	4.42 (0.06)	4.23[a,b] (0.09)
During final phase (90–100 m)	4.27[e] (0.07)	4.23[e] (0.10)	4.18[e] (0.07)	4.03 (0.04)	3.76[a,b,c] (0.13)
Stride length (m)					
Average over 100 m	2.01[b,c,d,e] (0.02)	1.87[a,d,e] (0.04)	1.80[a,d,e] (0.03)	1.66[a,b,c,e] (0.04)	1.49[a,b,c,d] (0.03)
During acceleration (0–10 m)	1.29[c,d,e] (0.03)	1.21 (0.02)	1.16[a] (0.03)	1.14[a] (0.03)	1.11[a] (0.02)
During peak velocity phase	2.19[b,c,d,e] (0.03)	2.02[a,d,e] (0.04)	1.96[a,d,e] (0.03)	1.79[a,b,c,e] (0.05)	1.60[a,b,c,d] (0.03)
During final phase (90–100 m)	2.26[b,c,d,e] (0.04)	2.04[a,d,e] (0.05)	1.95[a,e] (0.05)	1.81[a,b,e] (0.05)	1.61[a,b,c,d] (0.03)
Contact time (ms)					
During peak velocity phase	98[c,d,e] (0.7)	102[d,e] (0.1)	109[a,e] (0.2)	118[a,b,e] (2)	141[a,b,c,d] (5)
During final phase	103[c,d,e] (2)	106[d,e] (2)	117[a,e] (2)	130[a,b,e] (4)	156[a,b,c,e] (7)
Flight time (ms)					
During peak velocity phase (ms)	121[e] (0.2)	121[e] (0.3)	116[e] (0.2)	111[e] (0.3)	97[a,b,c,d] (0.4)
During final phase	125[d,e] (4)	122[d,e] (2)	115[e] (3)	108[a,b] (3)	96[a,b,c] (3)

[a,b,c,d,e] Group is significantly different ($P < 0.05$) from the 40- to 49-, 50- to 59-, 60- to 69-, 70- to 79-, and 80- to 89-yr-old group, respectively.

some effect on the results. Otherwise, there were small variations in weather conditions (no rain; temperature 16–18°C; humidity 70–80%) during afternoon finals (5:00–8:30 p.m.).

Statistical analysis. ANOVA was used to determine differences in the dependent variables (velocity, SL, SR, CT, and FT) among age groups. In the case of significant *F*-value from ANOVA, the Tukey *post hoc* analysis was used to identify the significance of differences between each pair of age groups. To add the power of ANOVA and reduce the number of group comparisons, two adjacent 5-yr categories were combined (Tables 2 and 3). Accordingly, the power of detecting a significant ($P < 0.05$) age effect reached a level of 0.72 (stride rate) up to 1.00 (velocity). Both linear and polynomial regression analyses were performed to determine the rate of change in performance variables with age. Pearson's correlation coefficient was used to examine the relationships between variables. Where appropriate, partial correlation was used to control the effect of age on these relationships. Statistical significance was set at the 0.05 level. All the analyses were performed using SPSS 9.0.1 for Windows (SPSS, Inc.).

RESULTS

Generally, males showed higher running velocity, higher SR, larger SL, and shorter CT than females. No clear gender difference was found in FT values. In the following, the main results will be examined separately for males and females.

Velocity. The 100-m race times ranged from 11.14 ± 0.19 s (40–44 yr) to 17.80 ± 0.57 s (85–89 yr) in males and

TABLE 3. Comparison of selected performance parameters of the 100-m run in 10-yr age groups of female runner (mean, SE).

Parameters	Age Group					
	35–39 yr (*N* = 3)	40–49 yr (*N* = 8)	50–59 yr (*N* = 8)	60–69 yr (*N* = 8)	70–79 yr (*N* = 4)	80–89 yr (*N* = 2)
Velocity ($m \cdot s^{-1}$)						
Average over 100 m	7.82[c,d,e,f] (0.07)	7.52[d,e,f] (0.10)	7.11[a,d,e,f] (0.09)	6.62[a,b,c,e,f] (0.12)	5.61[a,b,c,d,f] (0.09)	4.61[a,b,c,d,e] (0.09)
During acceleration (0–10 m)	4.42[c,d,e,f] (0.01)	4.20[d,e,f] (0.05)	4.11[a,d,e,f] (0.06)	3.85[a,b,c,e,f] (0.04)	3.44[a,b,c,d,f] (0.14)	2.94[a,b,c,d,e] (0.13)
During peak velocity phase	8.88[c,d,e,f] (0.07)	8.59[c,d,e,f] (0.13)	8.04[a,b,d,e,f] (0.10)	7.49[a,b,c,e,f] (0.12)	6.44[a,b,c,d,f] (0.15)	5.34[a,b,c,d,e] (0.14)
During final phase (90–100 m)	8.34[c,d,e,f] (0.18)	8.02[d,e,f] (0.14)	7.40[a,e,f] (0.13)	6.80[a,b,e,f] (0.20)	5.46[a,b,c,d] (0.17)	4.38[a,b,c,d] (0.16)
Stride rate (Hz)						
Average over 100 m	4.21 (0.03)	4.16 (0.06)	4.18 (0.08)	3.97 (0.05)	3.94 (0.08)	3.78 (0.10)
During acceleration (0–10 m)	3.59 (0.04)	3.50 (0.06)	3.59 (0.09)	3.38 (0.07)	3.26 (0.22)	3.16 (0.12)
During peak velocity phase	4.47 (0.04)	4.37 (0.06)	4.37 (0.08)	4.19 (0.05)	4.22 (0.09)	4.08 (0.07)
During final phase (90–100 m)	4.11 (0.01)	4.04 (0.07)	4.06[f] (0.10)	3.80 (0.06)	3.80 (0.06)	3.55[c] (0.19)
Stride length (m)						
Average over 100 m	1.86[c,d,e,f] (0.01)	1.81[e,f] (0.03)	1.70[a,e,f] (0.03)	1.67[a,e,f] (0.04)	1.42[a,b,c,d,f] (0.02)	1.22[a,b,c,d,e] (0.01)
During acceleration (0–10 m)	1.23[e,f] (0.01)	1.20[e,f] (0.02)	1.15[f] (0.02)	1.14[f] (0.02)	1.06[a,b] (0.04)	0.93[a,b,c,d] (0.01)
During peak velocity phase	1.99[e,f] (0.01)	1.97[d,e,f] (0.03)	1.84[e,f] (0.03)	1.79[b,e,f] (0.04)	1.52[a,b,c,d] (0.03)	1.31[a,b,c,d] (0.01)
During final phase (90–100 m)	2.03[e,f] (0.05)	1.99[e,f] (0.03)	1.83[e,f] (0.04)	1.79[e,f] (0.06)	1.43[a,b,c,d] (0.02)	1.23[a,b,c,d] (0.02)
Contact time (ms)						
During peak velocity phase	103[d,e,f] (2)	108[d,e,f] (1)	110[d,e,f] (1)	124[a,b,c,e,f] (4)	151[a,b,c,d,f] (4)	180[a,b,c,d,e] (6)
During final phase	110[d,e,f] (4)	113[d,e,f] (2)	116[d,e,f] (2)	134[a,b,c,e,f] (4)	158[a,b,c,d,f] (3)	208[a,b,c,d,e] (1)
Flight time (ms)						
During peak velocity phase	121[e,f] (3)	122[e,f] (2)	126[e,f] (4)	116[e,f] (4)	93[a,b,c,d] (2)	75[a,b,c,d] (3)
During final phase	123[e,f] (4)	128[e,f] (3)	131[e,f] (4)	118[e,f] (4)	95[a,b,c,d,f] (6)	65[a,b,c,d,e] (5)

[a,b,c,d,e,f] Group is significantly different ($P < 0.05$) from the 35- to 39-, 40 to 49-, 50- to 59-, 60- to 69-, 70- to 79-, and 80- to 89-yr-old group, respectively.

from 12.78 ± 0.20 s (35–39 yr) to 22.08 s (87 yr) in females (Fig. 1). Expressed as an average race velocity, the performance ranged from 8.98 ± 0.15 $m \cdot s^{-1}$ to 5.62 ± 0.18 $m \cdot s^{-1}$ in males and from 7.83 ± 0.12 $m \cdot s^{-1}$ to 4.53 $m \cdot s^{-1}$ in females. The average rate of decline in race velocity over the 50-yr age period was 5.8% and 6.9% per decade for males and females, respectively. However, the deterioration of performance was exponential rather than linear as shown by a second-degree polynomial curve fitting (Fig. 1).

The velocity curves of 100-m run are shown in Fig. 2, A and B, and the age-related differences in selected velocity values in Tables 2 and 3. The relative rate of age-associated decline in velocity over the first 10 m was 4.9% per decade for both genders. When controlled for age, average velocity during initial acceleration (0–10 m) correlated with race time both in males ($r = -0.73$; $P < 0.001$) and females ($r = -0.78$; $P < 0.001$).

In males, the distance required to reach the peak velocity (the fastest 10-m sequence) in the 40- to 49-yr-old runners (45 m) differed significantly ($P < 0.05$) from that in the 80–89 yr (25 m). However, the time to peak velocity (range 4.40–6.08 s) showed no significant differences between age groups. In 50–59 yr females, the distance to peak velocity (35 m) was significantly different from that (20 m) in 70–89 yr. No significant differences were observed between the age groups in time required to reach the peak velocity (range 4.15–5.58 s).

The runners' peak velocity showed clear age group differences in males and females (Tables 2 and 3). The age-related declines in peak velocity for male and female runners were 5.9% and 6.0% per decade, respectively. When controlled for age, the peak velocity showed an inverse correlation to race time both in males ($r = -0.84$; $P < 0.001$) and females ($r = -0.90$; $P < 0.001$).

The relative decrease of velocity from peak velocity sequence to the final phase of the run (range ~5–10% for males, and ~6–18% for females) correlated significantly with age both in male ($r = -0.51$; $P < 0.01$) and female runners ($r = -0.76$; $P < 0.001$).

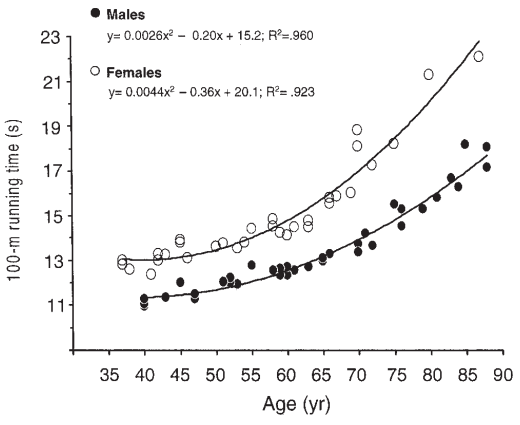

FIGURE 1—Individual values of 100-m running time as a function of age in male and female sprinters.

Stride rate. SR curves over the 100 m in Figures 2 C and D indicate that runners in all age groups and in both genders reached the maximum or near maximum SR between 10 and 20 m, whereupon SR decreased toward the end of the run. During acceleration, SR of the oldest male runners (80–89 yr) differed significantly from that of all other male groups ($P < 0.05$), and during the peak velocity phase from the 40- to 49- and 50- to 59-yr-old groups ($P < 0.01$) (Table 2). In females, there were no significant differences in SR values between adjacent age groups (Table 3). In males, average SR, initial acceleration SR, and SR during peak velocity sequence declined by 2.2%, 3.0%, and 1.9% per decade, respectively, whereas in females, the age-related declines in the average SR, acceleration SR, and peak velocity SR were 2.1%, 2.1%, and 1.6% per decade, respectively.

When controlled for age, SR during initial acceleration (0–10 m) correlated significantly with the velocity of this phase both in males ($r = 0.49$; $P < 0.01$) and in females ($r = 0.60$; $P < 0.001$). However, the average SR and SR during peak velocity and deceleration phases showed no significant correlation with velocity in either males or females. The decline in SR from the peak velocity sequence to the end of the run was associated with the reduction in velocity from the peak velocity sequence to the end of the run in males ($r = 0.37$; $P < 0.05$) and in females ($r = 0.48$; $P < 0.01$).

Stride length. SL curves in Figures 2 E and F, and the results in Tables 2 and 3 show age-related differences in SL during all phases of the 100-m run in both genders. During the acceleration, male and female runners in younger age groups were able to take longer strides and to increase their SL up to around 50 m, whereas in older runners, the maximum SL was achieved earlier. From the peak velocity phase to the deceleration phase of the run, SL remained unchanged in both males and in females. In males, average SL, initial acceleration SL, and SL of the peak velocity sequence declined by 4.7%, 2.9%, and 5.0% per decade, respectively, whereas in females, the average SL, acceleration SL, and SL of peak velocity sequence decreased by 5.1%, 3.6%, and 5.2% per decade, respectively. When expressed as relative values (SL/height), the decline in SL during peak velocity with advancing age was 4.1% per decade for males and 4.9% per decade for females.

When controlled for age, the average SL over 100 m, and SL during peak velocity and deceleration phases of the run correlated with velocity of those phases in both genders ($r > 0.43$; $P < 0.01$ in all cases). However, SL during initial acceleration over the first 10 m showed no significant correlation with the 10-m velocity in either males or females.

Ground contact time. The individual CT values during peak velocity are illustrated in Figure 3, A and B, and the age group differences in CT are shown in Tables 2 and 3. CT increased progressively as running velocity decreased with age. Also, the relative time spent in contact phase (% stride) increased linearly with age and velocity from an

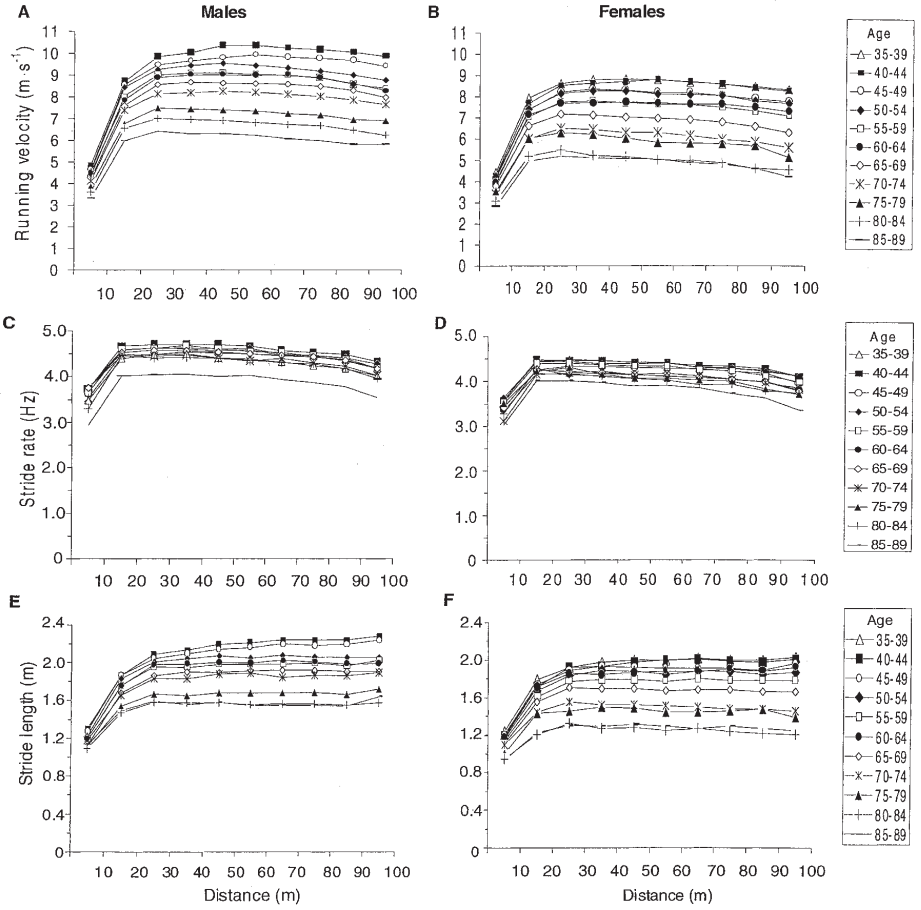

FIGURE 2—Mean values of velocity (A, B), stride rate (C, D), and stride length (E, F) during the 100-m run in each five-yr age group in male (left) and female (right) sprinters.

average 44% of stride time at 10.4 m·s^{-1} (40–44 yr) to 61% at 6.4 m·s^{-1} (85–88 yr) in males, and from an average 46% at 8.9 m·s^{-1} (35–39 yr) to 71% at 5.3 m·s^{-1} (80–87 yr) in females.

Significant correlations were found in both male and female runners between the velocities during peak velocity and final phases and CT during these phases (partial $r < -0.61$; $P < 0.001$ in all cases). However, there were no significant age-adjusted correlations between decrease in velocity from peak velocity sequence to final phase and the increase in CT from peak velocity sequence to final phase in either males or females.

Flight time. The individual values of FT during peak velocity are shown in Figures 3, A and B, and the age group differences in Tables 2 and 3. There were significant differences in FT values between the oldest and the other age groups.

When controlled for age, FT during peak velocity and deceleration phases was significantly related to velocity of these phases ($r = 0.48$; $P < 0.01$ in both cases) in females. In males, FT during the peak velocity phase was associated with the velocity of this phase ($r = 0.37$; $P < 0.05$), whereas no such correlation was observed during the deceleration phase. Furthermore, FT of the peak velocity and deceleration phases showed a significant correlation to SL of those phases in males (partial $r > 0.58$; $P < 0.001$ in both cases) and in females (partial $r > 0.68$; $P < 0.001$ in both cases).

DISCUSSION

There are a number of studies of the relationship between progressive velocity and stride pattern in the 100-m run in younger sprinters (1,3,4,8,10,25,26). However, to the authors' knowledge, the present study is the first to provide information of velocity and stride characteristics during each performance phase of the 100-m run in master sprinters. The major findings were as follows: 1) The sprinting velocity of elite male and female master athletes declined

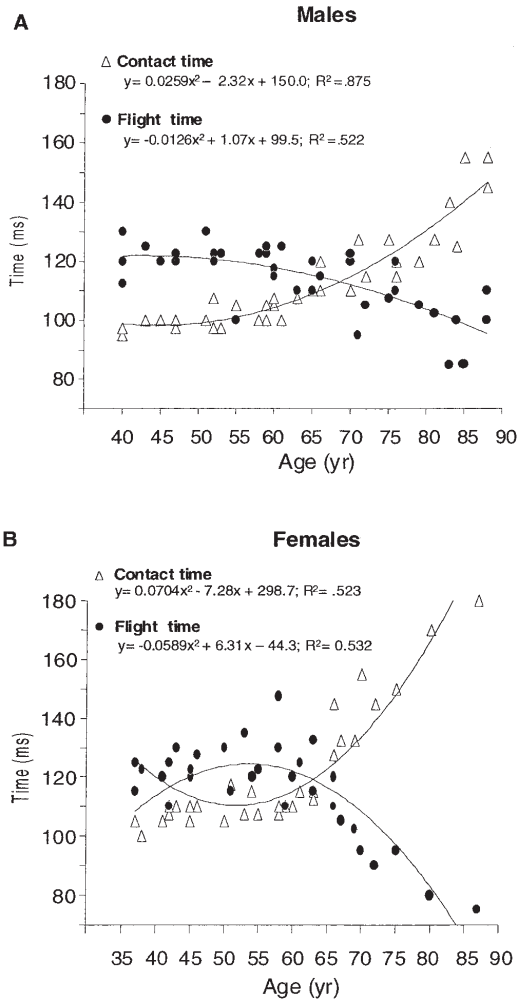

FIGURE 3—Individual values of contact and flight time during the fastest 10-m sequence as a function of age in male (A) and female (B) sprinters.

exponentially with age the differences becoming more evident after ~65–70 yr of age. 2) Age-associated differences in velocity were rather similar in each phase of the run in both genders. 3) The deterioration of the overall 100-m performance with age was primarily related to decrease in SL and increase in CT with advancing age in both male and female sprinters.

Velocity. Analysis of the acceleration phase of the run revealed that the time required to reach the peak velocity sequence remained unchanged, whereas the distance to peak velocity reduced significantly with age. Recent competition analysis (using laser devices) on young elite sprinters (28) indicated that the time required to reach peak velocity (males ~6.0–6.5 s, females ~5.0–6.5 s) is close to that found in the present study, but the distance to peak velocity (males 58–62 m, females 45–59 m) was, due to higher acceleration velocity, greater than that in these master sprinters.

The age-related decline in peak velocity in this study (~6% per decade) is close to the corresponding value of ~9% in Hamilton's study (11). Compared with elite young runners, the peak velocity achieved by the fastest male (40 yr) and female (41 yr) sprinters in this study were 13% and 14% lower than the highest reported peak velocity values in young male (12.05 $m \cdot s^{-1}$) and female (10.87 $m \cdot s^{-1}$) sprinters, respectively (1,7).

The decrease of velocity from peak velocity sequence to the end of the run for the fastest male and female sprinters in this study (5–6%) were about the same as corresponding values for young male (2–7%) and female runners (3–8%) in the recent major championships (1,3,7,25,28). When interpreting the effect of age on loss of velocity in the 100-m sprint it must be remembered that in older runners the peak velocity is achieved earlier, therefore the distance of deceleration phase is longer than in elite young runners.

Stride rate. The results of the current study indicate that SR has no major role in explaining the age-related decline in running velocity until the age of 80. However, after 80 yr of age there was a marked decline in SR, which seems to contribute to the decline in sprint performance in the oldest male and female age groups. Similarly, Hamilton's data (11) showed that SR remained steady from age 30 until the oldest age group.

The SR curves show three distinct phases. As illustrated in Figure 2, C and D, SR increases rapidly in the beginning of the race and the runners reach the maximum or near maximum SR already at 10–20 m. Thereafter, SR declines gradually toward the end of the run. In the last 10-m sequence, the decline in SR becomes more pronounced which is associated with a momentary increase in SL. Comparison of SR values of different velocity phases of the fastest runners in the current study with those values reported in major competitions (1,25) shows that the young sprinters are superior to master sprinters particularly in the ability to increase their SR during acceleration phase and in the ability to maintain high SR throughout the run. Interestingly, during the peak velocity sequence master sprinters had almost similar SR to elite young sprinters.

The ability to achieve high SR is thought to be affected by biological age and the development of the central nervous system (16). It has been reported that the highest running SR is reached already approximately at the age of 8 yr after which the trainability of SR becomes relatively limited (16). There is a paucity of data regarding to what extent the maximum SR in running or maximum frequency in other cyclic speed movements can be maintained with increasing age. Our findings and those of Hamilton (11) suggest that in competitive master sprinters the ability to achieve high frequency in cyclic whole-body sprinting movements does not change markedly before ~80 yr of age. However, it should be noted that SR is closely related to SL. As the sprinters in younger age groups were able to generate

greater SL, the air time increased, and consequently SR component was decreased. Due to this interdependence, the relatively small age-related decline in SR in these master sprinters may be partly influenced by changes in SL with age.

Stride length. The reduction in SL plays a decisive role in the age-related deterioration in 100-m sprint performance in these world-class master sprinters. This is clearly illustrated by findings that during each velocity phase SL declined markedly with age whereas SR showed smaller differences. Our findings are in agreement with the observations of Hamilton (11), who found that the decline in peak running velocity in master sprinters was accounted for by significant shortening of SL with increasing age. The SL values and the magnitude of the age-related decline in SL in Hamilton's study, ~6% per decade, are consistent with our results in the peak velocity phase.

SL curves in Figure 2, E and F, indicate that the changes in SL over the course of the 100-m race are quite similar in different age groups. During the acceleration phase of the run there was a large increase in SL followed by a phase of almost constant SL toward the end of the run. In the last 10-m sequence there is a slight increase in SL in males and in younger female sprinters, possibly due to a change in running technique before the finishing line. A closer inspection of SL curves indicate that the runners in younger age groups were able to increase SL for a longer distance with a greater SL than older runners. In relation to this, because SR reaches maximal value already at 10–20 m in all runners, the higher acceleration and the peak velocities found in younger sprinters seem to be explained by a higher ability to generate SL.

Previous findings of Ae et al. (1) indicate that the SL curves of the world's best male sprinters resemble in shape those of the fastest master sprinters in this study. However, compared with young sprinters, the absolute SL values of acceleration, peak velocity, and deceleration phases of the run were about 5%, 13%, and 11% lower, respectively, for master male sprinters (40–44 yr) and about 3%, 10%, and 10%, lower, respectively, for master female sprinters (35–39 yr). This comparison suggests that the ability to generate high SL during the peak velocity sequence, and to maintain it toward the end of the race, is an important characteristic of elite young sprinters and may explain the majority of the difference in velocity between master sprinters. The difference in the initial acceleration velocity seems to be related to differences both in SR and SL.

Because the deterioration in the sprint performance with age seems to be the most closely linked to the reduction in SL, the critical question is what influences SL. On the basis of knowledge available from younger subjects, it appears that SL in running is determined largely by the ability to develop great vertical forces during the ground contact phase (24,30). According to Mero and Komi (24), during maximum velocity of ~11 $m \cdot s^{-1}$, the sprinter has to produce a vertical force of more than three times his body weight, on one leg. Furthermore, because the contact phase in high-velocity sprinting lasts ~80–100 ms (and the propulsion phase only for ~60% of that time), the athlete must be capable of producing this force very rapidly. Related to this, a high percentage of fast-twitch (Type IIa, IIx) muscle fibers has shown to be predictive of great force production in young sprinters (23). To our knowledge, there is presently only one study (9) available on the impact of the aging process on the force production capacity in master national level sprinters and jumpers. This study demonstrated that vertical jump height in a standardized squat jump on a force platform decreased from ~0.33 m in 40- to 49-yr-olds to ~0.19 m in the athletes over 70. Also, the results of that study showed that the athletes over 70 were able to generate 58% of the maximum power output produced by 40- to 49-yr-old athletes. Similarly, evidence from untrained people has clearly indicated that explosive-strength characteristics of the leg muscles decline with age, especially from the sixth decade onward in both genders (2,15,19). This age-related decline in the muscle's ability to produce force rapidly seems to be primarily attributed to a decrease in muscle mass caused by a loss and an atrophy of fibers, in particular of Type II fibers (15,19,20). Also, it has been demonstrated that the decreased rate of force production with age may be affected by decrease of the nervous system's ability to activate muscles rapidly (14). However, it must be emphasized that running movements with repetitive part-phases impose unique demands on the force production capabilities of the neuromuscular system and thus the speculations of limiting factors for force production in sprinting based on acyclic single-repetition explosive movements is difficult. To understand the underlying mechanism for explaining the age-related reduction in force production and SL in sprint running, there is a need for sport-specific studies examining interrelationships between stride parameters, ground reaction forces, electromyography, and muscle fiber characteristics.

Ground contact time. CT was found to be an important stride parameter related to differences in velocity between age groups in both male and female runners. Consistent with the current results, studies have demonstrated that a short CT is a decisive factor for world-class performance among younger athletes (18,22,25). As an example, competition analysis at the World Championships by Moravec et al. (25) revealed that in the fastest male sprinters, CT during the peak velocity phase (11.6–11.8 $m \cdot s^{-1}$) were as low as 80–82 ms. Those values are 15% below the corresponding values for the fastest male sprinters (40–44 yr) in this study. Also, the sprint stride of young and master sprinters differs in time spent in contact and flight phases. In young male sprinters (25), the relative time spent in contact is ~38% compared with ~44% in the youngest male sprinters in our study. These comparisons suggest that the distinguishing feature of the elite young athletes is their ability to minimize CT while increasing FT and the length of the stride.

CT in sprint running is found to be associated with factors such as stride technique (18) and leg stiffness (17). For example, Kuitunen et al. (17) reported that high ankle joint stiffness was related to short CT. The great leg stiffness, in turn, is suggested to be dependent on preactivation of the muscles (24)

and on the stretch reflex potentiation during contact (6), which leads to a faster transition from the braking phase to the propulsion phase. Also, it is obvious that a shortened CT requires a rapid recruitment of the more powerful Type II motor units. Finally, a mechanical factor that is known to contribute to short CT is the small distance between body's center of gravity and first ground contact point (18). Whether there are age-related changes in leg stiffness, stretch reflex response, and muscle activation during stretch-shortening cycle type muscle action is not known yet.

Flight time. The present data showed that as running velocity decreased with age, there was a gradual decline in FT and SL. However, a closer inspection of individual data in Figure 3, A and B, indicate little or no decline in FT before age ~70 yr in males and ~65 yr in females. Therefore, it appears that in these masters sprinters across a range of velocity from ~5.3 $m \cdot s^{-1}$ to ~7.5 $m \cdot s^{-1}$, there is a rather linear increase in FT and SL with increasing running velocity, whereas at higher velocities (7.5–10.4 $m \cdot s^{-1}$) the increment in FT for a given increase in running velocity is proportionally smaller than that in SL. Thus, the interesting question is why FT does not change consistently as a result of change in running velocity and SL at higher velocities. Because FT is determined mainly by runner's resultant take-off velocity, it may be that until ~65–70 yr of age there were only small differences in impulses generated during propulsion portion of the contact phase. Regarding changes in CT with age, it seems that these equivalent impulses were achieved by different combinations of effective force and CT. For the faster younger runners, the impulse was achieved by the application of greater ground-reaction forces during briefer contact times, whereas older runners applied lesser ground forces during longer contact times (30). The possible explanation for the small or no change in FT, despite increases in SL as velocity is increased, could be the differences in take-off and landing characteristics of the stride (e.g., higher take-off angle in slower runners) (4).

Methodological considerations. This study has certain limitations in addition to its cross-sectional design. First, even though the present analysis was limited to the very best sprinters in each age group, we do not know the effect of genetic or nonphysiological factors on our findings. For example, based on our interview, the oldest female runners (70–87 yr) had not competed in athletics in their youth and may not have similar genetic constitution or training background to younger female sprinters. Second, the evaluation of the effect of age on biomechanical parameters is complicated by differences in the running velocity and height of the athletes. Studies on young runners have shown that SR and SL increase linearly at the lower velocities but at the higher velocities (~7.0–9.5 $m \cdot s^{-1}$) SR increases to a relatively greater extent than SL (21). Hoffmann (12,13) found that among younger sprinters, SR tended to increase whereas SL tended to decline as the height (and leg length) of the athlete decreased, and thus it is possible that some portion of differences in SR and SL with age in older runners could be explained by these factors. Finally, as only the very best sprinters were selected, there was a small number of subjects per group. Therefore, the results could be affected by sampling bias and Type II errors.

CONCLUSIONS

The results of the present study showed similar relative decline in velocity in all phases of the run with advancing age in elite master sprinters. The deterioration of overall 100-m performance with age was primarily related to reduction in stride length and increase in ground contact time. Insight into the nature of the decline in the sprinting ability with advancing age may, apart from being of basic scientific value, have implications for the planning of training programs for aging athletes.

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REFERENCES

1. Ae, M., M. Suzuki, T. Miyanishi, H. Okada, and T. Hirano. Analysis of racing patterns in 100 m sprint of the world's best top sprinters. In: *The Biomechanical Reports on 3rd IAAF World Championships in Athletics*, Biomechanical research groups for track and fields (Ed.). Tokyo: Baseball Magazine Sha (Co.), 1994, pp. 14–49.
2. Bosco, C., and P. V. Komi. Influence of aging on the mechanical behavior of leg extensor muscles. *Eur. J. Appl. Physiol.* 45:209–219, 1980.
3. Brüggemann, G.-P., and B. Glad. Time analysis of the sprint events. In: *Scientific Research Project at the Games of the XXIVth Olympiad-Seoul 1988: Final Report*, G-B. Brüggemann and B. Glad (Eds.). Monaco: International Athletic Foundation/International Amateur Athletic Federation, 1990, pp. 11–89.
4. Chow, J. W. Maximum speed of female high school runners. *Int. J. Sport Biomech.* 3:110–127, 1987.
5. Chow, J. W. A panning videographic technique to obtain selected kinematic characteristics of the strides in sprint hurdling. *J. Appl. Biomech.* 9:149–159, 1993.
6. Dietz, V., D. Schmidtbleicher, and J. Noth. Neuronal mechanisms of human locomotion. *J. Neurophysiol.* 42:1212–1222, 1979.
7. Ferro, A., A. Rivera, I. Pagola, et al. Biomechanical analysis of the 7th World Championships in Athletics Seville 1999. *New Studies in Athletics* 16:25–60, 2001.
8. Gajer, B., C. Thepaut-Mathieu, and D. Lehenaff. Evolution of stride and amplitude during course of the 100 m event in athletics. *New Studies in Athletics* 14:43–50, 1999.
9. Grassi, B., P. Cerretelli, M. V. Narici, and C. Marconi. Peak anaerobic power in master athletes. *Eur. J. Appl. Physiol.* 62:394–399, 1991.
10. Gundlach, H. Laufgeschwindigkeit und Schrittgestaltung im 100-m Lauf [Running speed and stride formation in 100-m run]. *Theorie und Praxis der Körperkultur* 12:346–359, 1963.
11. Hamilton, N. Changes in sprint stride kinematics with age in master's athletes. *J. Appl. Biomech.* 9:15–26, 1993.
12. Hoffmann, K. Stature, leg length, and stride frequency. *Track Technique* 46:1463–1469, 1971.
13. Hoffmann, K. Stride length and frequency of female sprinters. *Track Technique* 48:1522–1524, 1972.

14. Häkkinen, K., U.-M. Pastinen, R. Karsikas, and V. Linnamo. Neuromuscular performance in voluntary bilateral and unilateral contractions and during electrical stimulation in men at different ages. *Eur. J. Appl. Physiol.* 70:518–527, 1995.
15. Häkkinen, K., M. Alén, M. Kallinen, et al. Muscle CSA, force production, and activation of leg extensors during isometric and dynamic actions in middle-aged and elderly men and women. *JAPA* 6:232–247, 1998.
16. Joch, W. Dimensionen der motorischen Schnelligkeit [Dimensions of motor speed]. *Die Lehre der Leichtathletik* 27:1521–1528, 1988.
17. Kuitunen, S., P. V. Komi, and H. Kyröläinen. Knee and ankle joint stiffness in sprint running. *Med. Sci. Sports Exerc.* 34:166–173, 2002.
18. Kunz, H., and D. A. Kaufmann. Biomechanical analysis of sprinting: decathletes versus champions. *Br. J. Sports Med.* 15:177–181, 1981.
19. Larsson, L., G. Grimby, and J. Karlsson. Muscle strength and speed of movement in relation to age and muscle morphology. *J. Appl. Physiol.* 46:451–456, 1979.
20. Lexell, J., C. C. Taylor, and M. Sjöström. What is the cause of the ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year-old men. *J. Neurol. Sci.* 84:275–294, 1988.
21. Luhtanen, P., and P. V. Komi. Mechanical factors influencing running speed. In: *Biomechanics VI*, E. Asmussen and K. Jörgensen (Eds.). Baltimore: University Park Press, 1978, pp. 23–29.
22. Mann, R., and J. Herman. Kinematic analysis of Olympic sprint performance: men's 200 meters. *Int. J. Sport Biomech.* 1:151–162, 1985.
23. Mero, A., P. Luhtanen, J. T. Viitasalo, and P. V. Komi. Relationships between the maximal running velocity, muscle fiber characteristics, force production and force relaxation of sprinters. *Scand. J. Sports Sci.* 3:16–22, 1981.
24. Mero, A., and P. V. Komi. Force-, EMG-, and elasticity-velocity relationship at submaximal, maximal and supramaximal running speeds in sprinters. *Eur. J. Appl. Physiol.* 55:553–561, 1986.
25. Moravec, P., J. Ruzicka, P. Susanka, et al. The 1987 International Athletic Foundation/IAAF Scientific Project Report: time analysis of the 100 metres events at the II World Championships in Athletics. *New Studies in Athletics* 3:61–96, 1988.
26. Murase, Y., T. Hoshikawa, N. Yasuda, Y. Ikegami, and H. Matsui. Analysis of the changes in progressive speed during 100-meter dash. In: *Biomechanics V-B*, P. V. Komi (Ed.). Baltimore: University Park Press, 1976, pp. 200–207.
27. Records Committee of the World Masters Athletics. World track and field outdoor age-group records. *Natl. Masters News* April:16–17, 2002.
28. Ritzdorf, W. 100 meters. In: Biomechanical Research Project at the VIth World Championships in Athletics, Athens 1997: preliminary report, H. Müller and H. Hommel (Eds.). *New Studies in Athletics* 12:43–73, 1997.
29. Spirduso, W. W. The physically elite elderly. In: *Physical Dimensions of Aging*, W. W. Spirduso (Ed.). Champaign, IL: Human Kinetics, 1995, pp. 389–417.
30. Weyand, P. G., D. B. Sternlight, M. J. Bellizzi, and S. Wright. Faster top running speeds are achieved with greater ground forces not more rapid leg movements. *J. Appl. Physiol.* 89:1991–1999, 2000.

PAPER II

BIOMECHANICAL AND SKELETAL MUSCLE DETERMINANTS OF MAXIMUM RUNNING SPEED WITH AGING

by

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Biomechanical and Skeletal Muscle Determinants of Maximum Running Speed with Aging

II

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[1]Department of Health Sciences, University of Jyväskylä, FINLAND; [2]Department of Biology of Physical Activity, University of Jyväskylä, FINLAND; [3]Kuopio University Hospital, Department of Physical and Rehabilitation Medicine, Kuopio, FINLAND; [4]KIHU - Research Institute for Olympic Sports, Jyväskylä, FINLAND; [5]The Finnish Centre for Interdisciplinary Gerontology, Jyväskylä, FINLAND; [6]Department of Medical Rehabilitation, Oulu University Hospital and Institute of Health Sciences, University of Oulu, Oulu, FINLAND; and [7]Pajulahti Sports Center, Nastola, FINLAND

ABSTRACT

KORHONEN, M. T., A. A. MERO, M. ALÉN, S. SIPILÄ, K. HÄKKINEN, T. LIIKAVAINIO, J. T. VIITASALO, M. T. HAVERINEN, and H. SUOMINEN. Biomechanical and Skeletal Muscle Determinants of Maximum Running Speed with Aging. *Med. Sci. Sports Exerc.*, Vol. 41, No. 4, pp. 844–856, 2009. **Purpose**: Aging diminishes the ability to run fast, but the specific mechanisms responsible for this deterioration remain largely unknown. In the present study, we investigated the age-related decline in sprint running ability through a cross-sectional examination of biomechanical and skeletal muscle characteristics in 77 competitive male sprinters aged 17–82 yr. **Methods**: Ground reaction force (GRF) and kinematic stride cycle parameters were measured during the maximum-velocity phase using a 9.4-m-long force platform. Knee extensor (KE) and ankle plantar flexor (PF) structural characteristics were investigated using ultrasonography and muscle biopsies (vastus lateralis). Force production characteristics of leg extensor muscles were determined by dynamic and isometric contractions. **Results**: The main findings were as follows: 1) the progressive age-related decline in maximum running velocity (V_{max}) was mainly related to a reduction in stride length (L_{str}) and an increase in ground contact time (t_c), whereas stride frequency showed a minor decline and swing time remained unaffected; 2) the magnitude of average braking and push-off resultant GRFs declined with age and associated with L_{str}, t_c, and V_{max}; 3) there was an age-related decline in muscle thickness, Type II fiber area and maximal and rapid force-generating capacity of the lower limb muscles; and 4) muscle thickness (KE + PF) was a significant predictor of braking GRF, whereas the countermovement jump height explained most of the variance in push-off GRF in stepwise regression analysis. **Conclusions**: Age-related slowing of maximum running speed was characterized by a decline in stride length and an increase in contact time along with a lower magnitude of GRFs. The sprint-trained athletes demonstrated an age-related selective muscular atrophy and reduced force capacity that contributed to the deterioration in sprint running ability with age. **Key Words:** LOCOMOTION, MASTER ATHLETE, MUSCLE FIBER, SPRINT RUNNING, STRENGTH

APPLIED SCIENCES

The ability to run fast is a major factor determining level of performance in various sporting and recreational physical activities. However, with aging, the ability to perform short sprints declines and may eventually inhibit participation in physical activities. Given the growing number of people remaining highly physically active into older age, information on the mechanisms responsible for the decline in maximal locomotor performance may be of increasing overall importance and have implications for the planning of exercises to preserve and potentially improve speed performance in older people. Accordingly, examination of the specific biomechanical and physiological changes that impair maximum running velocity (V_{max}) in older athletes is needed to understand the degree to which these changes are preventable by regular training. So far, studies of age-related changes in V_{max} have focused mainly on stride cycle parameters (12,20). However, stride parameters do not provide information on the factors underlying changes in performance; hence, it would be important to determine the interrelationship between stride cycle parameters, ground reaction forces (GRFs), and neuromuscular factors.

The two basic mechanisms that determine V_{max} are the forces generated to the ground and the speed at which the legs can be swung forward and backward (36). On the basis of studies in young adults, V_{max} is primarily related to the magnitude of contact forces that the runners are capable of producing during the short contact phase. For example, in the study by Weyand et al. (36), a 1.8-fold intersubject difference

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in V_{max} (from 6.2 to 11.1 $m \cdot s^{-1}$) and 1.7-fold difference in stride length were attributable to a 1.3-fold change in vertical GRF. The faster runners also had higher stride frequency. However, this was achieved by reduced contact time, whereas the time taken to swing the limb into the position for the next stride did not vary between runners. These findings are in line with the studies by Mero (25) and Mero and Komi (26) that used resultant GRF as a specific force indicator in young male and female runners with different sprinting abilities. The higher V_{max} and longer strides of the fastest runners were explained by their superior ability to produce greater ground contact forces.

Skeletal muscle characteristics have been commonly identified as critical determinants of sprint performance. Investigations in young athletes have suggested that sprinting ability is limited by muscle mass (3,23,35), fiber type composition (5,25,27), and fascicle length (3,23). However, it is not uncommon to observe wide variation in muscle properties among athletes of similar performance level (2,27). This suggests that the ability to run fast depends on a combination of factors. In addition to muscle structural characteristics, several studies have shown that whole-muscle mechanical performance is associated with sprinting ability (11,39). On the other hand, the association between muscle strength and sprint performance may be influenced, among other factors, by the type of contraction. For example, there is some evidence to suggest that rapid muscle force capacity is more closely related to sprint performance than maximal strength (9,22). However, the extent to which these findings in young sprint athletes apply to older runners remains unknown.

Accordingly, the main purpose of this study was to examine specific biomechanical characteristics that may determine the ability to run at maximum speed with increasing age. To address this aim, we measured both GRF and stride cycle parameters in male sprinters aged 17–82 yr. The secondary aim was to identify age-related differences in the morphological and mechanical properties of the lower limb muscles and to determine the relationship between the muscle characteristics and sprint performance.

METHODS

Subjects. This study was part of a larger investigation on aging, speed performance, and skeletal muscle characteristics (19). Eighteen young adult (17–33 yr) and 59 elite master (40–82 yr) male sprinters were chosen for the present study. The subjects arrived from all over Finland. All 77 athletes were included in sprint performance tests, but because of the subjects' decisions not to complete certain assessment tasks (fear of injury, timing of testing not suitable for the current training/competition preparation schedule), the muscle strength and structural characteristics data were obtained from 58 to 77 subjects. The missing data were mainly from the youngest age group. The subjects had achieved good national- or international-level results in 100- to 400-m sprint events and were continuing to train and compete systematically. The runners in different age groups had similar relative performance level: the 60-m running times in this study ranged from 109 ± 3% (17–33 yr) to 106 ± 4% (70–82 yr) of the age-group world record times. Furthermore, there were no significant age-group differences in the reported all-time personal best 100-m times (ranged from 10.96 ± 0.28 s in the 17–33 yr age group to 11.46 ± 0.30 s in the 70–82 yr age group, $n = 37$). A questionnaire indicated a decrease in total training hours (from 10.6 to 6.4 $h \cdot wk^{-1}$, $P < 0.001$) and sessions (from 5.7 to 3.9 times per wk, $P < 0.001$) from the youngest to the oldest group, whereas training years increased with age (from 12.8 to 27.3 yr, $P < 0.05$). With increasing age, the number of hours spent on speed and strength training decreased (from 5.3 to 3.1 $h \cdot wk^{-1}$, $P < 0.001$, and from 4.8 to 1.0 $h \cdot wk^{-1}$, $P < 0.001$, respectively), whereas the amount of other exercises (aerobic running, cross-country skiing, ball games) tended to increase with age (from 0.5 to 2.3 $h \cdot wk^{-1}$, $P = 0.08$). A written consent was obtained from all subjects. The Ethics Committee of the University of Jyväskylä, in conformity with the Declaration of Helsinki, approved the study.

Anthropometry. Body height was measured with a height gauge, and body mass was measured with a balance beam scale. Total body fat percentage was assessed using bioelectrical impedance (Spectrum II; RJL Systems, Detroit, MI). Upper leg length was determined as the distance from the lateral condyle of the femur to the greater trochanter, and lower leg length was determined as the distance between the lateral malleolus of the fibula and the lateral condyle of the femur. Leg length was calculated as the sum of the upper and lower leg lengths and was used for normalization of the biomechanical variables.

Ultrasonographic measurements. Knee extensor (KE) and ankle plantar flexor (PF) morphological characteristics were measured using B-mode ultrasonography (SSD-1400; Aloka, Tokyo, Japan), as described earlier (21,23), with slight modifications. Briefly, a 5-cm linear-array probe (7.5 MHz) was positioned perpendicular to the surface of the muscle, and in the ultrasonic images, the subcutaneous adipose tissue layer, superior and inferior aponeurosis, and fascicles between the aponeuroses were identified. The thickness of the KE muscles (vastus lateralis (VL), vastus medialis (VM), vastus intermedius (VIM), rectus femoris (RF)) and PF muscles (gastrocnemius medialis (GM), gastrocnemius lateralis (GL)) was determined as the distance from the adipose tissue–muscle interface to the intermuscular interface. The measurements for the VM were taken at 30% and those for the VL, VIM, and RF at 50% of the distance between the lateral condyle of the femur and the greater trochanter (for VIM two locations: under VL and under RF), whereas the anatomic site for the GM and GL at 30% proximal between the lateral malleolus of the fibula and the lateral condyle of the tibia. The sum of the muscle thicknesses of all the knee extensor and plantar flexor muscles (KE + PF muscle thickness) was used as an indicator of

muscle mass. The pennation angle was measured as the angle between the fascicle and the deep aponeuroses, and fascicle length was estimated from the muscle thickness and pennation angle as follows: Fascicle length = Muscle thickness × sin (pennation angle)$^{-1}$. Fascicle pennation angle and length were determined from the VL, GM, and GL. The ratios of VL fascicle length to upper leg length and of GM and GL to lower leg length were also examined. The interday repeatability (coefficient of variation, CV%) of the muscle architectural measurements using this method has previously been shown to be 2.5% for muscle thickness, 3.8% for pennation angle, and 5.0% for fascicle length (21). In addition, previous studies have provided evidence that muscle thickness, as determined by ultrasonography, is a good estimate for muscle volume (28) and muscle mass (32).

Muscle biopsy and histochemical analyses. Details of the muscle sampling procedure and myofibrillar ATPase histochemical analyses have been described earlier (19). Briefly, muscle biopsy samples were taken from the middle portion of the vastus lateralis of the dominant leg (take-off foot in jumping events). Serial cryosections (10 μm) were stained for myofibrillar ATPase after acid (pH 4.37, 4.60) and alkaline (pH 10.30) preincubations. The number and cross-sectional area of the various fiber types were analyzed using a microscope combined with a computer-assisted image analysis system (Tema; Scanbeam, Hadsund, Denmark). For the purpose of this study, the fibers were divided into Types I and II fibers. Fiber-type characteristics were calculated from an average of 492 fibers (range, 89–1100) in each biopsy sample.

Strength testing. Dynamic and isometric strength of the lower limb muscles was determined as described previously (8,19). Briefly, one repetition maximum (1-RM) dynamic strength of the leg extensors was measured using a concentric half-squat exercise (from 90° knee angle) in the Smith machine (Frapp fitness, Joensuu, Finland). Dynamic speed–strength ability was assessed by a countermovement jump (CMJ) on a contact mat. Maximum vertical jump height was assessed by determining the rise of the body's center of gravity (calculated from flight time). During the test, the hands were kept on the hips to minimize differences in technique.

Bilateral isometric force of the leg extensors was measured by an electromechanical dynamometer (University of Jyväskylä) with the subject seated with knees and hip at 107° and 110° flexion, respectively. On a verbal command, the subjects performed an isometric leg extension as hard and as fast as possible during 2.5–4 s. Maximal force was defined as the highest force value recorded, and maximal rate of force development (RFD), a measure of isometric speed-strength quality, was defined as the greatest increase in force in a given 5-ms period using 1-ms moving intervals (Δforce/Δtime). Furthermore, specific force (maximal force/KE thickness) and specific RFD (maximal RFD/KE thickness) were determined to provide an insight into the muscles' intrinsic force/speed generation capacities. The subjects performed approximately three to four trials for each strength test (up to five trials in squat 1-RM) until their performance no longer improved.

Sprint measurements. The subjects ran a maximal 60-m sprint twice on an indoor synthetic track with spiked running shoes (rest between runs of 7 min). The athletes started from a static forward–lean standing position with the front leg 70 cm behind the starting line (first photocell gates). Further, to minimize the aging effect on starting actions (reaction time, technique), own start without commands were used. Vertical and horizontal anteroposterior GRFs and timing parameters were measured during the maximal speed phase (from 30 m onward) using a special 9.4-m-long force platform system. It consisted of nine tartan-surface force plates (five two-dimensional and three three-dimensional force plates, 0.9/1.0 m; natural frequency, ≥170 Hz; nonlinearity, ≤1%; cross talk, ≤2%, TR test, Finland; and one Kistler 3-dimensional force plate, 0.9/0.9 m; natural frequency, 400 Hz; Honeycomb, Kistler, Switzerland) connected in series. The force signals were sampled at 1000 Hz and stored on a microcomputer via an AT Codas A/D converter card (Dataq Instruments, Inc., Akron, OH).

The maximum 10-m running velocity (30–40 m) and the 60-m trial time (t_{60m}) were obtained by four pairs of double-beam photocell gates. In addition, a laser radar (Laveg Sport; Jenoptik, Jena, Germany), positioned 6 m behind the start line, was used to analyze the instantaneous running speeds of the athletes. The results indicated that 91% of the athletes had reached their V_{max} by the 40-m mark (range, 19–48 m; mean 30 ± 6 m), and those athletes who continued to accelerate up to 40–48 m achieved 99% of their V_{max} by the 40-m mark (data not shown). Thus, the 30- to 40-m distance used for force platform measurements represented the phase of maximum velocity sprinting for all the athletes.

The force platform data were analyzed using custom-written software (University of Jyväskylä, Finland). The vertical and horizontal GRFs were integrated with respect to time phases and were then combined to obtain the average resultant GRFs. A horizontal GRF curve was used to divide the force components into the braking and push-off phases (26). Time of total contact (t_c), braking phase (t_{brake}), push-off phase (t_{push}), swing (t_{sw}), and aerial phase (t_{aer}) were obtained from the force platform records (Fig. 1). Stride cycle time (t_{str}) was defined as the time between consecutive footfalls of the same foot, and stride frequency ($Freq_{str}$) is the inverted value of the stride cycle time [$1/t_{str}$, (= step frequency / 2)] (36). Duty factor was defined as time of contact relative to entire stride cycle duration (t_c/t_{str}). Stride length (L_{str}), the distance between successive contacts of the same foot (step length × 2), was measured from spike marks on a thin paper sheet firmly attached over the force platform. In previous measurements in young sprinters, the accuracy of this method has been ±2 cm compared with film analysis (A. M., unpublished observations).

Each trial involved four to six contacts on the force platform. For consistency, the first four contacts of the two

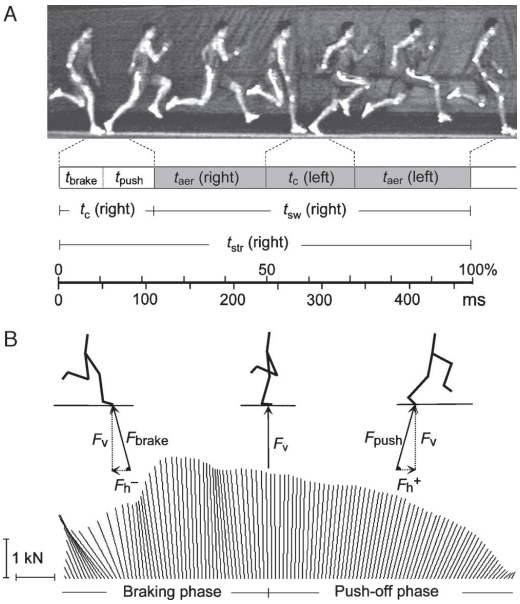

FIGURE 1—Description of the components of the stride cycle (A) and resultant force vector diagram (B) that reflects the changes in magnitude and orientation of the GRF through contact. Stride cycle parameters were defined according to Weyand et al. (36). t_c = contact time, t_{aer} = aerial time, t_{sw} = swing time. F_{brake} = resultant GRF of the braking contact phase, F_{push} = resultant GRF of the push-off contact phase, F_v = vertical GRF, F_h^- = horizontal braking GRF, F_h^+ = horizontal push-off GRF.

trials were taken for the analyses. Because no significant bilateral differences were found in the biomechanical variables, the results for the right and left sides were averaged for each subject. The intrasubject variability of the biomechanical variables was assessed for 18 young (24 ± 4 yr) and 25 older (70 ± 4 yr) runners. All variables had a coefficient of variation (calculated from a total of 8 contacts/4 strides) of 0.9%–6.0%. No age-related differences were observed in the CVs.

The amplitudes of the resultant GRFs (F_{brake} and F_{push}) are given as average net forces relative to body weight ($F = (N - N_{bw}) / kg_{bw}$), and the direction of GRF are given as the mean angle of the average net resultant GRF in the braking and push-off phases (25,26). Further, to examine the RFD applied to the ground, the F_{brake} and F_{push} were divided by the respective t_{brake} and t_{push} (expressed as $N \cdot s^{-1} \cdot kg^{-1}$). Leg length showed significant correlations with $Freq_{str}$ ($r = -0.39$, $P < 0.001$), L_{str} ($r = 0.39$, $P < 0.001$), and t_{sw} ($r = 0.28$, $P < 0.05$) when controlled for speed (partial correlation). Because there was an age-related difference in leg length (Table 1), both absolute and relative leg length–adjusted L_{str}, $Freq_{str}$, and t_{sw} (i.e., divided by 1/leg length) were analyzed. Figure 1 illustrates the biomechanical parameters examined in this study.

Statistical analyses. Linear and quadratic regression analyses were performed to determine the association between sprint performance and muscle morphological and functional characteristics with age (Figs. 2–6). The mean percent changes in selected variables across the age range (% per decade) were calculated from the difference in the mean values of the youngest, 17–33 yr (mean, 23.8 yr), and the oldest, 70–82 yr (mean, 74.9 yr), groups. ANOVA was used to determine differences between 10-yr age groups. In the event of significant age effect, Tukey's *post hoc* test was used to identify the significant differences between each pair of age groups. The mean statistical power for detecting significant ($P < 0.05$) age effect in various comparisons was 0.95. Three parameters showed power values less than 0.80 ($F_{push\text{-}angle}{}^{\circ}$ = 0.78; pennation angle of VL = 0.74; Leg extension specific RFD = 0.75). Pearson's correlation coefficient was used to examine the relationships between continuous variables, and where appropriate, partial correlation was used to control the effect of age in the relationship. Stepwise multiple regression analyses were conducted to find out the combination of muscle characteristics and age that explained the most variance in V_{max} and GRFs and to examine the association of F_{push}, F_{brake}, and age with V_{max}, L_{str}, and t_c. Statistical significance was set at $P < 0.05$ for all analyses.

RESULTS

Subject characteristics. There was an age-related decline in body mass ($r = -0.40$, $P < 0.001$), body height ($r = -0.63$, $P < 0.001$), and leg length ($r = -0.55$, $P < 0.001$), whereas percent body fat increased with age ($r = 0.30$, $P < 0.01$). Leg length relative to height did not correlate with age (Table 1).

TABLE 1. Selected physical characteristics of subjects in different age groups.

Variable	Age Group				
	17–33 yr	40–49 yr	50–59 yr	60–69 yr	70–82 yr
n	18	14	15	16	14
Age (yr)	23.8 ± 4.2	44.4 ± 3.6	53.8 ± 2.8	65.6 ± 2.3	74.9 ± 3.8
Body mass (kg)	77.5 ± 5.9[e]	80.2 ± 8.4[d,e]	73.1 ± 5.4	70.8 ± 5.9[b]	70.0 ± 9.2[a,b]
% body fat	16 ± 3[e]	18 ± 3	18 ± 3	17 ± 4	21 ± 4[a]
Height (m)	1.80 ± 0.04[c,d,e]	1.82 ± 0.07[c,d,e]	1.74 ± 0.04[a,b,e]	1.73 ± 0.04[a,b]	1.68 ± 0.05[a,b,c]
Leg length (m)	0.87 ± 0.03[d,e]	0.88 ± 0.05[c,d,e]	0.84 ± 0.03[b]	0.83 ± 0.03[a,b]	0.82 ± 0.04[a,b]
Leg length (% height)	48.6 ± 1.2	48.6 ± 1.5	48.4 ± 1.2	48.0 ± 1.1	48.5 ± 1.1

Values are means ± SD. *n*, number of subjects.
[a,b,c,d,e] Group is significantly ($P < 0.05$) different from the 17- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 82-yr-old groups, respectively.

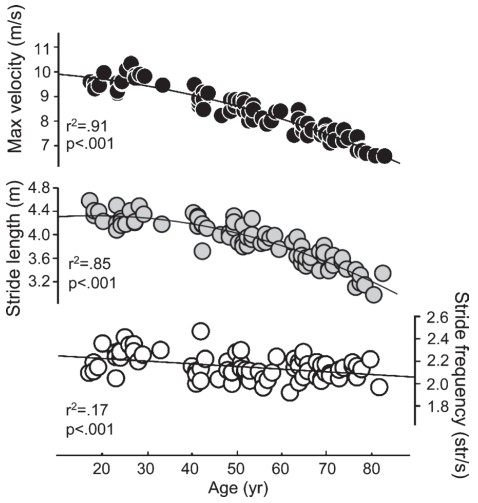

FIGURE 2—Relationships between age and maximum running velocity (V_{max}), stride length (L_{str}), and stride frequency ($Freq_{str}$). L_{str} was determined as the distance between the consecutive contacts of the same foot (step length × 2) and $Freq_{str}$ from the inverse of the total stride cycle time [$1/t_{str}$, (= step frequency / 2)]. Regression equations: $V_{max} = 10.02 - 0.007x - 0.0004x^2$; $L_{str} = 4.23 + 0.01x - 0.0003x^2$; $Freq_{str} = 4.55 - 0.005x$ (all $n = 77$).

Biomechanical parameters. The 60-m running times increased from 6.98 ± 0.17 s in 17- to 33-yr-old runners to 9.23 ± 0.41 s among 70- to 82-yr olds ($P < 0.001$; +6% per decade; Appendix 1 for age group comparisons). There was a progressive age-related decline in V_{max} (−5% per decade) and L_{str} (−4% per decade; Fig. 2), which both became significant by the 40- to 49-yr age group in comparison with the youngest group (Appendix 1). $Freq_{str}$ also showed a reduction with age (−1% per decade; Fig. 2). However, after the significant decline from the youngest (2.25 Hz) to the 40- to 49-yr group (2.13 Hz), $Freq_{str}$ remained the same until the oldest group (2.12 Hz; Appendix 1). When expressed relative to leg length, the age-related decline remained significant for L_{str} ($r = -0.77$, $P < 0.001$; −3% per decade) but not for $Freq_{str}$ ($r = 0.10$; Appendix 1).

Of the temporal variables (Fig. 3), there was an age-related increase in t_c (+5% per decade), t_{brake} (+9% per decade), and t_{push} (+2% per decade) and a decrease in t_{aer} (−2% per decade), whereas t_{sw} remained unchanged. However, t_{sw} normalized to leg length increased with age ($r = 0.44$, $P < 0.001$, +1% per decade). The t_{push}/t_{brake} ratio showed nonlinear reduction with age ($r^2 = 0.17$, $P < 0.001$), reaching significance for the oldest group (Appendix 1). Duty factor (t_c/t_{str}) increased with age ($r = 0.73$, $P < 0.001$).

The GRFs of the braking (F_{brake}) and push-off (F_{push}) phases declined gradually with age (−4% per decade and −6% per decade, respectively; Fig. 4A). The F_{push}/F_{brake} ratio showed lower values in the 70- to 82-yr-old group (Fig. 4B). The mean angle of F_{push} became more vertically oriented with age, whereas no age effect existed in the braking GRF angle (Fig. 4C). The age-associated increase in the mean angle of F_{push} reached significance only in the 60- to 69-yr-old group (Appendix 1). Further, the decline in GRFs along with increase in contact times with age led to clear reductions in the rate of GRF development in both braking (F_{brake}/t_{brake}: −9% per decade) and push-off phases (F_{push}/t_{push}: −8% per decade).

Muscle morphology. There was an age-related decline in KE (−6% per decade), PF (−2.5% per decade), and KE + PF muscle thickness (−5.5% per decade; Fig. 5; Appendix 2). When compared with the youngest groups, KE and KE + PF thickness showed a decline by age 50–59 yr and thereafter, whereas the age group differences in PF thickness did not reach significance (Appendix 2).

The mean cross-sectional area of Type I fibers showed no significant association with age, but a reduction in Type II fiber area (−7% per decade) and Type II-to-I fiber area ratio (−4.5% per decade) was observed (Fig. 5B). The relative number of Types I and II fibers did not correlate with age.

Pennation angle showed a progressive age-related decline for VL ($r = -0.43$, $P < 0.001$) but was not associated with age for GM or GL. For VL, a significant decline in pennation angle was observed in the oldest age group in comparison with the youngest group (Appendix 2).

There were no significant age-related differences in fascicle length for VL, GM, or GL. Furthermore, fascicle length relative to leg length did not correlate with age in the VL, GM, or GL.

Muscle strength. There was an age-related decline in maximal dynamic strength (concentric half-squat 1-RM,

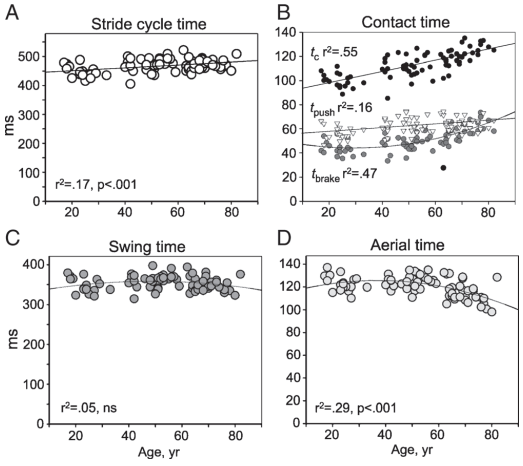

FIGURE 3—Relationships between age and stride cycle time, t_{str} (A), total contact time, t_c, braking time, t_{brake}, push-off time (*triangles*), t_{push} (B), swing time, t_{sw} (C), and aerial time, t_{aer} (D). $t_{str} = 441.3 + 0.49x$; $t_c = 89.6 + 0.45x$, $P < 0.001$; $t_{brake} = 51.1 - 0.48x + 0.008x^2$, $P < 0.001$, $t_{push} = 54.6 + 0.14x$, $P < 0.001$, $t_{sw} = 327.45 + 1.27x - 0.013x^2$; $t_{aer} = 113.1 + 0.68x - 0.009x^2$ (all $n = 77$).

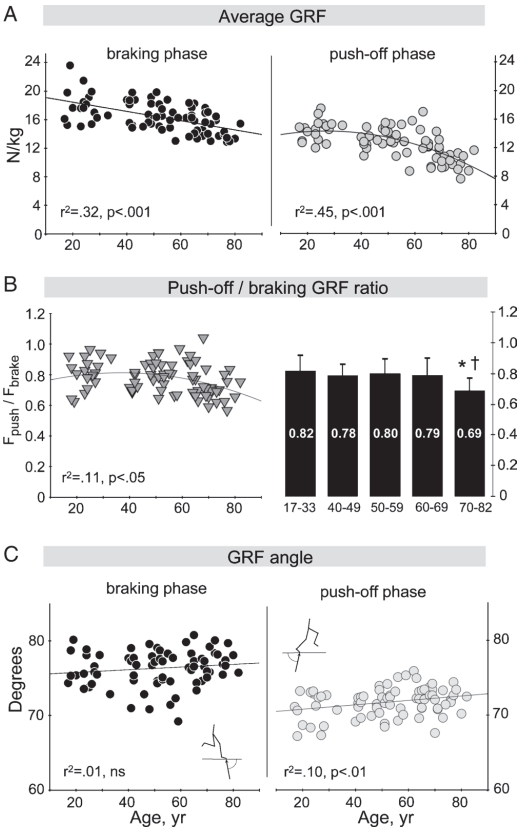

FIGURE 4—Relationships between age and the average net resultant GRF of the braking (F_{brake}) and push-off (F_{push}) contact phases (A), F_{push} as a fraction of F_{brake} (B), and mean angle of the resultant GRF from vertical, averaged over the braking (F_{brake}°) and push-off (F_{push}°) phases (C). $F_{brake} = 19.77 - 0.07x$; $F_{push} = 13.07 + 0.09x - 0.002x^2$; $F_{push}/F_{brake} = 0.725 + 0.005x - 7E - 005x^2$; $F_{brake}° = 75.7 + 0.013x$; $F_{push}° = 69.9 + 0.033x$ (all $n = 77$). *$P < 0.05$ versus 17- to 33-yr-old group, †$P < 0.01$ versus 50- to 59-yr-old group.

−9% per decade), maximal isometric force (bilateral leg extension, −8% per decade), vertical jump height (CMJ, −11% per decade), and maximal rate of isometric force development (leg extension RFD, −10% per decade; Fig. 6; Appendix 3). Isometric force normalized to KE muscle thickness was not associated with age ($r = -0.17$), whereas the isometric RFD/KE thickness ratio declined with age ($r = -0.37$, $P < 0.01$). ANOVA of RFD/KE thickness ratio showed an overall decline in with age ($P < 0.05$), but in the *post hoc* analysis, the differences between the age groups did not reach significance (Appendix 3).

Relationships among biomechanical parameters. Table 2 shows the simple and age-adjusted correlations between the biomechanical parameters. In the overall sample, V_{max} was significantly associated with all parameters, except t_{sw}. Of the parameters, L_{str}, t_c, t_{brake}, and F_{push} were the best correlates of V_{max}. When controlled for age, most of the significant correlations between the parameters remained, with t_c showing the strongest relationship with V_{max}.

Stepwise multiple regression analysis, with the inclusion of F_{push}, F_{brake}, and chronological age in the models, was used to determine the association of GRFs with V_{max}, L_{str}, and t_c. Age and F_{push} jointly explained 91% and 81%, respectively, of the variance in V_{max} and L_{str}. For the t_c, age, F_{push}, and F_{brake} were all significant predictors and together explained 70% of the total variance in t_c. When age was intentionally excluded from these regression models, both F_{push} and F_{brake} entered in the equations and together accounted for 53%, 46%, and 61%, respectively, of the variance in V_{max}, L_{str}, and t_c.

Relationships among muscle morphology, strength, and biomechanical parameters. Table 3 shows the age-adjusted correlations among the muscle structure, strength, and running parameters. KE + PF thickness was associated with V_{max} and t_{60m}. No other morphological characteristics were related to running parameters (data not shown). Of the muscle strength measures, squat 1-RM, CMJ, and maximal isometric force correlated with the sprint parameters, but no associations were found between isometric RFD and sprint performance (not shown).

Table 4 shows the stepwise regression models of the muscle predictors for V_{max}, F_{brake}, and F_{push}. The variables entered were KE + PF thickness, Type II fiber percentage, Type II-to-I fiber area ratio, maximal isometric force, CMJ, and chronological age ($n = 54$). Age was the strongest predictor of V_{max} and explained 89% of the total variance, with maximal isometric force being the only other factor (1%) to appear in the model. When age was intentionally excluded from the model, CMJ and KE + PF thickness appeared in the model and together explained 81% of the variance in V_{max} (not shown).

In the GRFs, KE + PF thickness was the only variable to enter the model for F_{brake} (26%), whereas maximum CMJ height was the only significant predictor of F_{push} (34%). Age did not enter the models for F_{brake} and F_{push}.

DISCUSSION

The present study showed that in competitive male sprinters the slowing of maximum running speed with age was characterized by a decline in stride length and an increase in ground contact time along with a lower magnitude of GRFs. The athletes demonstrated age-related changes in muscle structure and force production capacity of the lower limb muscles that contributed to the deterioration in sprint running performance. Figure 7 shows a simplified diagram summarizing the observed biomechanical and skeletal muscle changes with age that may be connected to the decline in sprinting ability.

Kinematic stride cycle parameters. On the first level of the mechanical analysis, we found that a decrease in

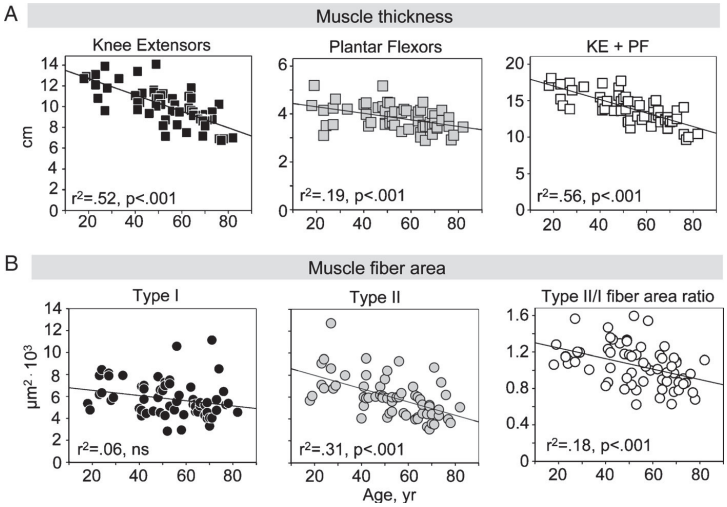

FIGURE 5—Relationships between age and knee extensor (KE) and ankle plantar flexor (PF) muscle thickness (A), mean cross-sectional areas of the vastus lateralis muscle fibers (B). KE muscle thickness = 14.29 − 0.079*x* (*n* = 58); PF muscle thickness = 4.56 − 0.013*x* (*n* = 58); KE + PF muscle thickness = 18.86 − 0.093*x* (*n* = 58); Type I fiber area = 7031 − 23.7*x*; Type II fiber area = 9199 − 60.8*x*; Type II-to-type I fiber area ratio = 1.36 − 0.006*x* (fiber data, all *n* = 65).

L_{str} contributed more to the decline in V_{max} than $Freq_{str}$ (Fig. 2). Further, the reduction in $Freq_{str}$ was associated entirely with increased t_c because t_{sw} did not vary between runners of different ages. These results are in line with age-related changes in stride cycle parameters in previous studies (12,20). However, because differences in body size may influence stride variables (13), we also adjusted L_{str} and $Freq_{str}$ to leg length. The decline with age was still evident for L_{str} but did not persist for $Freq_{str}$. This suggests that it was primarily the L_{str} aspect of velocity that was affected by age.

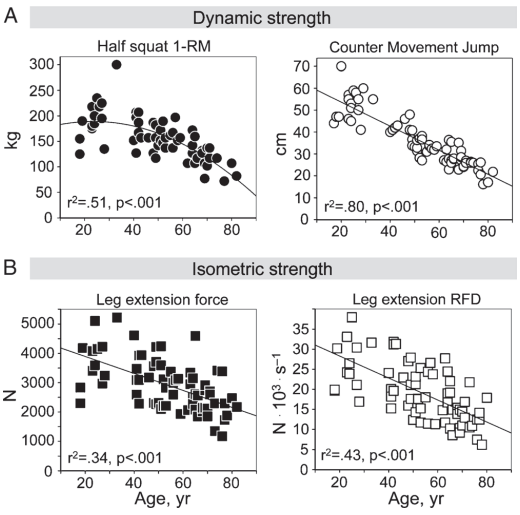

FIGURE 6—Relationships between age and dynamic (A) and isometric (B) strength measures. Half-squat 1-RM = 171 + 1.55*x* − 0.033x^2 (*n* = 62); CMJ height = 64.7 − 0.55*x* (*n* = 77); maximal force of isometric leg extension = 4473 − 29*x* (*n* = 72); maximal rate of force development (RFD) of isometric leg extension = 33,798 − 275*x* (*n* = 71).

Some studies have proposed that the ability to reposition the limbs in the air is an important determinant of sprinting speed (34). However, other studies have challenged this view and concluded that, despite having a more powerful muscle profile, a faster sprint specialist has a similar minimum swing duration to that of slower runners (36). In this connection, it is believed that the mechanical energy required for the repositioning of the swinging leg can occur passively through elastic recoil and energy transfer between body segments rather than by power generated within muscles (36). Our results showed a lack of difference in t_{sw} between the young and older runners (Fig. 3C). However, when t_{sw} was corrected for dimensional changes (i.e., divided by leg length), a significant increase in swing duration was observed in the three oldest age groups in comparison to the youngest group. Therefore, it cannot be ruled out that t_{sw} or the ability to rotate the legs backward and forward is a contributing factor in determining sprinting speed in older age groups. An evaluation of the swing phase from other points of view, e.g., segment and joint angular velocities/accelerations during forward and backward swing phases, might provide further information on this topic. For example, studies involving young elite sprinters have indicated that differences in swing-back velocity immediately before ground contact can explain differences in maximum velocity between runners (4).

TABLE 2. Simple and age-controlled correlations among the biomechanical variables.

Variables	V_{max}	L_{str}	$Freq_{str}$	t_c	t_{brake}	t_{push}	t_{sw}	t_{aer}	F_{brake}
				Pearson Correlation Coefficient					
Maximum velocity (V_{max})									
Stride length (L_{str})	**0.89‡**								
Stride frequency ($Freq_{str}$)	**0.53‡**	0.09							
Contact time (t_c)	**−0.84‡**	**−0.62‡**	**−0.70‡**						
Braking time (t_{brake})	**−0.73‡**	**−0.61‡**	**−0.48‡**	**0.81‡**					
Push-off time (t_{push})	**−0.42‡**	−0.21	**−0.53‡**	**0.57‡**	−0.02				
Swing time (t_{sw})	−0.11	**0.33†**	**−0.85‡**	**0.27***	0.08	**0.35***			
Aerial time (t_{aer})	**0.40‡**	**0.69‡**	**−0.41‡**	**−0.35†**	**−0.41‡**	0.03	**0.79‡**		
Braking GRF (F_{brake})	**0.59‡**	**0.61‡**	**0.26***	**−0.66‡**	**−0.58‡**	**−0.32†**	0.11	**0.52‡**	
Push-off GRF (F_{push})	**0.71‡**	**0.65‡**	**0.33†**	**−0.74‡**	**−0.83‡**	−0.10	0.08	**0.54‡**	**0.63‡**
	V_{max}	L_{str}	$Freq_{str}$	t_c	t_{brake}	t_{push}	t_{sw}	t_{aer}	F_{brake}
				Partial Correlation Adjusted for Age					
Maximum velocity (V_{max})									
Stride length (L_{str})	**0.35†**								
Stride frequency ($Freq_{str}$)	**0.48‡**	**−0.65‡**							
Contact time (t_c)	**−0.65‡**	0.12	**−0.65‡**						
Braking time (t_{brake})	**−0.56‡**	−0.15	**−0.32†**	**0.67‡**					
Push-off time (t_{push})	−0.14	**0.33†**	**−0.44‡**	**0.45‡**	**−0.36‡**				
Swing time (t_{sw})	**−0.29***	**0.73‡**	**−0.93‡**	**0.39‡**	0.09	**0.38†**			
Aerial time (t_{aer})	−0.02	**0.74‡**	**−0.71‡**	−0.04	−0.20	0.17	**0.88‡**		
Braking GRF (F_{brake})	0.21	0.15	0.04	**−0.44‡**	**−0.35†**	−0.13	0.14	**0.37‡**	
Push-off GRF (F_{push})	**0.44‡**	**0.28***	0.10	**−0.53‡**	**−0.73‡**	0.21	0.12	**0.39‡**	**0.44‡**

Values are correlations (r).
Significant correlations are in bold (* $P < 0.05$, † $P < 0.01$, ‡ $P < 0.001$).

Ground reaction forces. The present study demonstrated that the magnitudes of GRFs in sprinting are reduced substantially with age (Fig. 4A) and reflected in changes in L_{str}, t_c, and, consequently, in V_{max}. Our findings are in general agreement with those of previous studies that have examined GRF and stride characteristics in young runners with different sprinting abilities (18,25,26,36). For instance, the treadmill running study by Weyand et al. (36) found that V_{max} (1.8-fold difference: 6.2–11.1 $m \cdot s^{-1}$) was highly sensitive to a small variation (1.26-fold difference) in vertical forces. Higher vertical forces had a positive effect on both the maximal L_{str} and minimal t_c that runners were able to achieve.

In this investigation, resultant GRF was used as a specific force indicator, thereby providing an insight into the interaction between vertical and horizontal force components. Although vertical force dominates the resultant GRF (Fig. 1B) and is likely to have a strong effect on the minimum time needed to be spent on the ground to produce sufficient vertical impulse to support body weight and to create t_{aer} long enough for repositioning the swing leg, horizontal GRFs may also play a major role in attaining higher sprinting speed (15). Our results indicated a small but significant change in the mean angle of F_{push} (i.e., decrease in the horizontal push-off/vertical GRF ratio) in older runners (Fig. 4C). However, to what extent these age differences in GRF direction, even minor, may impair the acceleration of the body in the optimal horizontal direction and thus affect sprint velocity is unknown at present.

The present study also indicated an age-related decline in F_{push}/F_{brake} as well as t_{push}/t_{brake} ratios, but these decrements were evident only in the oldest group (Fig. 4B). It could be hypothesized that high eccentric impact loads are less tolerated at older ages, resulting in a longer braking phase and a decreased elastic energy/force potentiation during the concentric phase. The consideration of suboptimal stretch-shortening cycle action during ground contact of running in elderly men seems to be supported by the recent study of Cavagna et al. (7). Their results indicated a reduced elastic recovery (−20%) in old (74 ± 6 yr) compared with young (21 ± 2 yr) subjects running at moderate speeds (4.2–4.7 $m \cdot s^{-1}$). More detailed analysis of the effects of age on support leg movements (14) and spring-like function of muscle-tendon complex (31) during maximum speed running awaits further studies.

Muscle morphology and sprint performance. Studies in young athletes have shown that although sprinters vary in body height, they are typically heavier and have greater muscle mass along with a faster fiber-type profile in their leg muscles than middle- and long-distance

TABLE 3. Age-adjusted correlations between selected biomechanical variables and muscle structural and strength characteristics.

	V_{max}	L_{str}	$Freq_{str}$	t_c	F_{brake}	F_{push}	t_{60m}
				Partial Correlation Adjusted for Age			
KE + PF thickness	**0.28***	0.16	0.11	−0.08	0.22	0.10	**−0.26***
Half-squat 1-RM	**0.33†**	−0.04	**0.30***	**−0.28***	−0.00	0.14	**−0.34†**
CMJ height	**0.34†**	0.04	0.22	**−0.23***	0.00	0.17	**−0.28***
Maximum isometric force	**0.24***	0.12	0.09	−0.04	0.13	0.16	−0.18

Values are correlations (r).
Significant correlations are in bold (* $P < 0.05$, † $P < 0.01$, ‡ $P < 0.001$).
See Table 2 and text for abbreviations.

TABLE 4. Stepwise multiple-regression analysis for the skeletal muscle factors predicting maximum sprinting velocity (V_{max}), and the average net resultant GRF (related to body weight) in of the braking (F_{brake}) and push-off (F_{push}) contact phases.

Dependent Variable	Predictors	Cumulative r^2	P
V_{max}	Age	0.88	<0.001
	Maximal isometric force	0.89	0.012
F_{brake}	KE + PF muscle thickness	0.26	<0.001
F_{push}	CMJ height	0.34	<0.001

runners (5,16). Furthermore, the fascicle length and pennation angle may vary according to running event specialization (3) and could predict overall sprint performance in sprinters (2,23). The present results imply that the decline in the contractile force and velocity potential of muscle with age were mainly attributable to reductions in muscle thickness and Type II fiber area (Fig. 5) because the muscle fiber-type distribution and the muscle architectural characteristics remained largely unchanged. Furthermore, on the basis of our earlier study (19), regular sprint training does seem to reduce the typical age-related decrease in the single-fiber mechanical properties (specific force, shortening velocity) and thus might not be a factor in the deterioration in the strength and sprint performance in these athletes.

The combined KE + PF muscle thickness was the strongest predictor of F_{brake} in the regression analysis, explaining 26% of the total variance (Table 4). It is worth noting that the lower body weight in older sprinters is partially caused by age-related loss of muscle mass, and the present approach of controlling the average net GRFs for body mass (i.e., $(N - N_{bw}) / kg_{bw}$) may underestimate "true" prediction of muscle thickness to GRFs. When the average net GRFs were used in the stepwise regression analysis, muscle thickness showed an increased prediction for F_{brake} (50%) and was also a primary predictor for F_{push} (54%) with CMJ playing a secondary role (5%; data not shown). Hence, in aging sprinters, muscle volume does seem to play a significant role in the ability to tolerate the great contact forces needed to achieve higher sprinting speeds. This view is consistent with the results of a recent study by Weyand and Davis (35) that estimated the vertical force requirements for different velocities in young male and female athletes. It

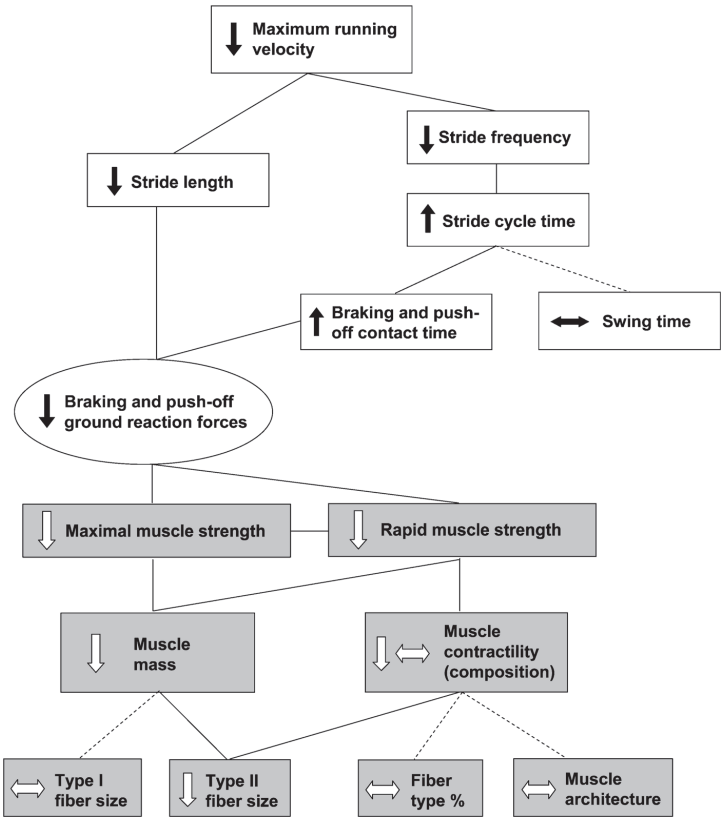

FIGURE 7—Simplified diagram of the selected biomechanical and skeletal muscle factors that may determine maximum running speed with aging. *Arrows* represent significant age-related decrease (↓), increase (↑) and no alteration (↔) in the variables, as indicated by the regression analyses (Figs. 2–6).

was concluded that, for runners of same stature and body composition, the larger muscle masses of the faster sprint specialists are directly related to the greater ground contact forces necessary for attaining higher velocities (35).

Muscle strength and sprint performance. Maximal dynamic and isometric leg strength as well as CMJ performance and rate of isometric force development showed a gradual age-related decline (Fig. 6). When isometric force production was normalized to leg muscle thickness, the age-associated decline disappeared for maximal force but remained true for RFD. This slowing of isometric RFD is consistent with our observation of age-related preferential atrophy of fast fibers, although other factors such as rapid neural activation of motor units may also play a role in well-trained older athletes (29). On the basis of the training data, possibly the deterioration in muscle function and associated neuromuscular properties is affected by the lack of proper strength training needed for the effective stimulation of fast motor units. This assumption is also consistent with our recent results (8), indicating selective hypertrophy of Type II fibers along with improved rapid neural activation of muscle, when supplementing sprint training with maximal and explosive strength exercises in elite older sprinters.

Among the muscle functional parameters, CMJ was the only predictor of GRFs, explaining 34% of the variance in F_{push} (Table 4). Several studies in young athletes have also indicated that the CMJ performance is related to overall sprint times, V_{max}, and stride cycle parameters (9,22,27,39). Apparently, despite many performance dissimilarities (e.g., acyclic vertical vs cyclic horizontal), CMJ, to some extent, simulates fast stretch–shorten cycle contraction of sprint stride contact. On the other hand, the level of prediction of CMJ for F_{push} (34%) as well as the age-adjusted associations of the maximal and rapid strength measures with sprint performance were weak in this study (r = 0.23–0.36; Table 3), which is in contrast to many (27,38,39) but not all (37) studies in young athletes. One possible explanation for these low correlations between strength qualities and sprint performance could be that, with age, sprint performance becomes relatively more dependent on technical factors. For example, the elderly sprinters may show greater limb movement constraints (decreased range of motion) because of reduced joint function, which would directly affect stride length (12).

Specificity of training adaptations. Previous findings in untrained (24) and strength-trained (6,30) men have suggested that the age-related decline in rapid muscle force/power–generating capacity occurs at a higher rate than that in maximal isometric and dynamic strength. For example, the study by Pearson et al. (30) among elite master Olympic weightlifters (40–87 yr) and age-matched controls indicated that the rate of loss in maximal isometric knee extension force (−5% to −6% per decade) was approximately half of that observed for leg extension muscle power (−12% to −13% per decade). To address this issue, we calculated changes in strength measures and sprinting velocity on the basis of the values observed in the young athlete group (Fig. 8). Our data

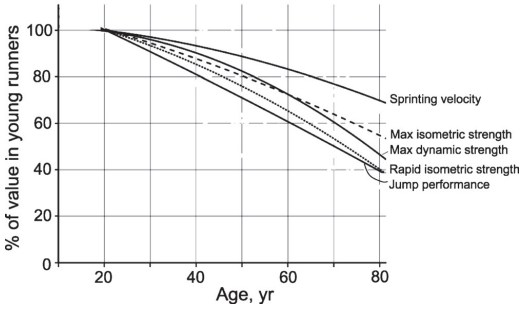

FIGURE 8—Age-related changes in sprint and strength characteristics expressed as a percentage of the value for the young athlete group. Maximum sprinting velocity = V_{max}; max isometric strength = maximal force of bilateral leg extension; max dynamic strength = concentric half-squat 1-repetition maximum strength; rapid isometric strength = maximal RFD during bilateral leg extension; jump performance = maximum CMJ height.

suggest that in continuously sprint-trained athletes, the decline in rapid isometric force and vertical jumping capacity (∼−10% to −11% per decade) proceeds only a slightly faster than that in maximal strength (∼−8% to −9% per decade). Furthermore, it was somewhat unexpected that sprint performance (V_{max} and 60-m time), which is likely to impose higher requirements on the integration of muscle force production and neuromuscular coordination than the present simple strength tasks, was the least affected by age in these sprinters (∼−5% to −6% per decade). This result seems to confirm the concept of high training specificity and the adaptability of the neuromuscular performance characteristics during aging, and that in large part, the decline in performance in older people is due to a reduction in specific exercise stimulus rather than aging *per se*. This knowledge is encouraging for aging people who aim to exercise regularly with specific training modes with a view to continuing their higher-intensity sporting and recreational physical activities.

Limitations. Because of the cross-sectional nature of the study, the findings may have been affected by genetic and constitutional factors. Longitudinal studies are needed to provide more definitive insights into the age-related change in maximum running speed. A potential confounding factor is the assessment of muscle thickness by ultrasonography because this method is unable to distinguish between muscle and intramuscular fat. However, this may not significantly influence our results because the amount of intramuscular fat in the calf muscles of these master sprinters was minimal when examined by computer tomography (H. S., unpublished observations). Nevertheless, a more reliable assessment of muscle mass loss warrants the use of other techniques, such as magnetic resonance imaging, and should also focus on other sprint-specific muscle groups (knee flexors, hip extensors, hip flexors). Although we recruited highly competitive sprinters to minimize the effect of physical activity on our measures, the fact that there were age-related reductions in training volume, especially in strength training, may have contributed to the rate

of decline in variables. A similar trend of overall reduction in training stimulus with age has been noted in many studies on endurance athletes and may be explained by age-related reductions in motivation and "intrinsic drive" to train intensively (33) and/or decreased trainability due to physiological changes, e.g., impaired recovery and muscle repair processes (10). Finally, a limitation of the study is that there were incomplete data sets for muscle structure and strength tests. Although no bias is expected because of homogeneity of the athletes in different age groups, the sprint performance predictions on the basis of muscle characteristics could be improved with a larger sample size.

CONCLUSIONS

The findings of the present study suggest that the age-related decline in maximum velocity sprinting is primarily related to reduction in stride length and increase contact time secondary to decreased ability to generate ground contact forces. Selective muscular atrophy and reduced maximal and rapid muscle force capacity all could contribute to the age-related deterioration in maximal running performance in sprint-trained athletes.

Valuable information on the effects of aging and long-term training on skeletal muscle and physical performance characteristics can be obtained by studying master athletes from different sports. A smaller decrease in sprinting speed compared with strength performance in these athletes supports the concept of training specificity and the favorable effect of regular sprint training on complex locomotor skills during aging (Fig. 8). The present data indicate, however, limited benefit of sprint training alone in prevention of Type II muscle fiber atrophy. This result suggests that the degree of recruitment of fast motor units by short-duration, high-velocity contractions are insufficient for maintaining fast muscle mass and force production. Given the present as well as other findings (1,17) on the effect of long-term aerobic versus resistance training on muscle characteristics, optimal training should also include intensive strength exercises.

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REFERENCES

1. Aagaard P, Magnusson PS, Larsson B, Kjaer M, Krustrup P. Mechanical muscle function, morphology, and fiber type in lifelong trained elderly. *Med Sci Sports Exerc*. 2007;39(11):1989–96.
2. Abe T, Fukashiro S, Harada Y, Kawamoto K. Relationship between sprint performance and muscle fascicle length in female sprinters. *J Physiol Anthropol Appl Human Sci*. 2001;20(2):141–7.
3. Abe T, Kumagai K, Brechue WF. Fascicle length of leg muscles is greater in sprinters than distance runners. *Med Sci Sports Exerc*. 2000;32(6):1125–9.
4. Ae M, Ito A, Suzuki M. The men's 100 meters. *New Stud Athl*. 1992;7(1):47–52.
5. Andersen JL. Muscle fiber type characteristics of the runner. In: Bangsbo J, Larsen HB, editors. *Running & Science*. Copenhagen (Denmark): Munksgaard; 2001. p. 49–65.
6. Anton MM, Spirduso WW, Tanaka H. Age-related declines in anaerobic muscular performance: weightlifting and powerlifting. *Med Sci Sports Exerc*. 2004;36(1):143–7.
7. Cavagna GA, Legramandi MA, Peyre-Tartaruga LA. Old men running: mechanical work and elastic bounce. *Proc Biol Sci*. 2008;275(1633):411–8.
8. Cristea A, Korhonen MT, Häkkinen K, et al. Effects of combined strength and sprint training on regulation of muscle contraction at the whole-muscle and single fibre levels in elite master sprinters. *Acta Physiol (Oxf)*. 2008;193:275–89.
9. Cronin JB, Hansen KT. Strength and power predictors of sports speed. *J Strength Cond Res*. 2005;19(2):349–57.
10. Fell J, Williams D. The effect of aging on skeletal-muscle recovery from exercise: possible implications for aging athletes. *J Aging Phys Act*. 2008;16(1):97–115.
11. Häkkinen K, Keskinen KL. Muscle cross-sectional area and voluntary force production characteristics in elite strength- and endurance-trained athletes and sprinters. *Eur J Appl Physiol Occup Physiol*. 1989;59(3):215–20.
12. Hamilton N. Changes in sprint stride kinematics with age in master's athletes. *J Appl Biomech*. 1993;9:15–26.
13. Hoffmann K. Stature, leg length, and stride frequency. *Track Technique*. 1971;46:1463–9.
14. Ito A, Fukuda K, Kijima K. Mid-phase movements of Tyson Gay and Asafa Powell in the 100 metres at the 2007 World Championships in Athletics. *New Stud Athl*. 2008;23(2):39–43.
15. Keränen T, Nummela A. Ground reaction forces at submaximal and maximal running speeds. In: *Strength Training for Sport, Health, Aging and Rehabilitation. 5th International Conference on Strength Training; Oct 18–21*. Denmark: University of Southern Denmark; 2007. p. 344–5.
16. Khosla T. Standards on age, height and weight in Olympic running events for men. *Br J Sports Med*. 1978;12(2):97–101.
17. Klitgaard H, Mantoni M, Schiaffino S, et al. Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. *Acta Physiol Scand*. 1990;140(1):41–54.
18. Kobayashi K, Tsuchie H, Matsuo A, Fukunaga T, Kawakami Y. Changes in sprint performance and kinetics during acceleration phase of running of a world record holder. In: *The 13th Annual Congress of the European College of Sport Science; July 9–12*. Estoril (Portugal); 2008. p. 591.
19. Korhonen MT, Cristea A, Alen M, et al. Aging, muscle fiber type, and contractile function in sprint-trained athletes. *J Appl Physiol*. 2006;101(3):906–17.
20. Korhonen MT, Mero A, Suominen H. Age-related differences in 100-m sprint performance in male and female master runners. *Med Sci Sports Exerc*. 2003;35(8):1419–28.
21. Kubo K, Kanehisa H, Azuma K, et al. Muscle architectural characteristics in young and elderly men and women. *Int J Sports Med*. 2003;24(2):125–30.

22. Kukolj M, Ropret R, Ugarkovic D, Jaric S. Anthropometric, strength, and power predictors of sprinting performance. *J Sports Med Phys Fitness*. 1999;39(2):120–2.
23. Kumagai K, Abe T, Brechue WF, Ryushi T, Takano S, Mizuno M. Sprint performance is related to muscle fascicle length in male 100-m sprinters. *J Appl Physiol*. 2000;88(3):811–6.
24. Lauretani F, Russo CR, Bandinelli S, et al. Age-associated changes in skeletal muscles and their effect on mobility: an operational diagnosis of sarcopenia. *J Appl Physiol*. 2003;95(5):1851–60.
25. Mero A. Electromyographic activity, force and anaerobic energy production in sprint running with special reference to different constant speeds ranging from submaximal to supramaximal [PhD dissertation]. Jyväskylä (Finland): University of Jyväskylä; 1987.
26. Mero A, Komi PV. Force-, EMG-, and elasticity-velocity relationships at submaximal, maximal and supramaximal running speeds in sprinters. *Eur J Appl Physiol*. 1986;55(5):553–61.
27. Mero A, Luhtanen P, Viitasalo JT, Komi PV. Relationships between the maximal running velocity, muscle fiber characteristics, force production and force relaxation in sprinters. *Scand J Sports Sci*. 1981;3(1):16–22.
28. Miyatani M, Kanehisa H, Ito M, Kawakami Y, Fukunaga T. The accuracy of volume estimates using ultrasound muscle thickness measurements in different muscle groups. *Eur J Appl Physiol*. 2004;91(2–3):264–72.
29. Ojanen T, Rauhala T, Häkkinen K. Strength and power profiles of the lower and upper extremities in master throwers at different ages. *J Strength Cond Res*. 2007;21(1):216–22.
30. Pearson SJ, Young A, Macaluso A, et al. Muscle function in elite master weightlifters. *Med Sci Sports Exerc*. 2002;34(7):1199–206.
31. Roberts TJ. The integrated function of muscles and tendons during locomotion. *Comp Biochem Physiol A Mol Integr Physiol*. 2002;133(4):1087–99.
32. Sanada K, Kearns CF, Midorikawa T, Abe T. Prediction and validation of total and regional skeletal muscle mass by ultrasound in Japanese adults. *Eur J Appl Physiol*. 2006;96(1):24–31.
33. Tanaka H, Seals DR. Endurance exercise performance in masters athletes: age-associated changes and underlying physiological mechanisms. *J Physiol*. 2008;586(1):55–63.
34. van Ingen Schenau GJ, de Koning JJ, de Groot G. Optimisation of sprinting performance in running, cycling and speed skating. *Sports Med*. 1994;17(4):259–75.
35. Weyand PG, Davis JA. Running performance has a structural basis. *J Exp Biol*. 2005;208(Pt 14):2625–31.
36. Weyand PG, Sternlight DB, Bellizzi MJ, Wright S. Faster top running speeds are achieved with greater ground forces not more rapid leg movements. *J Appl Physiol*. 2000;89(5):1991–9.
37. Wilson GJ, Murphy AJ. The use of isometric tests of muscular function in athletic assessment. *Sports Med*. 1996;22(1):19–37.
38. Wisloff U, Castagna C, Helgerud J, Jones R, Hoff J. Strong correlation of maximal squat strength with sprint performance and vertical jump height in elite soccer players. *Br J Sports Med*. 2004;38(3):285–8.
39. Young W, McLean B, Ardagna J. Relationship between strength qualities and sprinting performance. *J Sports Med Phys Fitness*. 1995;35(1):13–9.

APPENDIX 1
Biomechanical Characteristics of Running by Age Groups

	Age Group				
Variables	**17–33 yr**	**40–49 yr**	**50–59 yr**	**60–69 yr**	**70–82 yr**
n	18	14	15	16	14
t_{60m} (s)	6.98 ± 0.17*	7.63 ± 0.30*	7.99 ± 0.31*	8.54 ± 0.28*	9.23 ± 0.41*
V_{max} ($m \cdot s^{-1}$)	9.65 ± 0.31*	8.80 ± 0.33*	8.34 ± 0.33*	7.73 ± 0.28*	7.09 ± 0.37*
L_{str} (m)	4.30 ± 0.14*	4.14 ± 0.17*	3.94 ± 0.14*	3.65 ± 0.15*	3.35 ± 0.19*
L_{str}/L_{leg} ($m \cdot m^{-1}$)	4.93 ± 0.18*	4.69 ± 0.20[a,d,e]	4.67 ± 0.17[a,d,e]	4.41 ± 0.23*	4.12 ± 0.24*
$Freq_{str}$ (Hz)	2.25 ± 0.10*	2.13 ± 0.12[a]	2.12 ± 0.10[a]	2.12 ± 0.10[a]	2.12 ± 0.07[a]
$Freq_{str}/L_{leg}$ ($Hz \cdot m^{-1}$)	2.58 ± 0.16	2.42 ± 0.24	2.52 ± 0.17	2.57 ± 0.14	2.60 ± 0.15
t_c (ms)	100 ± 6*	111 ± 10[a,e]	112 ± 9[a,e]	116 ± 7[a,e]	127 ± 6*
t_{brake} (ms)	44 ± 6[d,e]	48 ± 8[e]	50 ± 6[e]	52 ± 9[a,e]	63 ± 5*
t_{push} (ms)	56 ± 6[b,d,e]	64 ± 7[a]	62 ± 5	64 ± 6[a]	63 ± 5[a]
t_{push}/t_{brake}	1.32 ± 0.24[e]	1.38 ± 0.30[e]	1.26 ± 0.17	1.29 ± 0.42	1.01 ± 0.15[a,b]
t_{sw} (ms)	347 ± 19	360 ± 19	361 ± 16	355 ± 20	347 ± 14
t_{sw}/L_{leg}	398 ± 22[c,d,e]	409 ± 24	428 ± 19[a]	429 ± 31[a]	426 ± 27[a]
t_{aer} (ms)	123 ± 7[e]	125 ± 5[e]	125 ± 7[e]	119 ± 9[e]	110 ± 8*
t_{str} (ms)	447 ± 20*	471 ± 24[a]	473 ± 21[a]	472 ± 22[a]	473 ± 15[a]
t_c/t_{str}	0.224 ± 0.009*	0.236 ± 0.010[a,e]	0.236 ± 0.012[a,e]	0.247 ± 0.013[a,e]	0.267 ± 0.013*
F_{brake} $N\text{-}N_{bw}$	1389 ± 190[c,d,e]	1374 ± 196[d,e]	1213 ± 142[a,e]	1120 ± 132[a,b]	1015 ± 204[a,b,c]
F_{brake} $(N\text{-}N_{bw}) \cdot kg^{-1}$	18.1 ± 2.2[d,e]	17.2 ± 1.8[e]	16.4 ± 1.7[e]	15.8 ± 1.7[a]	14.4 ± 1.4[a,b,c]
RFD_{brake} $(N \cdot s^{-1} \cdot kg^{-1})$	415 ± 60[c,d,e]	375 ± 100[e]	336 ± 63[a,e]	320 ± 105[a,e]	231 ± 33*
F_{push} $(N\text{-}N_{bw})$	1123 ± 143[c,d,e]	1068 ± 115[d,e]	963 ± 143[a,e]	875 ± 123[a,b,e]	694 ± 113*
F_{push} $(N\text{-}N_{bw}) \cdot kg^{-1}$	14.5 ± 1.5[d,e]	13.4 ± 1.3[e]	13.0 ± 1.8[e]	12.4 ± 1.9[a,e]	9.9 ± 1.1*
RFD_{push} $(N \cdot s^{-1} \cdot kg^{-1})$	259 ± 38*	211 ± 25[a,e]	215 ± 36[a,e]	193 ± 23[a,e]	158 ± 23*
F_{push}/F_{brake}	0.82 ± 0.10[e]	0.78 ± 0.08	0.80 ± 0.10[e]	0.79 ± 0.12	0.69 ± 0.08[a,c]
$F_{brake\text{-}angle}$ (°)	76.3 ± 2.2	76.0 ± 2.8	75.5 ± 2.9	76.9 ± 1.9	77.3 ± 1.6
$F_{push\text{-}angle}$ (°)	70.6 ± 2.2[d]	71.4 ± 1.4	71.9 ± 2.1	72.8 ± 1.6[a]	71.6 ± 1.7

Values are means ± SD. *n*, number of subjects.
* ,a,b,c,d,e Group is significantly ($P < 0.05$) different from all other age groups, 17- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 82-yr-old groups, respectively.
See Figure 1 and text for the description of the variables.

APPENDIX 2
Muscle Structural Characteristics by Age Groups

Variables	Age Group				
	17–33 yr	40–49 yr	50–59 yr	60–69 yr	70–82 yr
Muscle thickness (cm)	n = 9	n = 12	n = 12	n = 15	n = 10
KE	12.2 ± 1.3[c,d,e]	11.1 ± 1.4[c,d,e]	9.6 ± 1.3[a,b]	9.3 ± 1.2[a,b]	8.1 ± 1.2[a, b]
PF	4.1 ± 0.6	4.1 ± 0.5	3.9 ± 0.4	3.6 ± 0.45	3.6 ± 0.4
KE + PF	16.3 ± 1.5[c,d,e]	15.2 ± 1.5[c,d,e]	13.5 ± 1.4[a,b,e]	13.0 ± 1.2[a,b]	11.7 ± 1.5[a,b,c]
Fascicle length (cm)	n = 9	n = 12	n = 12	n = 15	n = 10
VL	8.1 ± 2.4	8.0 ± 1.3	8.0 ± 2.5	8.1 ± 1.5	7.9 ± 1.4
GM	6.2 ± 1.8	6.2 ± 1.4	5.4 ± 1.6	6.1 ± 1.1	5.0 ± 0.6
GL	7.9 ± 2.5	8.3 ± 2.4	7.2 ± 2.2	7.4 ± 1.4	6.5 ± 1.4
Relative fascicle length ($cm \cdot cm^{-1}$)	n = 9	n = 12	n = 12	n = 15	n = 10
VL/upper leg length	0.18 ± 0.05	0.17 ± 0.03	0.18 ± 0.06	0.18 ± 0.03	0.18 ± 0.03
GM/lower leg length	0.15 ± 0.05	0.15 ± 0.04	0.14 ± 0.04	0.16 ± 0.03	0.14 ± 0.02
GL/lower leg length	0.19 ± 0.06	0.20 ± 0.06	0.19 ± 0.06	0.20 ± 0.04	0.17 ± 0.03
Pennation angle (°)	n = 9	n = 12	n = 12	n = 15	n = 10
VL	19.3 ± 5.2[e]	17.7 ± 3.4	16.0 ± 4.8	15.3 ± 3.2	13.2 ± 3.3[a]
GM	23.1 ± 4.5	23.1 ± 4.7	25.8 ± 6.3	20.3 ± 4.2	24.7 ± 5.3
GL	13.6 ± 2.6	12.9 ± 3.7	14.5 ± 3.6	12.4 ± 3.1	13.7 ± 4.6
Fiber CSA (μm^2)	n = 11	n = 14	n = 14	n = 15	n = 11
Mean	7400 ± 1600[d,e]	5950 ± 1100	5900 ± 1600	5200 ± 1300[a]	5300 ± 1800[a]
Type I	6800 ± 1300	5600 ± 1200	5850 ± 2100	5300 ± 1150	5700 ± 2300
Type II	7900 ± 2000[c,d,e]	6300 ± 1300	5950 ± 1600[a]	5150 ± 1600[a]	5050 ± 1700[a]
Type II-to-I CSA ratio	1.17 ± 0.15[e]	1.15 ± 0.22[e]	1.08 ± 0.27	0.96 ± 0.18	0.91 ± 0.15[a,b]
Type II (%)	52 ± 13	52 ± 13	54 ± 8	53 ± 13	60 ± 12

Values are means ± SD. n, number of subjects.
[a,b,c,d,e] Group is significantly ($P < 0.05$) different from the 17- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 82-yr-old groups, respectively.
CSA, cross-sectional area; KE, sum of thickness of knee extensor muscles (vastus lateralis (VL), vastus medialis, rectus femoris (RF), vastus intermedius (two locations: under VL and under RF)); PF, sum of thickness of ankle plantar flexor muscles (gastrocnemius medialis (GM) and gastrocnemius lateralis (GL)).

APPENDIX 3
Muscle Strength Characteristics by Age Groups

Variables	Age Group				
	17–33 yr	40–49 yr	50–59 yr	60–69 yr	70–82 yr
Half squat 1-RM (kg)	n = 14	n = 14	n = 14	n = 12	n = 8
	195 ± 44[c,d,e]	166 ± 30[d,e]	160 ± 18[a,d,e]	124 ± 25[a,b,c]	109 ± 23[a,b,c]
CMJ height (cm)	n = 18	n = 14	n = 15	n = 16	n = 14
	52 ± 7*	41 ± 3*	33 ± 3[a,b,e]	29 ± 4[a,b,e]	23 ± 3*
Max isometric force (N)	n = 13	n = 14	n = 15	n = 16	n = 14
	3800 ± 850[c,d,e]	3300 ± 750[e]	2800 ± 650[a]	2600 ± 700[a]	2250 ± 650[a,b]
Specific isometric force ($N \cdot cm_{KE}^{-1}$)	n = 9	n = 12	n = 12	n = 15	n = 10
	308 ± 51	309 ± 69	299 ± 70	282 ± 73	260 ± 67
Isometric RFD ($N \times 10^3 \cdot s^{-1}$)	n = 13	n = 14	n = 15	n = 16	n = 13
	26.8 ± 6.4[c,d,e]	23.1 ± 5.8[d,e]	18.3 ± 5.5[a]	15.8 ± 5.5[a,b]	13.0 ± 4.2[a,b]
Specific isometric RFD ($N \cdot s^{-1} \cdot cm_{KE}^{-1}$)	n = 9	n = 12	n = 12	n = 15	n = 10
	2130 ± 400	2060 ± 530	2050 ± 760	1630 ± 460	1530 ± 510

Values are means ± SD. n, number of subjects.
[*,a,b,c,d,e] Group is significantly ($P < 0.05$) different from all other age groups, the 17- to 33-, 40- to 49-, 50- to 59-, 60- to 69-, and 70- to 82-yr-old groups, respectively.
CMJ, countermovement jump; RFD, rate of force development; cm_{KE}, sum of thickness of knee extensor muscles.

PAPER III

VARIABILITY AND SYMMETRY OF FORCE PLATFORM VARIABLES IN MAXIMUM-SPEED RUNNING IN YOUNG AND OLDER ATHLETES

by

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Variability and Symmetry of Force Platform Variables in Maximum-Speed Running in Young and Older Athletes

III

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ABSTRACT
Eighteen young (23±4 yr) and 25 older (70±4 yr) competitive male sprinters were examined for ground reaction force (GRF) and temporal-spatial variables. The data were collected during maximum-speed phase, and variability and symmetry indices were calculated from a total of 8 steps. There was little variation (CV<6%) in the vertical and resultant GRF and temporal-spatial variables, while vertical impact loading had high variability (CV:10–21%). The CV of the horizontal braking and push-off GRFs, vertical loading rate, and aerial time were higher in the older than in the young sprinters. There was a variable-specific asymmetry between legs but it was not related to leg dominance. No age differences existed in the symmetry indices. Results indicate that only selected force platform variables of sprinting are symmetric and repeatable enough so that their use for comparison purposes is appropriate. Data also suggest that aging increase variability in certain biomechanical measures, whereas symmetry may not be affected by age.

Keywords: Biomechanics, Ground reaction forces, Repeatability, Reliability, Locomotion, Aging

Running Head: Aging and biomechanics of sprinting

Conflict of interest: None

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INTRODUCTION

With aging the ability to run fast declines progressively (Hamilton,1993; Korhonen et al.,2003; Korhonen et al.,2009; Roberts et al.,1997). Although the previous studies have described nature of the age-related decline in maximum running speed, no authors have addressed the effect of aging on the parameter variability and symmetry. The knowledge of the degree of variability has importance for experimental work in indicating which measures can confidently detect small training- or age-related changes in an athlete's performance. Examination of symmetry has also direct relevance for the data collection indicating whether unilateral trials give symmetrical values for different variables, and provides insight into the potential functional imbalance of neuromuscular performance.

Investigations on young runners have indicated that many basic temporal-spatial and ground reaction force (GRF) variables of sprinting show relatively small intra-individual variability, and that a large number of trials/steps may not be required to obtain stable and reliable data (Bradshaw et al.,2007; Hunter et al.,2004; Mero & Komi,1986). However, these investigations have provided no conclusive evidence, because the only study (Mero & Komi,1986) that investigated the variability during maximum-speed running was limited to two GRF variables and the other studies (Bradshaw et al.,2007; Hunter et al.,2004) focused accelerated sprinting excluding direct comparison with the maximum-speed running. Furthermore, with increasing age reproducing the complex movement pattern of sprinting may become more challenging as the neuromuscular system and motor control deteriorates (Spirduso,2001).

In most sprint running studies, the biomechanical variables have been measured on only one side of the body, with the assumption that similar results would be obtained for the contralateral side. However, studies regarding symmetry during submaximal-speed running (at 2.9-6.8 $m \cdot s^{-1}$) in young athletes have indicated that symmetry in biomechanical measures between opposing legs cannot be automatically presumed (Belli et al.,1995; Williams et al.,1987). Moreover, there is some evidence to suggest that leg dominance/preference can increase asymmetry during submaximal-speed running and walking, because the dominant leg may be more responsible for propulsion, whereas the non-dominant leg plays a stabilizing function (Sadeghi et al.,2000). However, it remains unclear whether these findings on slow running or walking apply to sprint running with much higher force and movement speed requirements. Moreover, aging could increase asymmetry in sprinting due to an increase in bilateral asymmetry in muscle power and force production of lower limbs (Perry et al.,2007).

Despite the importance of parameter repeatability and symmetry for accurate performance assessment, there is a lack of information of these components in sprint running. Therefore, the purpose of this investigation was to determine variability and symmetry of force platform measures of maximum-speed sprinting in young and older runners. We took as our first hypothesis that older age is associated with increased parameter variability in sprint running. Our second hypothesis was that older age leads to increased performance asymmetry during maximal running.

METHODS

Eighteen young adult (age 23±4 yrs) and 25 older (age 70±4 yrs) sprinters were examined during preseason. The young participants were taller (1.79±0.04 m vs. 1.72±0.04 m, P<0.001) and heavier (77±6 kg vs. 71±8 kg, P=0.007) than the participants in the older group. However, this may not significantly influence our results as the forces were normalized to body weight, and the age-related difference in step length was similar when adjusted or unadjusted for the differences in body height. The participants had achieved national- or international-level performances in 100–400 m sprint events, and all were currently participating in competitions. The running times of the 60-m sprint trials performed in this study were 111±4% and 109±6% of the indoor age-based world record times for young and older participants, respectively.

In order to analyse bilateral symmetry of stride characteristics, the dominant (preferred) leg of the participant was defined as the participant's perceived dominant leg and the leg used for the take-off in a one-footed jump. Eleven of the young participants and 14 of the older participants were right-footed. A strength test (David Rehab 2200 dynamometer, David Fitness and Medical, Helsinki, Finland) of a subset of participants indicated that isometric knee extension torque was, on average, 3.7% and 5.0% higher in the dominant leg than in the non-dominant leg for the young (n=15) and older (n=18) participants, respectively. Thirteen of the young participants and 18 of the older participants were midfoot strikers, and the rest were forefoot strikers. (The forefoot strikers did not show an initial vertical impact force, Fz_{impact}, Figure 1a.). According to the medical histories and focused medical examination, all participants were healthy and free of musculoskeletal or neurological impairments which may affect the normal sprint running pattern. This study was approved by the Ethics Committee of the University of Jyväskylä, and written informed consent was obtained from the participants.

After a warm-up of about 30–45 mins and several submaximal practice trials, the participants performed two maximal 30-m sprints and two maximal 60-m sprints. The sprints were performed from a standing start in spiked running shoes on an indoor synthetic track that was bordered by an open area of 6-10 m on both sides. A rest interval between each run of 5–7 mins was given so as to avoid neuromuscular fatigue, which may lead to loss of maximal performance and an increase in the variability of the sprinting movement pattern.

Vertical and horizontal GRFs and step temporal-spatial variables were measured during the maximum-speed phase of the 60-m trials (from 30 m onwards) using a 9.4-m long force platform system. The force platform system consisted of nine tartan-surfaced force plates (five 2-D and three 3-D force plates, 0.9×1.0m, natural frequency ≥ 170Hz, nonlinearity ≤ 1%, cross talk ≤ 2%, TR-test, Jyväskylä, Finland; and one Kistler 3-D force plate, 0.9×0.9m, natural frequency 400Hz, Kistler, Winterthur, Switzerland) connected in series. The force platform system was firmly attached to a concrete base that was set below the track surface. The force signals were sampled at 1000Hz and stored on a microcomputer via an AT Codas A/D converter card (Dataq Instruments, Akron, OH, USA). The sprints over the force platform were also videotaped (Redlake Motionscope, 500C; 125Hz, 1/500; Redlake, San Diego, CA, USA) in lateral view to evaluate the foot-strike pattern during a run. The camera field of view was 9m, and showed all the foot contacts on the force platform system that were used in the biomechanical analyses. Step length, $L_{step,}$ was measured (to 0.5cm) from spike marks on a thin paper sheet that was firmly attached over the force platform (the paper was changed after about every fourth trial). In our previous measurements on young sprinters the difference between this paper method and film analysis was ±2 cm (A. Mero, unpublished observation). However, due to the potential errors related to the video analyses (digitization process), the

measurement of $L_{step,}$ is likely to be more accurate by using the paper method (exact spike marks) and was therefore used in this study. The maximal 10-m sprint velocity and 60-m sprint times were obtained using double-beam photocell gates. In addition, a laser radar (Laveg Sport; Jenoptik, Jena, Germany) positioned 6 m behind the start line, was used to analyze the instantaneous running speeds of the athletes. The results showed that the athletes achieved 99-100% of their maximum speed in the 30- to 40-m distance used for force platform measurements.

The force platform data were analysed using custom-written software. Vertical and horizontal antero-posterior force-time curves and the resultant force vector diagram are shown in Figure 1. The variables describing the maximum rate of impact loading and the average rate of impact loading were calculated from the initial part of the vertical force-time trace (Gottschall & Kram,2005). The maximum rate of impact loading, LR_{max}, was defined as the greatest instantaneous slope of the GRF trace (Figure 1a);

$$LR_{max} = \max\left\{\frac{dFz(t)}{dt}\right\} \quad (1)$$

The average rate of impact loading, LR_{ave}, was defined as

$$LR_{ave} = \frac{Fz_{impact}}{\Delta t_F} \quad (2)$$

where Δt_F is the time interval from the beginning of the ground contact to the instant of the impact peak, Fz_{impact}.

The other GRF variables included the maximal and average values of the vertical (*Fz*), horizontal antero-posterior (*Fy*), and resultant (*Fr*) forces. The resultant GRF was a 2-D vector composed of the vertical and anterior/posterior GRF components. The transition point from negative to positive values in the antero-posterior GRF curve was used to divide the force components into braking and propulsion phases (Mero & Komi,1986). For these analyses, a vertical force threshold of 20N was used to identify the beginning and end of the ground contact. Of the temporal variables, contact time ($t_{contact}$), aerial time (t_{aerial}), and step frequency ($Freq_{step}=1/(t_{contact}+t_{aerial})$) were obtained from the force-time traces. The dominant side step for the parameters of t_{aerial}, $Freq_{step}$, and L_{step} was defined as the step that begins from the dominant leg contact (dominant side $Freq_{step}$ included the $t_{contact}$ of the dominant leg).

The asymmetry between dominant and non-dominant leg variables was quantified with a symmetry index (*SI*) (Herzog et al.,1989);

$$SI(\%) = \frac{X_D - X_{ND}}{1/2(X_D + X_{ND})} \cdot 100\% \quad (3)$$

where X_D is the variable for the dominant leg, and X_{ND} is the variable for the non-dominant leg. When *SI* of the individual is zero, there is perfect symmetry between the legs. A positive *SI* indicates a higher value for the dominant leg than for the non-dominant leg, and a negative *SI* indicates a lower value for the dominant leg than for the non-dominant leg. The data were also examined using the absolute symmetry index, $ASI=|SI|$,

so that averaging positive and negative symmetry indices over several participants does not lead to a zero value (Giakas & Baltzopoulos,1997).

Each trial involved 4–6 contacts on the force platform. For consistency, the first four contacts of the two trials were taken in the analyses for each participant. In the analyses the values for the dominant leg and non-dominant leg were calculated from the two trials and averaged intra-individually. Furthermore, the forces were normalized to body weight (bw) in order to allow comparisons between participants.

All statistical analyses were performed using SPSS 16.0 software (SPSS Inc., Chicago, IL, USA). The coefficient of variation, CV = (SD/mean) × 100, was calculated to assess the relative intra-individual variability of the variables for the dominant leg and for the non-dominant leg. In the force platform measurements, the errors related to instrumentation and scoring are small and the major source of variability is expected to be due to the biovariation associated with human movement (Hamill & McNiven,1990). A 2×2 ANOVA (two age groups × two sides) was used to examine the influence of age and leg dominance on the CV values (Figure 2). Age group comparisons for the mean values of all the dependent variables (dominant and non-dominant side values pooled together), and symmetry indices (*SI* and *ASI*) were made using ANOVA (Table 1). A paired *t*-test, with Holm correction for multiple comparisons, was used to compare the mean values of the GRF and step temporal-spatial variables between dominant and non-dominant legs within groups. Test-retest comparison of maximum running speed and 60-m running time within groups was made using paired *t*-tests. The level of significance was set to $P<0.05$.

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RESULTS

Older age was associated with a decline in maximum running speed (9.50±0.42m· s^{-1} *vs*. 7.30±0.57m·s^{-1}, *P*<0.001) and an increase in the 60-m times (7.09±0.21s vs. 9.06±0.65 s, *P*<0.001). For vertical GRFs, the young participants produced a greater LR_{max}, (*P*=0.009), Fz_{max} (*P*<0.001) and Fz_{ave} (*P*<0.001), whereas LR_{ave} was similar (*P*=0.177), and Fz_{impact} lower (*P*=0.012) in the young participants than in the older participants. All the horizontal GRFs showed higher values for the young participants than for the older participants (all *P*<0.001). The resultant GRFs, $Fr_{brake\text{-}max}$ (*P*=0.022), $Fr_{brake\text{-}ave}$ (*P*<0.001), $Fr_{push\text{-}max}$ (*P*<0.001), and $Fr_{push\text{-}ave}$ (*P*<0.001) were higher in the young participants than in the old participants.

For the temporal-spatial variables, the young participants had a shorter $t_{contact}$ and a greater t_{aerial}, $Freq_{step}$, and L_{step} compared to the older participants (all *P*<0.01). When L_{step} was calculated in relation to body height, the age-related decline remained significant (L_{step}/body height ratio=1.20±0.03 vs. 1.03±0.06; *P*<0.001).

The CVs ranged from 1.5% to 17.4% in the young participants, and from 1.4% to 21.1% in the older participants (Figure 2). In general, the variation in both groups was low (CV < 6%) in the vertical and resultant forces and in all the step temporal-spatial variables, but clearly higher for vertical loading (LR_{max}, LR_{ave}, Fz_{impact}) and horizontal forces. The test-retest CV values for the maximum speed and 60-m times were 0.6±0.3% and 0.4±0.2% in the young group of participants, and 0.7±0.5% and 0.5±0.5% in the older group of participants, respectively.

The CVs of LR_{max} (*P*=0.037), $Fy_{brake\text{-}max}$ (*P*<0.001), $Fy_{push\text{-}max}$ (*P*<0.001), $Fy_{push\text{-}ave}$ (*P*=0.003), and t_{aerial} (*P*<0.001), showed higher variability in the older participants than in the younger participants (Figure 2). No effect of lateral dominance on the CV values was observed.

There were no differences in the variable mean values between the dominant and non-dominant legs in either group (Table 1). The symmetry index ranged from –5.4% to +5.6% in the young group, and from –2.6% to +2.7% in older group (Table 1). There was one significant age-related difference in the symmetry index, with younger participants showing greater asymmetry for t_{aerial} than older participants (*P*=0.037).

The absolute inter-leg asymmetry (*ASI*) values ranged from 2.7% to 14.3% in the young group, and from 2.2% to 18.8% in the older group (Table 1). Age had a significant effect on absolute symmetry index of Fz_{max} that was higher in the young (3.8%) than in older (2.2%) group (*P*=0.016).

DISCUSSION

The primary new findings from the present investigations were as follows. First, among the many commonly used force platform variables only step temporal-spatial (L_{step}, $Freq_{step}$, $t_{contact}$) and selected vertical (Fz_{max}, Fz_{ave}) and resultant ($Fr_{brake\text{-}ave}$, $Fr_{push\text{-}max}$, $Fr_{push\text{-}ave}$) GRF variables provided repeatable and symmetric values during maximum-speed running. Second, the older runners showed higher CVs than young runners in the GRF variables measuring maximum vertical loading rate, horizontal braking and push-off forces, and aerial time. This finding provides partial support for the hypothesis that aging leads to increased variability in sprinting. Third, the data also suggest that symmetry of the biomechanical measures is not affected by age, thus not supporting our second hypothesis of age-related increase in asymmetry.

The present investigation demonstrated that in both age groups the CVs of all the temporal-spatial variables and selected vertical and resultant GRF variables were less than 6% (Fig. 2). These variables can be suitable for inter-individual comparisons and reliable enough to identify the relatively small training-induced changes in an athlete's performance. For example, in a recent study (Cristea et al.,2008) older sprinters showed 3% and 8% increases in L_{step} and $Fr_{push\text{-}ave}$ in response to training that are likely to be detected by the variability levels of 1.5% and 5.5%, respectively, observed in this investigation. In contrast, all the vertical loading and some horizontal GRF ($Fy_{brake\text{-}max}$, $Fy_{brake\text{-}ave}$) variables demonstrated larger variability (CV>10%) in the present work and should be used with caution for sprint assessment. However, the relatively large CV% values of horizontal GRFs reflect, in part, the low absolute values of these variables.

Our results about the magnitude of variability in the force platform measures are in line with the findings of the only available study on the variability of maximum-speed sprinting (Mero & Komi,1986). The study examined reproducibility of $Fr_{brake\text{-}ave}$ and $Fr_{push\text{-}ave}$ during maximal speed phase (35-45 m; 8.8-10.2 m/s) in 11 male and 8 female young adult competitive sprinters (Mero & Komi,1986). The CV for the Fr_{ave} of two right foot contact was 7.3% and 5.1% during the braking and push-off phase, respectively. However, the study was limited to two GRF measures. Accordingly, the present study is the first to provide more comprehensive information on the variability and symmetry of the measures during maximum-speed sprinting and provide for basis of comparison for future studies.

Older runners exhibited greater CV than young runners for the GRF variables describing both braking and push-off phases of the contact (Fig. 2). The main difference was in the horizontal component where three out of four examined parameters showed age-related increase in the CV values. From a practical standpoint, our results indicate that older runners have comparable variability to young runners in the variables that are characterized by relatively good repeatability (CV<6%) and similar number of trials may be used to describe the basic biomechanical characteristics of sprint running. However, in horizontal GRFs more steps are required to produce reliable values, especially for older runners, to ensure the biomechanical relevance of the differences obtained.

The mechanism of the increased in variability was not addressed in this study and could only be cautiously speculated based on data of non-sprint activities. Overall control of locomotor movements requires integration of central nervous system with sensory inputs (visual, vestibular, proprioceptive) to produce appropriate motor response (Dietz,2003). Evidence also suggests that complex networks of neurons located in the spinal cord (central pattern generators), with the control of higher centers and feedback from peripheral receptors, are important for the automated rhythmic movement generation in locomotion (Dietz,2003; Dimitrijevic et al.,1998; Grillner,1981). Age-related degeneration within the central or peripheral nervous system may lead to impairment in

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automatic pattern generation and increase variability in locomotion (Prince et al.,1997). For example, recent study including a large sample of healthy older adults (n=558, 79.4±4.1 yrs) showed that increased variability in step time in walking was related to impairment in the central nervous system, whereas step width variability was related to sensory impairment (Brach et al.,2008). Studies have also provided evidence that the nervous system's ability to control leg muscle force generation is impaired with age, particularly in eccentric contractions (Carville et al.,2007; Christou & Carlton,2002). Thus, one possibility is that aging sprint athletes experience impairment in the force control by nervous system, which is reflected in an increased in variability in the biomechanical parameters. However, if the neuromuscular force control was the mechanism, one might expect significant CV differences also in vertical GRFs that are on average 5 times greater than horizontal GRFs; this did not occur suggesting that other mechanism may be responsible.

Some authors assume that increased variability in horizontal braking and push-off GRFs could be due to differences in running style (Lees & Bouracier,1994). In their study they examined loading and horizontal GRFs in 7 experienced and 7 inexperienced male runners during overground 60-m running trials (4.7-5.2 m/s). The inexperienced runners showed greater variability in braking and push-off impulses (running style variables), whereas there were no group differences in LR_{max}, LR_{ave} or Fz_{impact}. Further, non-runners had similar LR_{max} and LR_{ave} but higher Fz_{impact} that can reflect a heavier heel strike and reduced shock absorption ability. Accordingly, those group differences in GRF pattern seem to be quite similar to those found between young and older sprinters in the current study suggesting that differences in running style and experience (amount of practice) of the athletes may have played role in the age-related increase in variability, especially in horizontal forces.

Preferred use and superior performance and skill of one leg versus the other is commonly observed for various motor activities (Peters,1988). This may be due to genetically determined brain laterality that is intensified by increased use of one side in daily activities and exercise (Gabbard & Hart,1996; Peters,1988). The *SI* values between the dominant and non-dominant legs of this study seem to suggest no effect of lateral dominance on sprint running (Table 1). Our result is in line with some studies of slow running (Hamill et al.,1984) and walking (Gundersen et al.,1989; Hamill et al.,1984).

The *SI* equation is "limited" in that pooled data across participants can lead to a zero value, if some participants show superior measurements by the dominant leg and some superior by non-dominant leg. When we did not take into account leg dominance but examined only inter-leg differences, the *ASI* symmetry values ranged from 2.7% to 14.3% (mean 7.8%) in young and from 2.2% to 18.8% (mean 7.8%) in older runners. According to this result, marked variable-specific asymmetries between legs are present during sprinting, supporting previous studies on submaximal-speed running (Belli et al.,1995; Vagenas & Hoshizaki,1992; Williams et al.,1987; Zifchock et al.,2006). The smallest ASI values (≤ 6%) were found in selected temporal-spatial variables (L_{step}, $Freq_{step}$, $t_{contact}$) and vertical (Fz_{max}, Fz_{ave}) and resultant GRFs ($Fr_{brake\text{-}ave}$, $Fr_{push\text{-}max}$, $Fr_{push\text{-}ave}$) and might be expected to reflect, in part, small variability of these parameters.

The *SI* and *ASI* results provided evidence that older age has no effect on symmetry during sprint running at maximal force effort. These results do support earlier findings of inter-leg asymmetry during walking in healthy individuals of different age (Liikavainio et al.,2007; Menz et al.,2004; Stacoff et al.,2005). One could thus infer that increased asymmetry in different modes of human locomotion is not a normal concomitant of aging.

The maintenance of symmetry in older runners may indicate that although the structure and function of neuromuscular locomotor system show age-related degeneration, the movement coordination and force characteristics of each side of the

body are similarly affected by age. It is partially supported by our finding on isometric strength test showing that in older athletes the strength advantage of the dominant leg was 5%, which compared quite well to that (3.7%) in young runners. In sprint running the stride pattern is highly dependent on the ability to tolerate the great contact forces. Thus, if strength asymmetry is large, it could affect symmetry in maximal running.

Neural basis of bilateral symmetry in human running is not known and is very difficult to investigate. However, studies have provided insight into the neural mechanisms responsible for left-right coordination in walking that may apply to running. Some investigators have assumed that the primary site of symmetry regulation during walking is the central pattern generator of the spinal cord (Yogev et al.,2007). Studies using split-belt treadmill have suggested that there are autonomous pattern generators for each leg that communicates with its counterpart for the contralateral leg to ensure bilateral coordination (Dietz et al.,1994; Yang et al.,2005). The regulation of symmetry may also rely on proprioceptive feedback (Dietz et al.,1994), and control by higher centers that is evidenced by excessive asymmetry in people with certain unilateral central nervous system pathology (Wall & Turnbull,1986). Accordingly, the lack of any significant difference between the asymmetry of biomechanical parameters in young and older runners in this study suggests that the capacity of integrated neuromuscular system to coordinate very fast symmetric movements is not compromised by aging. This may partially reflect favourable effects of lifelong training on movement coordination (Spirduso,2001). To clarify this point it would be interesting to compare older athletes and non-athletes in locomotion, motor control, and structure and function of the brain.

Taken together, our results suggest that asymmetry in biomechanical variables of sprint running is not influenced by age, vary with the parameter of interest, and cannot be predicted from leg dominance. Unilateral leg examination may not be representative and this should be taken into account for comprehensive sprint performance evaluation. Furthermore, the observation that asymmetry was not related to leg dominance raises a question as to whether general definitions used for leg dominance (e.g., leg used for one-foot jumping or kicking a ball) are valid for locomotion action (Sadeghi et al.,2000). In this regard, determination of absolute inter-leg differences (*ASI*) could be a more appropriate method for estimating symmetry in sprint running.

Certain limitations of our study should be pointed out. First, in the current study the measurements were taken on the same day. Information regarding inter-day repeatability is needed to complement the findings of the present study. Second, our study sample consisted only of highly-trained competitive male sprinters. The reproducibility and symmetry of biomechanical variables may be different in female athletes or inexperienced runners. Third, a limitation of the study is the relatively small number of steps analysed. However, we believe that a larger number of maximal overall sprint trials are not practically possible for runners to perform without a confounding fatigue effect, and that it is important to establish the reliability of assessment tasks that are applicable to normal training and competition situations.

On the other hand, a main advantage of the present study was that the measurements were made using a unique long force platform system which allowed the recording of consecutive steps bilaterally during the same trial. This significantly cut time in data collection and is likely to reduce the targeting-, velocity-, and fatigue-induced variability in the measurement which may be a matter for concern when a single force plate with multiple maximal sprint trials is used (Abendroth-Smith,1996; Hunter et al.,2004). Actually, based on previous study by Hunter et al. (2004), with single force plate method about 7-8 sprints are needed to obtain 4-5 successful ground contacts for one side.

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In conclusion, these results indicate that only selected force platform variables are symmetric and repeatable enough so that their use for experimental work and comparison purposes is appropriate. Our data also provide evidence that older age increase variability in certain GRF and step temporal-spatial variables of sprint running, whereas the symmetry of the biomechanical measures may not be affected by age.
In contrast to endurance running and walking, studies evaluating the reliability of biomechanical variables in sprint running have been very limited. This lack of knowledge must be regarded as a serious problem that may influence the accuracy of performance assessment and could lead to wrong conclusions. Thus the present data have potentially important implications and can be used as a reference in the selection of biomechanical variables designed to investigate sprint performance in young and older runners.

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REFERENCES

Abendroth-Smith, J. (1996). Stride adjustments during a running approach toward a force plate. *Research Quarterly for Exercise and Sport, 67*, 97-101.

Belli, A.,Lacour, J. R.,Komi, P. V.,Candau, R., & Denis, C. (1995). Mechanical step variability during treadmill running. *European Journal of Applied Physiology, 70*, 510-517.

Brach, J. S.,Studenski, S.,Perera, S.,VanSwearingen, J. M., & Newman, A. B. (2008). Stance time and step width variability have unique contributing impairments in older persons. *Gait and Posture, 27*, 431-439.

Bradshaw, E. J.,Maulder, P. S., & Keogh, J. W. (2007). Biological movement variability during the sprint start: performance enhancement or hindrance? *Sports Biomechanics, 6*, 246-260.

Carville, S. F.,Perry, M. C.,Rutherford, O. M.,Smith, I. C., & Newham, D. J. (2007). Steadiness of quadriceps contractions in young and older adults with and without a history of falling. *European Journal of Applied Physiology, 100*, 527-533.

Christou, E. A., & Carlton, L. G. (2002). Age and contraction type influence motor output variability in rapid discrete tasks. *Journal of Applied Physiology, 93*, 489-498.

Cristea, A.,Korhonen, M. T.,Hakkinen, K.,Mero, A.,Alen, M.,Sipila, S.,Viitasalo, J. T.,Koljonen, M. J.,Suominen, H., & Larsson, L. (2008). Effects of combined strength and sprint training on regulation of muscle contraction at the whole-muscle and single-fibre levels in elite master sprinters. *Acta Physiologica (Oxf), 193*, 275-289.

Dietz, V. (2003). Spinal cord pattern generators for locomotion. *Clinical Neurophysiology, 114*, 1379-1389.

Dietz, V.,Zijlstra, W., & Duysens, J. (1994). Human neuronal interlimb coordination during split-belt locomotion. *Experimental Brain Research, 101*, 513-520.

Dimitrijevic M.,Gerasimenko Y., Pinter M. (1998). Evidence for a spinal central pattern generator in humans. *Annals of the New York Academy of Science, 860*, 360-376.

Gabbard, C., & Hart, S. (1996). A question of foot dominance. *Journal of General Psychology, 123*, 289-296.

Giakas, G., & Baltzopoulos, V. (1997). Time and frequency domain analysis of ground reaction forces during walking: an investigation of variability and symmetry. *Gait and Posture, 5*, 189-197.

Gottschall, J. S., & Kram, R. (2005). Ground reaction forces during downhill and uphill running. *Journal of Biomechanics, 38*, 445-452.

Grillner, S. (1981). Control of locomotion in bepeds, tetrapods, and fish. In *Handbook of Physiology: Section 1: Motor control* (Vol. 2, pp. 1179-1234). Washington, DC: American Physiological Society.

Gundersen, L. A.,Valle, D. R.,Barr, A. E.,Danoff, J. V.,Stanhope, S. J., & Snyder-Mackler, L. (1989). Bilateral analysis of the knee and ankle during gait: an examination of the relationship between lateral dominance and symmetry. *Physical Therapy, 69*, 640-650.

Hamill, J.,Bates, B. T., & Knutzen, K. M. (1984). Ground reaction force symmetry during walking and running. *Research Quarterly for Exercise and Sport, 55*, 289-293.

Hamill, J., & McNiven, S. L. (1990). Reliability of selected ground reaction force parameters during walking. *Human Movement Science, 9*, 117-131.

Hamilton, N. (1993). Changes in sprint stride kinematics with age in master's athletes. *Journal of Applied Biomechanics, 9*, 15-26.

Herzog W, Nigg B. M, Read, L, Olsson E. (1989). Asymmetries in ground reaction force patterns in normal human gait. *Medicine and Science in Sports and Exercise, 21*, 110-114.

Hunter, J. P.,Marshall, R. N., & McNair, P. (2004). Reliability of biomechanical variables

of sprint running. *Medicine and Science in Sports and Exercise, 36*, 850-861.
Korhonen, M. T.,Mero, A., & Suominen, H. (2003). Age-related differences in 100-m sprint performance in male and female master runners. *Medicine and Science in Sports and Exercise, 35*, 1419-1428.
Korhonen, M. T.,Mero, A. A.,Alen, M.,Sipila, S.,Hakkinen, K.,Liikavainio, T.,Viitasalo, J. T.,Haverinen, M. T., & Suominen, H. (2009). Biomechanical and skeletal muscle determinants of maximum running speed with aging. *Medicine and Science in Sports and Exercise, 41*, 844-856.
Lees, A., & Bouracier, J. (1994). The longitudinal variability of ground reaction forces in experienced and inexperienced runners. *Ergonomics, 37*, 197-206.
Liikavainio, T.,Isolehto, J.,Helminen, H. J.,Perttunen, J.,Lepola, V.,Kiviranta, I.,Arokoski, J. P., & Komi, P. V. (2007). Loading and gait symmetry during level and stair walking in asymptomatic subjects with knee osteoarthritis: importance of quadriceps femoris in reducing impact force during heel strike? *Knee, 14*, 231-238.
Menz, H. B.,Latt, M. D.,Tiedemann, A.,Mun San Kwan, M., & Lord, S. R. (2004). Reliability of the GAITRite walkway system for the quantification of temporo-spatial parameters of gait in young and older people. *Gait and Posture, 20*, 20-25.
Mero, A., & Komi, P. V. (1986). Force-, EMG-, and elasticity-velocity relationships at submaximal, maximal and supramaximal running speeds in sprinters. *European Journal of Applied Physiology, 55*, 553-561.
Perry, M. C.,Carville, S. F.,Smith, I. C.,Rutherford, O. M., & Newham, D. J. (2007). Strength, power output and symmetry of leg muscles: effect of age and history of falling. *European Journal of Applied Physiology, 100*, 553-561.
Peters M. (1988). Footedness: asymmetries in foot preference and skill and neuro-psychological assessment of foot movement. *Psychology Bulletin, 103*, 179-192.
Prince, F.,Corriveau, H.,Hebert, R., & Winter, D. A. (1997). Gait in the elderly. *Gait and Posture, 5*, 128-135.
Roberts, E. M.,Cheung, T. K.,Abdel Hafez, A. M., & Hong, D. (1997). Swing limb biomechanics of 60- to 65-year-old male runners. *Gait and Posture, 5*, 42-53.
Sadeghi, H.,Allard, P.,Prince, F., & Labelle, H. (2000). Symmetry and limb dominance in able-bodied gait: a review. *Gait and Posture, 12*, 34-45.
Spirduso, W. W. (2001). Aging and motor control. In C. V. Gisolfi, G. D. Lamb & E. Nadel (Eds.), *Perspectives in exercise science and sports medicine: Exercise in older adults* (Vol. 8, pp. 53-114): Cooper Publishing Group.
Stacoff, A.,Diezi, C.,Luder, G.,Stussi, E, Kramers-de Quervain, I (2005). Ground reaction forces on stairs: effects of stair inclination and age. *Gait Posture, 21*, 24-38.
Vagenas G & Hoshizaki B (1992). A multivariable analysis of lower extremity kinematic asymmetry in running. *International Journal of Sport Biomechanics, 8*, 11-29.
Wall, J. C., & Turnbull, G. I. (1986). Gait asymmetries in residual hemiplegia. *Archives of Physical Medicine and Rehabilitation, 67*, 550-553.
Williams, K. R.,Cavanagh, P. R., & Ziff, J. L. (1987). Biomechanical studies of elite female distance runners. *International Journal of Sports Medicine, 8*, s107-118.
Yang, J. F.,Lamont, E. V., & Pang, M. Y. (2005). Split-belt treadmill stepping in infants suggests autonomous pattern generators for the left and right leg in humans. *Journal of Neuroscience, 25*, 6869-6876.
Yogev, G.,Plotnik, M.,Peretz, C.,Giladi, N., & Hausdorff, J. M. (2007). Gait asymmetry in patients with Parkinson's disease and elderly fallers: when does the bilateral coordination of gait require attention? *Experimental Brain Research, 177*, 336-346.
Zifchock, R. A.,Davis, I., & Hamill, J. (2006). Kinetic asymmetry in female runners with and without retrospective tibial stress fractures. *Journal of Biomechanics, 39*, 2792-2797.

FIGURE CAPTIONS

Figure 1.
Example of (a) vertical and (b) horizontal force-time curves, and (c) the resultant force vector diagram of the first right foot contact for a young sprinter with midfoot strike while running at 10.0 $m \cdot s^{-1}$. In panel c, the diagram illustrates the changes in magnitude and orientation of the resultant GRF through the contact. A vertical broken line in the first contact (a-c) indicates the border between the braking and push-off phases. Fz_{max} = maximal vertical force, Fz_{impact} = vertical impact force, LR_{max} = maximum rate of impact loading (the greatest instantaneous slope in the vertical GRF), LR_{ave} = average rate of impact loading (Fz_{impact} / the time interval from the beginning of the ground contact to the instant of the impact peak), $Fy_{brake\text{-}max}$ = maximal horizontal braking force, $Fy_{push\text{-}max}$ = maximal horizontal push-off force, $Fr_{brake\text{-}max}$ = maximal resultant braking force, $Fr_{push\text{-}max}$ = maximal resultant push-off force, $t_{contact}$ = ground contact time, t_{aerial} = aerial time, bw = body weight.

Figure 2.
Intra-individual coefficient of variations (CV %) of the (a) vertical, (b) horizontal, and (c) resultant components of the GRF, and (d) the temporal-spatial step variables for the dominant (dom) and non-dominant (non-dom) legs in young and older sprinters. Bars indicate group means ± SD calculated from four steps of each leg (from 2 trials). $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$ for the significant age effect. See text and Figure 1 for description of the variables. The number of participants for each variable is the same as in Table 1. Note the different scale on the *y*-axes.

Table 1. The components of the GRFs and temporal-spatial stride parameters of sprint running for the dominant and non-dominant sides, and the symmetry indices (SI, ASI) in young and older subjects.

	Young runners 9.50±0.42 m/s				Older runners 7.30±0.57 m/s			
	Dominant	**Non-dominant**	**SI %**	**ASI %**	**Dominant**	**Non-dominant**	**SI %**	**ASI %**
Vertical force								
LR_{max} (bw/s)	418±58	418±56	-0.6±15.4	11.8±9.9	338±121	339±112	0.6±20.4	18.1±11.3
LR_{ave} (bw/s)[a]	211±32	206±26	1.7±15.9	11.2±11.0	193±59	195±60	-1.3±25.6	18.8±16.8
Fz_{impact} (bw)[a]	2.01±0.79	2.12±0.72	-5.4±15.4	13.5±8.5	3.20±0.64	3.29±0.69	-2.6±16.7	10.1±13.4
Fz_{max} (bw)	3.34±0.25	3.35±0.26	-0.3±6.8	**3.8±2.6**	2.82±0.34	2.83±0.33	-0.3±4.0	**2.2±1.6***
$F_{z\,ave}$ (bw)	2.07±0.13	2.02±0.10	2.0±4.5	3.8±3.1	1.85±0.19	1.85±0.20	0.5±4.8	4.1±2.7
Horizontal force								
$Fy_{brake\text{-}max}$ (bw)	1.42±0.17	1.43±0.24	0.6±15.8	13.7±7.8	0.88±0.20	0.91±0.26	-2.0±22.6	18.3±14.3
$Fy_{brake\text{-}ave}$ (bw)	0.40±0.04	0.41±0.06	-0.3±17.0	14.3±9.8	0.31±0.04	0.32±0.05	-1.9±22.1	17.4±15.2
$Fy_{push\text{-}max}$ (bw)	0.74±0.09	0.73±0.08	3.0±7.9	6.5±5.2	0.50±0.07	0.50±0.07	2.7±6.8	6.5±3.7
$Fy_{push\text{-}ave}$ (bw)	0.41±0.03	0.40±0.04	2.9±11.3	10.1±5.7	0.29±0.04	0.28±0.03	2.1±11.1	9.7±6.5
Resultant force								
$Fr_{brake\text{-}max}$ (bw)	3.76±0.63	3.52±0.38	5.6±11.0	9.6±7.7	3.29±0.50	3.26±0.57	1.5±8.6	6.1±6.5
$Fr_{brake\text{-}ave}$ (bw)	2.70±0.25	2.62±0.17	2.8±6.7	5.8±4.4	2.40±0.29	2.39±0.30	0.7±4.7	4.1±2.7
$Fr_{push\text{-}max}$ (bw)	3.08±0.21	3.05±0.25	0.5±6.5	4.2±4.4	2.74±0.31	2.74±0.31	-0.2±5.6	4.8±3.0
$Fr_{push\text{-}ave}$ (bw)	1.90±0.15	1.87±0.17	0.4±6.6	3.9±4.3	1.61±0.19	1.62±0.18	-0.3±6.8	5.9±3.5
Temporal-spatial parameters								
$t_{contact}$ (ms)	102±7	101±7	1.4±4.7	4.0±2.9	129±16	127±16	1.5±3.6	3.3±2.1
t_{aerial} (ms)	129±11	123±9	**5.0±8.1**	8.0±5.2	116±9	117±12	**-0.6±8.7***	7.3±5.0
$Freq_{step}$ (Hz)	4.34±0.25	4.49±0.25	-3.6±5.8	5.7±3.8	4.14±0.27	4.16±0.30	-0.4±5.0	4.0±3.1
L_{step} (m)	2.16±0.07	2.13±0.07	1.7±3.2	2.7±2.3	1.77±0.10	1.77±0.12	-0.2±3.0	2.6±1.6

Values are group means ± SD. Number of subjects = 18 young and 25 older subjects ([a] these variables could be determined only for the midfoot strikers, n= 13 young and 18 older runners). *Significantly different (P<0.05) from the corresponding value in the young group (indicated by bold numbers). bw = body weight. See text and Fig. 1 for description of the parameters.

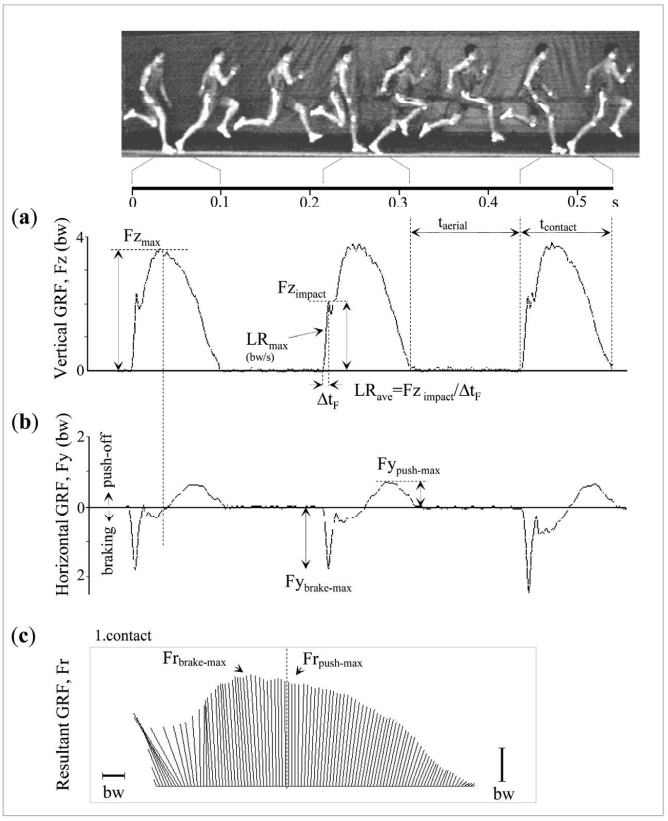

Figure 1

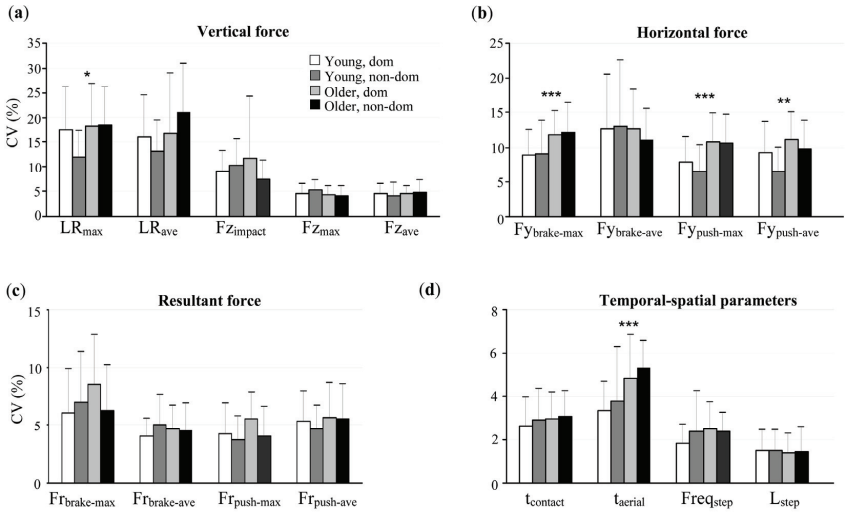

Figure 2

PAPER IV

AGE AND SEX DIFFERENCES IN BLOOD LACTATE RESPONSE TO SPRINT RUNNING IN ELITE MASTER ATHLETES

by

Korhonen MT, Suominen H, & Mero A (2005)

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Age and Sex Differences in Blood Lactate Response to Sprint Running in Elite Master Athletes

IV

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Abstract/Résumé

The effect of age and sex on anaerobic glycolytic capacity in master athletes is currently unclear. To study this issue, we determined blood lactate concentrations after competitive sprint running in male and female master athletes of different age. Eighty-one men (40–88 yrs) and 75 women (35–87 yrs) participating in the sprint events (100-m, 200-m, 400-m) in the European Veterans Athletics Championships were studied. Blood samples were taken from the fingertip and analysed for peak lactate concentration ($[La]_b$ peak). The $[La]_b$ peak following 100-m to 400-m races showed a curvilinear decline ($p < 0.001–0.05$) with age in both men and women. However, the age related differences in the $[La]_b$ peak were not significant before 70 years of age. No significant sex related differences were found in $[La]_b$ peak for any sprint event. The $[La]_b$ peak correlated significantly ($p < 0.001–0.05$) with running times in all sprint distances except for the age-controlled correlation in men for the 100-m and 200-m. In conclusion, the present study showed age but not sex differences in blood lactate response to competitive sprint running in master athletes. Although the $[La]_b$ peak level of the athletes was considerably higher than that reported for untrained men and women, these cross-sectional findings suggest that anaerobic energy production from glycolysis declines in later years and may be a factor in the deterioration in sprint performance.

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Nous connaissons mal les effets de l'âge et du sexe sur la capacité glycolytique anaérobie des athlètes vétérans. Afin de mieux cerner cette question, nous avons pris des mesures de la concentration sanguine de lactate après une compétition de sprint chez des vétérans des deux sexes d'âge varié. Quatre-vingt-un hommes (44 à 88 ans) et 75 femmes (35 à 87 ans) inscrits aux épreuves de sprint (100-m, 200-m, 400-m) au Championnat Européen des vétérans en athlétisme ont participé à l'étude. L'analyse de la concentration sanguine de lactate de pointe ($[La]_b$ peak) est faite à partir d'échantillons de sang prélevé au bout du doigt. Les $[La]_b$ peak à la suite des épreuves de sprint (100-m, 200-m, 400-m) montrent un décours curvilinéaire en fonction de l'âge tant chez les femmes que chez les hommes ($p <$ 0,001–0,05). Avant l'âge de 70 ans, les différences de $[La]_b$ peak ne sont pas significatives. En outre, dans toutes les épreuves de sprint, on n'observe aucune différence de $[La]_b$ peak entre les sexes. À l'exception de la corrélation ajustée à l'âge dans les sprints sur 100-m et sur 200-m chez les hommes, la corrélation entre les $[La]_b$ peak et le temps de course est significative dans toutes les épreuves de sprint ($p <$ 0,001–0,05). En conclusion, cette étude ne montre pas de différence entre les sexes mais des différences selon l'âge quant à la concentration de lactate observée à la suite d'une épreuve de sprint chez des athlètes vétérans; même si la $[La]_b$ peak est beaucoup plus élevée que ce qu'on observe dans la littérature scientifique à propos des femmes et des hommes non entraînés, les données transversales indiquent que la production anaérobie d'énergie dans la glycolyse diminue avec le vieillissement et que cette diminution peut affecter la performance en sprint.

IV

Introduction

During high-intensity sprinting most of the energy is supplied from anaerobic sources by the breakdown of phosphocreatine (PCr) and anaerobic glycolysis. PCr stores are very rapid energy sources but are limited in quantity and reduced to a very low level after short-term maximal sprinting, such as 10 s (Hirvonen et al., 1987). Anaerobic glycolysis which is active from the beginning of maximal exercise can be maintained for a longer time. It has been estimated that in a world-class 100-m sprint performance (~10 s) the energy provision is 50% from PCr and 50% from anaerobic glycolysis. In a 400-m sprint (~45 s), PCr contributes 12.5% and anaerobic glycolysis contributes 62.5% of the required energy, with the remaining 25% generated from aerobic processes (Newsholme et al., 1992). The increased relative demand on the anaerobic glycolytic system with increasing sprint distance up to 400 m is reflected in blood lactate levels. In one report, postrace blood lactate concentrations for elite male sprinters were 13.2 ± 1.8 mmol/L, 19.2 ± 1.5 mmol/L, and 22.0 ± 1.6 mmol/L after 100-m, 200-m, and 400-m races, respectively (Kindermann and Keul, 1977).

Single-muscle-fiber studies have demonstrated that fast-twitch fibers are favorable for rapid energy production during high-intensity sprint exercise. These fibers possess higher basal PCr content and consume more of their PCr stores during sprinting than slow-twitch fibers (Greenhaff et al., 1994; Tesch et al., 1989). Moreover, fast-twitch fibers are rich in key glycolytic enzymes and produce more lactic acid than slow-twitch fibers (Essén and Häggmark, 1975; Tesch et al., 1978). However, specific training can modulate the anaerobic metabolic potential of a particular fiber. Early studies have suggested that sprint training, unlike endurance training (Henriksson and Reitman, 1976), can lead to increases in glycolytic en-

zyme activity, especially in slow-twitch fibers (Saubert et al., 1973; Staudte et al., 1973). It has also been reported that in maximal muscular work, PCr stores are depleted more in sprint athletes than in endurance athletes, probably due to differences in training-induced adaptations in the capacity to recruit different muscle fibers (Rehunen et al., 1982).

On the basis of previous studies, the normal aging process involves changes in the muscle properties that are likely to impair one's capacity to produce energy through the anaerobic system. It is known that with aging comes a loss of muscle mass and a shift toward a more oxidative muscle profile mediated by the atrophy of fast-twitch fibers (e.g., Porter et al., 1995). These changes in muscle characteristics would indicate a reduced PCr metabolic capacity and a decreased rate of glycolysis and lactic acid formation. It is also possible that the anaerobic energy output decreases with age due to reductions in key glycolytic enzymes, particularly phosphofructokinase (Hunter et al., 2002; Keh-Evans et al., 1992). Further, a reduction in the amount of high intensity exercise could in part contribute to the age related decline in anaerobic metabolic capacity.

Peak lactate concentration in the blood ($[La]_b$ peak) is thought to provide useful information about anaerobic glycolytic activity in working muscles during supramaximal exercise. Although the value of $[La]_b$ peak as a quantitative estimate of glycolytic energy release can be questioned, the $[La]_b$ peak appears to be able to discriminate between persons of different glycolytic capacity and anaerobic sprint performance (Hill, 1999; Lacour et al., 1990).

We are aware of only few published studies that have examined the relationship between age and lactate accumulation following brief maximal exercise. Using a 30-s cycle sprint as an exercise test, Makrides et al. (1990) and Marsh et al. (1999) determined that older men (ages 60–70 yrs) have a lower $[La]_b$ peak than young men (20–30 yrs). Kindermann and Keul (1977) studied male subjects between the ages of 7 and 58 yrs and found that $[La]_b$ peak after an all-out 400-m run increased up to the age of 25 but then decreased steadily thereafter with increasing age. On the other hand, Reaburn and Mackinnon (1990) compared master male swimmers of different ages (28–80 yrs) and found no significant age related reduction in $[La]_b$ peak following a 100-m swimming test (~60–100 s). A shortcoming in these studies has been the small number, or total lack, of older male subjects over 70 years of age. Moreover, women were not included in any of these studies.

Therefore, in the present study we examined age and sex related differences in blood lactate concentrations after different sprinting events in elite level master runners. On the basis of previous findings of the age-associated changes in muscle properties, we hypothesized that the $[La]_b$ peak following maximal sprinting would decrease with age in master runners of both sexes.

Methods

The present study was part of a larger research project during the XII European Veterans Athletics Championships held in Jyväskylä, Finland, in July 2000 (Korhonen et al., 2003). During the championships, the highest ranked (according to the 100-, 200-, and 400-m performance of the previous year) male (40–88 ys) and female (35–87 yrs) sprinters were invited by personal letter to take part in the

study. Besides these athletes, other sprinters who qualified for semifinals in 100- to 400-m sprint events were contacted after the preliminary heats and were informed about the study. A total of 81 men and 75 women representing 13 countries were included.

The subjects of the study completed a detailed questionnaire (translated into 8 languages) about current and former training and competition performance. They also participated in a brief interview of about 30 min during which additional information on training methods and competition background was obtained. Separate questions were asked concerning the volume of sport-specific training (speed, speed-endurance, jumping, strength exercises) and other training (e.g., aerobic exercises, ball games) during the summer and winter seasons. Body mass and height were also measured during the interview.

IV

On the basis of the questionnaire and personal interview, most of the subjects had in their youth competed in sprint running events and maintained regular year-round training. There were small age related differences in the type and volume of training. However, comparison of race times with current world records showed that the subjects in the various age groups were of a similar elite level. Furthermore, these were the major championships of the year and thus all athletes were expected to be in peak condition.

Written consent was obtained from the subjects, who had been informed about the risks and benefits associated with participation. Approval for this study was granted by the Ethics Committee of the University of Jyväskylä.

DATA COLLECTION AND ANALYSES

The research project was carried out in close cooperation with the local organizing committee. One year prior to the championships, the research plan was presented to the competition organizers and arrangements were made for field laboratory and blood lactate measurements. All data were collected in a manner that would not interfere with the competition.

As a gross indication of energy production from lactic acid accumulation, the runners' $[La]_b$ peak was measured after the 100-m, 200-m, and 400-m finals by a 10-person testing group. Immediately after finishing the run, the athletes were accompanied by their personal testers to a field laboratory 25 m from the finish line. Two recovery blood samples (5 µl) for peak blood lactate concentration were taken from the fingertip within 2–8 min after the runs (100 m at 2 and 5 min; 200 m at 3 and 6 min; 400 m at 5 and 8 min of recovery). In the interim between the two measurements, the athletes were seated and thus their activity during the recovery period was standardized. The timing for the sampling of recovery blood lactate in different sprint events was selected based on previous studies on the kinetics of lactate accumulation following maximal sprint running (Hirvonen et al., 1987; Kindermann and Keul, 1977). The time difference between the consecutive final starts was 10 minutes, which allowed for the collection of blood after each final.

A total of 466 recovery blood samples were obtained. Of these blood samples, two were rejected because the athlete jogged/walked for the latter half of the race course (400 m). Moreover, the running performance (100-m, 200-m) of one elderly male runner did not represent the average performance level of his age group (running times 3 *SD* over the group mean), and consequently we disregarded the

lactate data of that subject. The athletes who participated in the blood lactate measurements represented 48%, 67%, and 67% of all male runners, and 42%, 68%, and 72% of all female runners, in 100-m, 200-m, and 400-m sprint finals, respectively. For the purpose of this study the highest value of blood lactate concentration was included in the analysis.

The $[La]_b$ peak was determined using the Lactate Pro™ (Arkray, Inc., Kyoto, Japan) instruments, which employ an amperometric method with enzymatic reaction. These instruments enabled rapid measurements of lactate concentration in whole blood (in the plasma compartment) under field conditions. The instruments were used in accordance with the manufacturer's instructions. Briefly, they were calibrated before the races by the use of calibration strips. Blood was drawn from the fingertip; the site was standardized for all athletes. After the sampling site was cleaned with an alcohol pad, the skin was dried and punctured using a lancing device. The first drop of blood was wiped off so as to exclude samples with perspiration that could lead to incorrect test results. Using light pressure applied to the flat of the hand, the second drop of blood was drawn at the tip of the reagent strip, and in 1 min the lactate concentration was displayed. We made sure that a sufficient volume of blood was added to the strip. The measuring range of Lactate Pro is from 0.8 to 23.3 mmol/L.

Previous studies have shown that the Lactate Pro gives reliable results for blood samples with high lactate concentrations (up to 20 mmol/L) and exhibits a high degree of accuracy with other laboratory lactate analysers (Pyne et al., 2000; Shimojo et al., 1993). In recent experiments in our laboratory, however, it was found that with blood lactate concentrations above 17 mmol/L the Lactate Pro gave lower values (~10 %) when compared with a standard enzymatic photofluorometry (unpublished observation).

STATISTICS

Differences in the dependent variables between the sexes and age groups were determined by ANOVA. In the case of a significant age effect, Tukey and Tamhane post hoc analyses were used to locate the differences. Comparisons of $[La]_b$ peak of men and women of similar running time were adjusted by using age as a covariate (ANCOVA). The rate of change in performance and $[La]_b$ peak with age was estimated by using both linear and polynomial regression analysis. Pearson's correlation coefficient was used to examine relationships between $[La]_b$ peak and sprint performance. Partial correlation was used to control for the effect of age on these relationships. All data are reported as means and standard deviations (*SD*) unless otherwise stated. Statistical analyses were performed using SPSS 11.0 for Windows (SPSS, Inc., Chicago) and significance was set at $p < 0.05$.

Results

PHYSICAL AND TRAINING CHARACTERISTICS

The physical and training characteristics of the athletes are shown in Tables 1–3. The body mass of the male athletes in the 100-m and 400-m, and of the female athletes in 400-m, was greater in the youngest than in the oldest age groups. The

Table 1 Selected Subject Characteristics ($M \pm SD$) of Male and Female Runners in the 100-m Sprint

Variable	Age Group 35–49	50–59	60–69	>70
n, M/F	6-7/ 6-8	6-7/ 5-8	5-8/ 5	9-13/ 6
Age (yrs)				
Men	44.1 ± 3.2	53.3 ± 3.0	62.0 ± 2.4	79.4 ± 6.1
Women	43.6 ± 3.5	54.9 ± 2.7	61.8 ± 2.4	78.7 ± 6.5
Height (cm)				
Men	177 ± 3[e]	176 ± 7[e]	172 ± 6[e]	169 ± 7[e]
Women	168 ± 7[d]	167 ± 7	166 ± 3	158 ± 8[a]
Body mass (kg)				
Men	80.2 ± 6.4[d,e]	75.0 ± 10.4[e]	71.5 ± 7.7[e]	68.4 ± 7.6[a,e]
Women	60.6 ± 4.4	58.8 ± 8.2	63.6 ± 6.2	57.6 ± 6.6
Training (hrs/wk)				
Men	5.8 ± 1.2	7.3 ± 1.6	6.1 ± 1.3	7.5 ± 4.0
Women	7.0 ± 3.0	8.7 ± 3.3	5.8 ± 1.3	5.1 ± 2.6
Sprint training[f]				
Men	92 ± 14	88 ± 11	89 ± 10	73 ± 22
Women	71 ± 32	77 ± 33	76 ± 20	63 ± 32
Duration (yrs)[g]				
Men	22 ± 12	21 ± 19	32 ± 19	31 ± 18
Women	22 ± 6	30 ± 12	18 ± 6	27 ± 22

Note: Significant difference, $p < 0.05$: [a] from 35–49-yr-olds; [b] from 50–59-yr-olds; [c] from 60–69-yr-olds; [d] from >70-yr-olds; [e] from female group. [f] Percentage of total training volume spent in sprint training. [g] Years of sprint training.

younger men in the 400-m and women in the 100-m and 400-m were also taller than their older counterparts. The men were generally heavier and taller than the women. No significant differences were found between age groups or sexes in training hours or in the percentage of time spent in sprint training.

AGE AND SEX DIFFERENCES IN POSTRACE $[LA]_B$ PEAK

Figure 1 shows the individual $[La]_b$ peak for the 100-m, 200-m, and 400-m runs as a function of age. $[La]_b$ peak after 100- to 400-m sprint events declined ($p < 0.001$–0.05) with increasing age in both sexes. The second-order polynomial model provided the best fit for lactate data, although in the men's 100-m and 200-m the relationship between age and $[La]_b$ peak was nearly linear. Ten-year age-group

Table 2 Selected Subject Characteristics ($M \pm SD$) of Male and Female Runners in the 200-m Sprint

Variable	Age Group 35–49	50–59	60–69	>70
n, M/F	10/ 12-17	11-12/ 11-13	10-13/ 6-7	9-12/ 3
Age (yrs)				
Men	43.9 ± 3.5	54.7 ± 3.3	63.2 ± 2.6	79.6 ± 6.0
Women	42.5 ± 3.9	54.2 ± 3.0	62.9 ± 2.4	79.0 ± 8.5
Height (cm)				
Men	176 ± 4[e]	175 ± 6[e]	175 ± 4[e]	170 ± 5[e]
Women	167 ± 7	167 ± 5	165 ± 4	157 ± 8
Body mass (kg)				
Men	74.7 ± 4.9[e]	74.1 ± 8.5[e]	71.8 ± 6.6[e]	68.2 ± 8.2[e]
Women	60.2 ± 6.7	59.4 ± 5.3	62.6 ± 5.9	55.0 ± 5.5
Training (hrs/wk)				
Men	6.8 ± 1.2	6.5 ± 1.5	6.3 ± 2.7	7.6 ± 3.9
Women	6.4 ± 2.0	7.0 ± 2.4	5.3 ± 1.8	3.8 ± 2.1
Sprint training[f]				
Men	86 ± 13	80 ± 17	77 ± 14	72 ± 19
Women	80 ± 15	69 ± 24	78 ± 21	73 ± 44
Duration (yrs)[g]				
Men	18 ± 10	26 ± 15	30 ± 19	35 ± 20
Women	20 ± 4	28 ± 4	25 ± 5	30 ± 31

Note: Significant difference, $p < 0.05$: [a] from 35–49-yr-olds; [b] from 50–59-yr-olds; [c] from 60–69-yr-olds; [d] from >70-yr-olds; [e] from female group. [f] Percentage of total training volume spent in sprint training. [g] Years of sprint training.

comparisons with ANOVA demonstrated no group differences in $[La]_b$ peak values before the oldest 70+ age group (Table 4). The $[La]_b$ peak after different sprint distances was on average 11%, 6%, and 5% higher (n.s.) in men than in women for the 100-m, 200-m, and 400-m events, respectively. The comparison of male and female runners with similar running times showed no significant differences in $[La]_b$ peak (Figure 2). In both sexes $[La]_b$ peak was lower ($p < 0.001$) in the 100-m than in the two longer distances, whereas $[La]_b$ peak values following the 200-m and 400-m did not differ significantly (data not shown). Similarly, in subjects competing in all events, $[La]_b$ peak following the 100-m differed from that of the two longer events (Figure 3).

Table 3 Selected Subject Characteristics ($M \pm SD$) of Male and Female Runners in the 400-m Sprint

Variable	Age Group 35–49	50–59	60–69	>70
n, M/F	7-10/ 9-15	9-12/ 7-10	5-7/ 7-11	10-15/ 2
Age (yrs)				
Men	43.9 ± 3.5	52.7 ± 2.8	63.6 ± 2.5	78.7 ± 5.3
Women	41.1 ± 4.3	53.9 ± 2.8	63.3 ± 2.4	71.0 ± 0.0
Height (cm)				
Men	179 ± 6[d,e]	175 ± 4[e]	175 ± 6[e]	171 ± 4[a,e]
Women	168 ± 6[d]	166 ± 4	163 ± 4	153 ± 7[a]
Body mass (kg)				
Men	75.6 ± 6.3[d,e]	72.6 ± 7.2[e]	69.8 ± 7.9[e]	67.7 ± 6.5[a,e]
Women	60.6 ± 5.3	59.6 ± 5.8	59.6 ± 4.0	51.0 ± 5.7
Training (hrs/wk)				
Men	7.8 ± 2.5	7.7 ± 1.2	6.6 ± 2.7	5.9 ± 2.6
Women	7.8 ± 2.2	6.2 ± 2.2	5.5 ± 2.2	5.0 ± 4.2
Sprint training[f]				
Men	85 ± 12	70 ± 24	71 ± 6	70 ± 16
Women	72 ± 19	70 ± 17	67 ± 15	58 ± 11
Duration (yrs)[g]				
Men	16 ± 7[d]	25 ± 13	31 ± 18	37 ± 17[a]
Women	20 ± 10	21 ± 11	23 ± 11	18 ± 10

Note: Significant difference, $p < 0.05$: [a] from 35–49-yr-olds; [b] from 50–59-yr-olds; [c] from 60–69-yr-olds; [d] from >70-yr-olds; [e] from female group. [f] Percentage of total training volume spent in sprint training. [g] Years of sprint training.

IV

RUNNING TIMES

As shown in Figure 4, there was a progressive curvilinear increase in running times ($p < 0.001$) with advancing age, and the polynomial model provided a good fit for the data. The women were on average 18.7% (range 13–28%), 18.9% (15–27%), and 19.0% (15–25%) slower than men in 100-m, 200-m, and 400-m events, respectively. The slopes of regression lines for men and women were not significantly different, indicating that the rate of the age-associated slowing of 100- to 400-m running speed was similar between sexes.

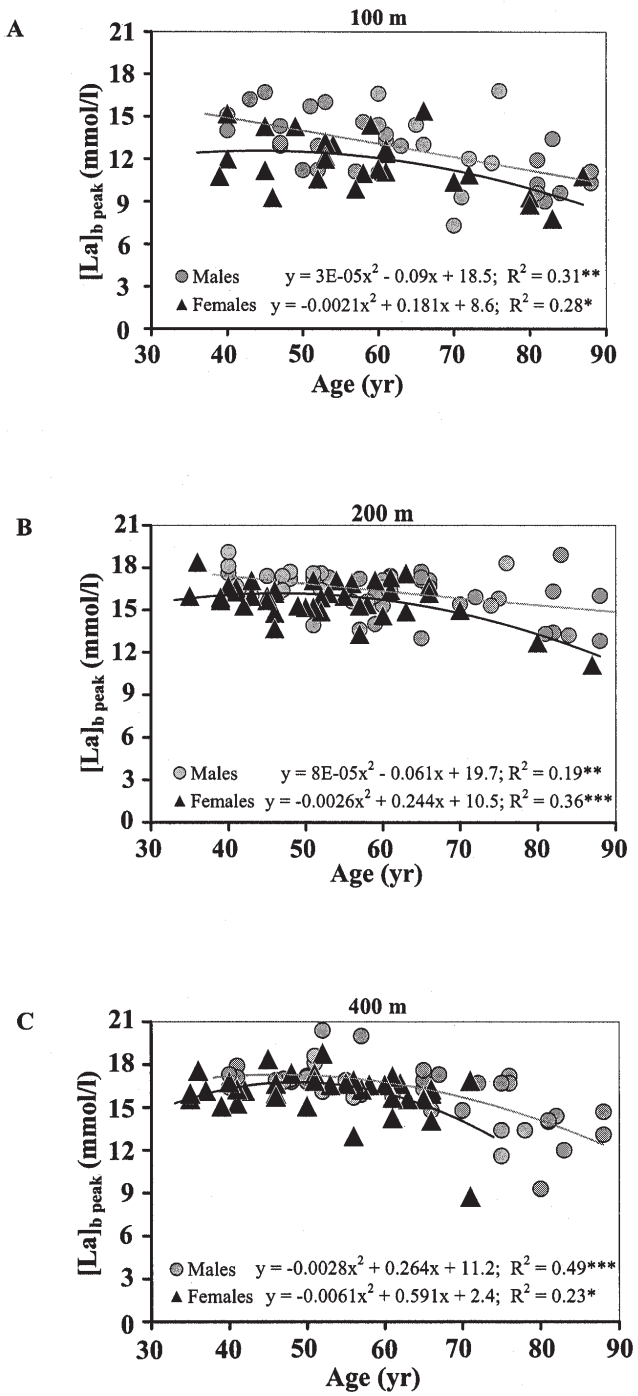

Figure 1. Relationship between age and peak blood lactate concentration after 100-m (A), 200-m (B), and 400-m (C) sprint races. Grey lines represent male regression, black lines represent female regression. Significance of regression equation: $*p < 0.05$, $**p < 0.01$, $***p < 0.001$. Lactate concentrations declined significantly with age in each sprint event. Sex had no effect on this relationship.

Table 4 Peak Blood Lactate Concentration After the 100-m, 200-m, and 400-m Sprint Events in Male and Female Runners

	Age Group			
	35–49	50–59	60–69	>70
100-m *n*: M/F	7/ 8	7/ 8	8/ 5	13/ 6
Men	14.6 ± 1.5[d]	13.2 ± 2.2	13.7 ± 1.6[d]	10.9 ± 2.4[a,c]
Women	12.7 ± 2.1[d]	12.4 ± 1.5	12.6 ± 1.7[d]	9.7 ± 1.2[a,c]
200-m *n*: M/F	10/ 17	12/ 13	13/ 7	12/ 3
Men	17.4 ± 0.9[d]	16.0 ± 1.5	16.6 ± 1.2	15.4 ± 2.0[a]
Women	16.0 ± 1.0[d]	15.8 ± 1.1[d]	16.2 ± 1.1[d]	12.9 ± 2.0[a,b,c]
400-m *n*: M/F	10/ 15	12/ 10	7/ 11	15/ 2
Men	17.0 ± 0.4[d]	17.5 ± 1.5[d]	16.8 ± 0.6[d]	14.1 ± 2.2[a,b,c]
Women	16.3 ± 0.9[d]	16.4 ± 1.5[d]	15.8 ± 0.9	12.9 ± 5.7[a,b]

Note: Significant difference, $p < 0.05$: [a] from the 35–49-yr-olds; [b] from the 50–59-yr-olds; [c] from the 60–69-yr-olds; [d] from the >70-yr-olds.

RELATIONSHIP BETWEEN $[LA]_B$ PEAK AND SPRINT PERFORMANCE

The simple and age-controlled correlation between $[La]_b$ peak and running times are given in Table 5. The $[La]_b$ peak correlated significantly with running times in all sprint distances except for the age-controlled correlation in men for the 100-m and 200-m.

Discussion

The major findings from this study were as follows. First, as hypothesized, $[La]_b$ peak following 100- to 400-m races declined with increasing age, the age related difference becoming more evident after 70 years of age. Second, no significant sex related differences were found in $[La]_b$ peak for any sprint event. Third, $[La]_b$ peak was associated with running times in all sprint distances except for the age-controlled correlation in men for the 100-m and 200-m. To the best of our knowledge, this was the first study to examine the age related differences in blood lactate response to sprint exercise in female master athletes and to evaluate possible sex differences in this respect.

AGE AND SEX DIFFERENCES IN $[LA]_B$ PEAK

In the present study, $[La]_b$ peak declined from 70 years of age in both sexes. Earlier, Kindermann and Keul (1977) reported a significant age related reduction in $[La]_b$ peak after the 400-m run in men from 25 to 58 years of age. Further, two

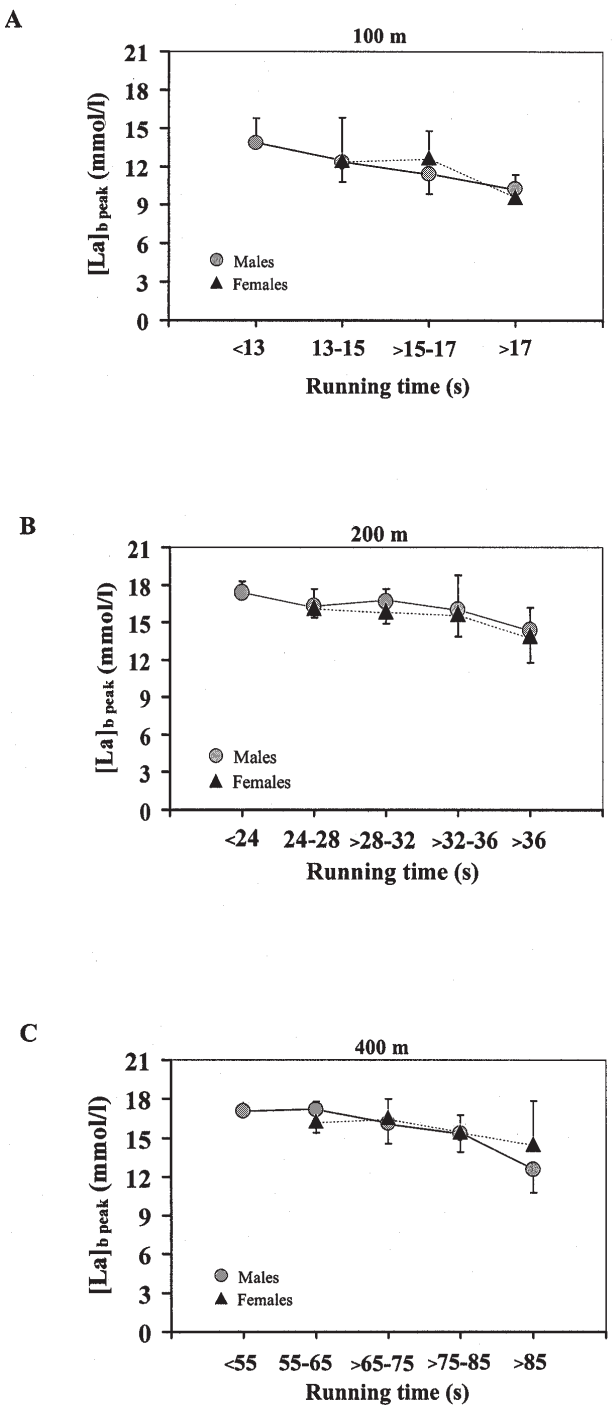

Figure 2. Mean group values ± *SD* of postrace peak blood lactate concentrations in subgroups of male and female athletes of similar running time. Lactate concentrations were similar for men and women in all sprint events.

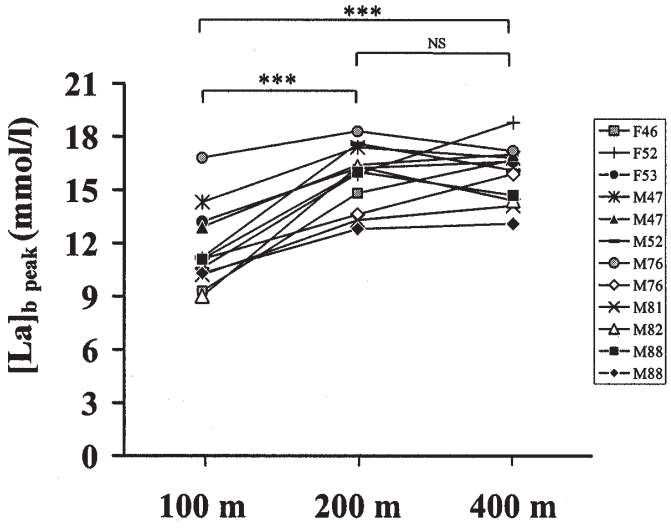

Figure 3. Individual values of postrace peak blood lactate concentrations for 12 athletes who participated in all sprint events. Data points indicating sex (M, F) and age are shown in the box at the right. Mean group value of peak blood latate concentration was significantly lower in 100-m than in 200-m or 400-m races (***$p < 0.001$, NS = not significant).

studies on the metabolic response to all-out 30-s cycle sprint showed that $[La]_b$ peak was significantly lower in men ages 60–70 than in healthy men ages 20–30 years (Makrides et al., 1990; Marsh et al., 1999). The study of Reaburn and Mackinnon (1990), however, showed no age-associated differences in lactate response to a 100-m swimming test in male master athletes ages 28–80. The lack of relationship between $[La]_b$ peak and age in that study may have resulted from the limited number ($n = 4$) of swimmers older than 55. The $[La]_b$ peak values in our older sprint runners as well as in sprint swimmers (Reaburn and Mackinnon, 1990) were considerably higher (>40%) than whole-blood lactate values found in age-matched untrained (Marsh et al., 1999) or resistance-trained (Slade et al., 2002) individuals after a 30-s cycle sprint. When compared with young male runners, the $[La]_b$ peak after 100-m, 200-m, and 400-m of the fastest male runners (40–49 yrs) in this study were about 5%, 10%, and 25% lower, respectively (Kindermann and Keul, 1977; Locatelli and Arsac, 1995). According to these comparisons, it is reasonable to assume that glycolytic energy production declines with the normal aging process but is trainable and, by regular sprint training, can be maintained at a high level into old age. The comparison of lactate data in this study with those previously reported must be viewed with caution, however, due to the differences in the mode and duration of exercise and the methods of assessing blood lactate.

The $[La]_b$ peak did not differ significantly between men and women, although there was a tendency for $[La]_b$ peak to be slightly higher in men (~5–11%)

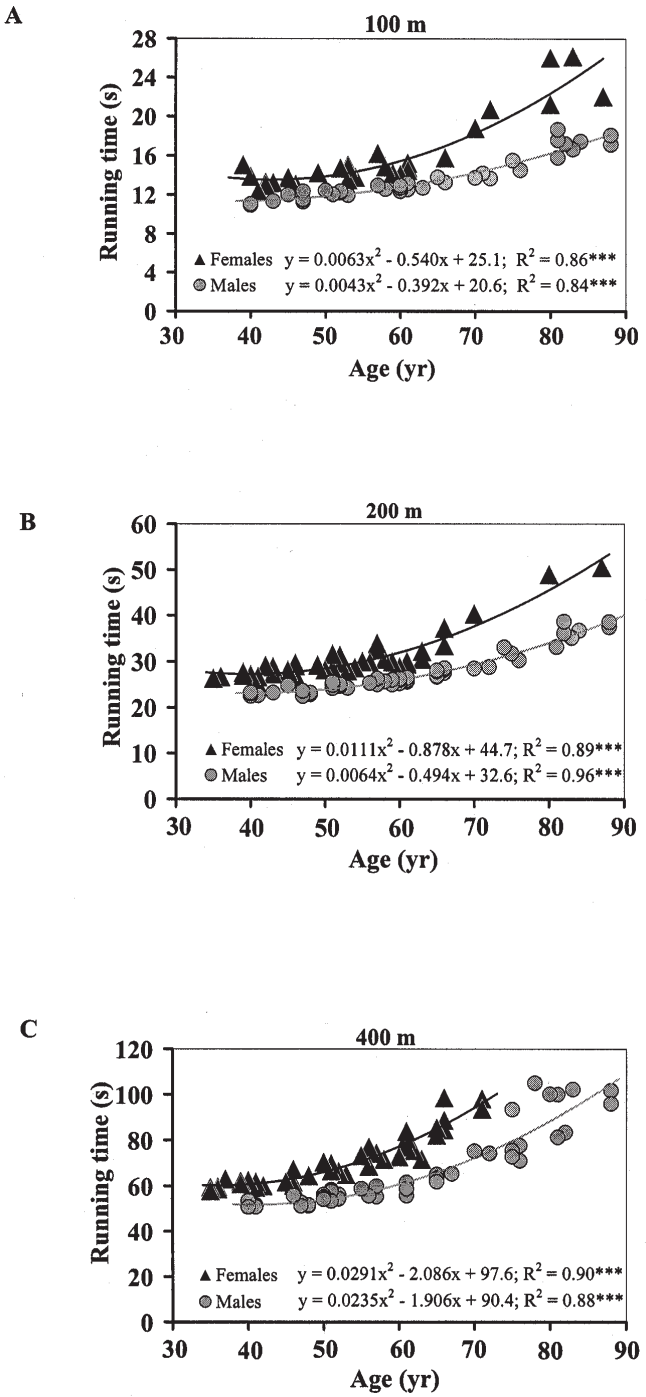

Figure 4. Relationship between age and running time in 100-m (A), 200-m (B), and 400-m (C) sprint races. Grey lines represent male regression, black lines represent female regression. Significance of regression equation, $***p < 0.001$. In each sprint event there was a significant age and sex effect, $p < 0.001$, on running times.

Table 5 Simple and Age-Controlled Correlation Between Postrace Peak Blood Lactate Concentration and Running Time in Different Sprint Events

	Men			Women		
	100-m	200-m	400-m	100-m	200-m	400-m
Lactate/Running time	–0.58 (<0.001)	–0.46 (<0.001)	–0.79 (<0.001)	–0.63 (<0.001)	–0.63 (<0.001)	–0.40 (0.012)
Lactate/Running time controlled for age	–0.19 (0.291)	–0.18 (0.245)	–0.62 (<0.001)	–0.46 (0.018)	–0.49 (0.002)	–0.37 (0.025)

Note: Values are correlation *r* with *p* values in parentheses.

than in women. In line with this, studies of young competitive sprinters have indicated that females attain a $[La]_b$ peak close to that of their male counterparts (Hill, 1999; Kindermann and Keul, 1977; Lacour et al., 1990). The comparison of $[La]_b$ peak of male and female runners of the same age and relative level of performance, however, is complicated by differences in race time. For example, it is possible that the longer running times for females as compared with males could act to increase the $[La]_b$ peak due to a longer period of glycolytic stress. On the other hand, the longer running times of females, especially in the 400-m sprint, could imply a greater involvement of aerobic processes for energy production. To gain more insight into this issue, we compared subgroups of male and female runners with similar running times. The results indicated no differences in lactate accumulation between male and female runners with similar running times (Figure 2). Accordingly, well-controlled studies at both the system and muscular level will be needed in order to establish whether there is a true sex difference in glycolytic energy use with aging.

The $[La]_b$ peak correlated significantly with running time in all sprint events except the age-adjusted correlation in the men's 100-m and 200-m. This suggests that the variations in sprint performance of these master sprinters are related to the anaerobic glycolytic capacity of their muscles. Previous studies in young runners have shown significant relationships between $[La]_b$ peak and sprinting speed over 400-m (Kindermann and Keul, 1977; Lacour et al., 1990; Weyand et al., 1994). In the 100-m and 200-m events, some (Fujitsuka et al., 1982; Weyand et al., 1994), but not all researchers (Hautier et al., 1994), have found that lactate levels were correlated with performance. The correlation between $[La]_b$ peak and performance in our study was relatively weak, which has also been the finding of other studies undertaken in younger athletes (Fujitsuka et al., 1982; Weyand et al., 1994). This may be because short sprinting is also dependent on the contribution of other energy processes and biomechanical factors.

POSSIBLE EXPLANATIONS FOR AGE RELATED DIFFERENCES IN $[LA]_B$ PEAK

Although the mechanisms underlying the age related decline in blood lactate accumulation after sprint exercise have not been systematically studied, the available physiological studies in humans and animals have identified certain potentially important factors for this decline. It is known, for example, that lactic acid formation is closely related to the mass of contracting muscle and number of muscle fibers recruited (e.g., Stainsby and Brooks, 1990). In the current study there was a trend for age related decline in body mass after age 70, and thus it is likely that the elderly runners exhibited a loss of active muscle mass which may have contributed to the reduction in lactate values. Furthermore, the loss of muscle mass with aging is thought to occur primarily via atrophy of the fast-twitch fibers (Porter et al., 1995), in which lactic acid production is higher than in slow-twitch fibers (Essén and Häggmark, 1975; Tesch et al., 1978). Our recent findings suggest that highly trained master sprinters have above average fast-twitch fiber size in the quadriceps muscles, but they too experience the typical age related reduction of fast fiber area (Korhonen et al., unpublished observation). Aging may also reduce maximal lactate accumulation due to decreased levels of the enzymes that regulate anaerobic glycolysis (Hunter et al., 2002; Keh-Evans et al., 1992). Accordingly, age changes in muscle characteristics could have played a role in reduced lactate production in the older sprinters in this study. Furthermore, it is noteworthy that one new study using ^{31}P-MRS during maximal voluntary isometric contractions (16 s, 60 s) indicates that older subjects rely on oxidative ATP production more than young subjects and derive a smaller proportion of their ATP from anaerobic glycolysis (Lanza, Befroy, and Kent-Braun, 2005). Consequently, one cannot exclude the possibility that age decline in $[La]_b$ peak can be due partly to a preferential reliance on oxidative metabolism during short maximal exercise.

A general observation is that high-intensity sprint training increases $[La]_b$ peak (Jacobs et al., 1987; Mero et al., 1993). Based on our questionnaire and interview, the percentage of time spent in sprint training tended to decrease in the oldest age groups. Thus the age related differences in $[La]_b$ peak may partly be the result of differences in training adaptations. It was of special interest to note that contrary to the case with young runners (e.g., Kindermann and Keul, 1977), the $[La]_b$ peak was similar in the 200-m and 400-m races in these master runners (see Figure 3). Thus it is tempting to speculate that in young runners the sprint training has stressed more the anaerobic lactic acid energy yield and has thus improved the buffer capacity (i.e., the ability to keep the concentration of hydrogen ions $[H^+]$ at a low level) of their muscles more than for the master runners in this study (Parkhouse et al., 1985; Sharp et al., 1986). An increased buffer capacity would enable more prolonged utilization of glycolysis before reaching a limiting pH, thus allowing an increase in lactate accumulation in the blood with sprint distances up to 400-m. Interestingly, a recent biochemical study suggests that lactate production itself is a consumer of H^+ and thus acts as buffering system against acidosis and fatigue during exhaustive exercise (Robergs et al., 2004).

The decrease in lactate accumulation with increasing age may also be associated with differences in race intensity, although the cause-effect relationship between race performance and lactate production is difficult to infer. One could ar-

gue that slower running velocities, independent of age, accounted for lower levels of lactate in older sprinters. The above findings, taken together, suggest that the possible reasons for age related differences in lactate accumulation after all-out sprint exercise are many and interrelated.

METHODOLOGICAL CONSIDERATIONS

The present study has at least three important limitations. First, due to the cross-sectional nature of the data, one could argue that the true age effect on sprint performance and $[La]_b$ peak could be masked by the study design. Comparison of the present cross-sectional data with the longitudinal data in highly trained master male sprinters (Conzelmann, 1997) suggests that the rate of decline in running performance with advancing age may be slightly slower when studied longitudinally. As far as we know, the effect of age on $[La]_b$ peak after brief maximal exercise (< 2 min) has not been examined using longitudinal approaches. Therefore longitudinal studies are needed so as to better understand the effect of age on performance and $[La]_b$ peak.

Second, an objection can be made to the use of blood lactate as an indicator of energy supply from anaerobic glycolysis. Blood lactate represents the balance of production, release, and removal of lactate, and thus underestimates the lactic acid production/glycolytic activity in working muscle. Studies in young adults, however, have indicated that blood lactate levels after supramaximal performances correlate strongly ($r = 0.82–0.88$) with muscle lactate at the cessation of sprinting (Cheetham et al., 1986; Hirvonen et al., 1992). Further, to our knowledge there is no evidence to suggest that aging compromises the release of lactate from muscle to blood during brief maximal exercise, thereby changing the prediction of $[La]_b$ peak for muscle lactic acid formation.

Third, in this study there is a lack of data for elderly women over 70 years of age, especially in the 200-m and 400-m sprint events. A marked reduction in muscle mass in old age might be expected to cause a decline in postexercise lactate response among the oldest female athletes. Therefore, larger samples of the older runners would have provided more reliable results of the age related differences in $[La]_b$ peak in a wide age range in women.

A strength of this study is that we measured $[La]_b$ peak under competition conditions. It has been suggested that although data for maximal aerobic capacity can be obtained using a treadmill, a reliable measure of maximal anaerobic contribution requires competitive effort (Hill, 1999; Lacour et al., 1990). In support of this, it has been observed that sprint athletes are able to reach considerably higher $[La]_b$ peak in important competition than in a maximal treadmill test, despite the same exercise time (Hill, 1999; Lacour et al., 1990). Given the major international competition as an experimental condition, it is plausible that the athletes in this study exerted maximal effort and achieved a true measure of their $[La]_b$ peak.

CONCLUSION

The results of the present cross-sectional study show that $[La]_b$ peak declines with advanced age in male and female master sprinters and may be a factor in the deterioration in sprint performance. The $[La]_b$ peak of the athletes, however, was con-

siderably higher than that previously reported for age-matched untrained men and women, suggesting a favorable influence of long-term sprint training on anaerobic glycolytic capacities during aging. Our results also indicate that there are no significant sex differences in lactate response to sprinting in elite master runners. The observed decline in $[La]_b$ peak in the oldest sprinters implies decreased ability to generate energy from anaerobic glycolysis, which is probably related to changes in skeletal muscle characteristics. Further studies are needed to elucidate the interaction of age-associated changes in musculature and energy production during anaerobic exercise, and to understand the potential benefits of exercise for the anaerobic energy system in aging persons.

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References

Cheetham, M.E., Boobis, L.H., Brooks, S., and Williams, C. (1986). Human muscle metabolism during sprint running. **J. Appl. Physiol.** 61: 54-60.

Conzelmann, A. (1997). The plasticity of courses of physical performance in the second part of life. In: G. Huber (Ed.), **Healthy Aging, Activity and Sports**, pp. 435-445. Gamburg: Health Promotion Publications.

Essén, B., and Häggmark T. (1975). Lactate concentration in type I and II muscle fibers during muscular contraction in man. **Acta Physiol. Scand.** 95: 344-346.

Fujitsuka, N., Yamamoto, T., Ohkuwa, T., Santo, M., and Miyarnura, M. (1982). Peak blood lactate after short periods of maximal treadmill running. **Eur. J. Appl. Physiol.** 48: 289-296.

Greenhaff, P.L., Nevill, M.E., Soderlund, K., Bodin, K., Boobis, L.H., Williams, C., and Hultman, E. (1994). The metabolic responses of human type I and II muscle fibres during maximal treadmill sprinting. **J. Physiol.** 478: 149-155.

Hautier, C.A., Wouassi, D., Arsac, L.M., Bitanga, E., Thiriet, P., and Lacour, J.R. (1994). Relationship between postcompetition blood lactate concentration and average running velocity over 100-m and 200-m races. **Eur. J. Appl. Physiol.** 68: 508-513.

Henriksson, J., and Reitman, J.S. (1976). Quantitative measures of enzyme activities in type I and type II muscle fibers of man after training. **Acta Physiol. Scand.** 97: 392-397.

Hill, D.W. (1999). Energy system contributions in middle-distance running events. **J. Sports Sci.** 17: 477-483.

Hirvonen, J., Nummela, A., Rusko, H., Rehunen, S., and Härkönen, M. (1992). Fatigue and changes of ATP, creatine phosphate, and lactate during the 400-m sprint. **Can. J. Sport Sci.** 17: 141-144.

Hirvonen, J., Rehunen, S., Rusko, H., and Härkönen, M. (1987). Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. **Eur. J. Appl. Physiol**. 56: 253-259.

Hunter, G.R., Newcomer, B.R., Weinsier, R.L., Karapondo, D.L., Larson-Meyer, D.E., Joanisse, D.R., and Bamman, M.M. (2002). Age is independently related to muscle metabolic capacity in premenopausal women. **J. Appl. Physiol.** 93: 70-76.

IV

Jacobs, I., Esbjörnsson, M., Sylven, C., Holm, I., and Jansson, E. (1987). Sprint training effects on muscle myoglobin, enzymes, fiber types, and blood lactate. **Med. Sci. Sports Exerc.** 19: 368-374.

Keh-Evans, L., Rice, C.L., Noble, E.G., Paterson, D.H., Cunningham, D.A., and Taylor, A.W. (1992). Comparison of histochemical, biochemical, and contractile properties of triceps surae of trained aged subjects. **Can. J. Aging** 11: 412-425.

Kindermann, W., and Keul, J. (1977). **Anaerobe Energiebereitstellung im Hochleistungssport** [Anaerobic capacity in elite sports]. Schorndorf: Verlag Karl Hofmann.

Korhonen, M.T., Mero, A., and Suominen, H. (2003). Age-related differences in 100-m sprint performance in male and female master runners. **Med. Sci. Sports Exerc.** 35: 1419-1428.

Lacour, J.R., Bouvat, E., and Barthelemy, J.C. (1990). Post-competition blood lactate concentrations as indicators of anaerobic energy expenditure during 400-m and 800-m races. **Eur. J. Appl. Physiol.** 61: 172-176.

Lanza, I.R., Befroy, D.E., and Kent-Braun, J.A. (2005). Age-related changes in ATP-producing pathways in human skeletal muscle in vivo. **J. Appl. Physiol.** 99: 1736-1744.

Locatelli, E., and Arsac, L. (1995). The mechanics and energetics of the 100m sprint. **New Stud. Athletics** 10: 81-87.

Makrides, L., Heigenhauser, G.J.F., and Jones, N.L. (1990). High-intensity endurance training in 20- to 30- and 60- to 70-yr-old healthy men. **J. Appl. Physiol.** 69: 1792-1798.

Marsh, G.D., Paterson, D.H., Govindasamy, D., and Cunningham, D.A. (1999). Anaerobic power of the arms and legs of young and older men. **Exp. Physiol.** 84: 589-597.

Mero, A., Rusko, H., Peltola, E., Pullinen, T., Nummela, A., and Hirvonen, J. (1993). Aerobic characteristics, oxygen debt and blood lactate in speed endurance athletes during training. **J. Sports Med. Phys. Fitness** 33: 130-136.

Newsholme, E.A., Blomstrand, E., McAndrew, N., and Parry-Billings, M. (1992). Biochemical causes of fatigue and overtraining. In: R.J. Shephard and P.-O. Åstrand (Eds.), **Endurance in Sport,** pp. 351-364. Oxford: Blackwell Scientific.

Parkhouse, W.S., McKenzie, D.C., Hochachka, P.W., and Ovalle, W.K. (1985). Buffering capacity of deproteinized human vastus lateralis muscle. **J. Appl. Physiol.** 58: 14-17.

Porter, M.M., Vandervoort, A.A., and Lexell, J. (1995). Aging of human muscle: Structure, function and adaptability. **Scand. J. Med. Sci. Sports** 5: 129-142.

Pyne, D.B., Boston, T., Martin, D.T., and Logan, A. (2000). Evaluation of the Lactate Pro blood lactate analyser. **Eur. J. Appl. Physiol.** 82: 112-116.

Reaburn, P.R.J., and Mackinnon, L.T. (1990). Blood lactate responses in older swimmers during active and passive recovery following maximal sprint swimming. **Eur. J. Appl. Physiol.** 61: 246-250.

Rehunen, S., Näveri, H., Kuoppasalmi, K., and Härkönen, M. (1982). High-energy phosphate compounds during exercise in human slow-twitch and fast-twitch muscle fibers. **Scand. J. Clin. Lab. Invest.** 42: 499-506.

Robergs, R.A., Ghiasvand, F., and Parker, D. (2004). Biochemistry of exercise-induced metabolic acidosis. **Am. J. Physiol. Regul. Integr. Comp. Physiol.** 287: R502-R516.

Saubert, C.W., Armstrong, R.B., Shepherd, R.E., and Gollnick, P.D. (1973). Anaerobic enzyme adaptations to sprint-training in rats. **Pflügers Arch.** 341: 305-312.

Sharp, R.L., Costill, D.L., Fink, W.J., and King, D.S. (1986). Effects of eight weeks of bicycle ergometer sprint training on human muscle buffer capacity. **Int. J. Sports Med.** 7: 13-17.

Shimojo, N., Naka, K., Uenoyama, H., Hamamoto, K., Yoshioka, K., and Okuda, K. (1993). Electrochemical assay system with single-use electrode strip for measuring lactate in whole blood. **Clin. Chem.** 39: 2312-2314.

Slade, J.M., Miszko, T.A., Laity, J.H., Agrawal, S.K., and Cress, E. (2002). Anaerobic power and physical function in strength-trained and non-strength-trained older adults. **J. Gerontol.** 57A: M168-M172.

Stainsby, W.N., and Brooks, G.A. (1990). Control of lactic acid metabolism in contracting muscles and during exercise. In: K.B. Pandolf and J.O. Holloszy (Eds.), **Exercise and Sport Sciences Reviews**, pp. 29-63. Philadelphia: Williams and Wilkins.

Staudte, H.W., Exner, G.U., and Pette, D. (1973). Effects of short-term high-intensity (sprint) training on some contractile and metabolic characteristics of fast and slow muscle of rat. **Pflügers Arch**. 344: 159-168.

Tesch, P., Sjödin, B., and Karlsson, J. (1978). Relationship between lactate accumulation, LDH activity, LDH isozyme and fibre type distribution in human skeletal muscle. **Acta Physiol. Scand.** 103: 40-46.

Tesch, P.A., Thorsson, A., and Fujitsuka, N. (1989). Creatine phosphate in fiber types of skeletal muscle before and after exhaustive exercise. **J. Appl. Physiol.** 66: 1756-1759.

Weyand, P.G., Cureton, K.J., Conley, D.S., Sloniker, M.A., and Liu, Y.L. (1994). Peak oxygen deficit predicts sprint and middle-distance track performance. **Med. Sci. Sports Exerc.** 26: 1174-1180.

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PAPER V

AGING, MUSCLE FIBER TYPE AND CONTRACTILE FUNCTION IN SPRINT-TRAINED ATHLETES

by

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

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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 (V_o) 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 IIa. Specific tension of type I and IIa MHC fibers did not differ between younger and older subjects. V_o of fibers expressing type I MHC was lower ($P < 0.05$) in older than in younger subjects, but there was no difference in V_o of type IIa 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 V_o 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 IIa 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 Klitgaard 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 IIa MHC isoforms from the

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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 conformed 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 determined at the midregion of the vastus lateralis muscle (biopsy site) by a B-mode ultrasound instrument (SSD-1400, Aloka, Japan) as described previously (39). Briefly, during the ultrasound scanning procedure a 5-cm linear-array probe (7.5 MHz) was positioned perpendicular to the surface of the muscle and in the ultrasound images the subcutaneous adipose tissue layer and superior and inferior aponeurosis, and a number of muscle fiber fascicles between aponeuroses were identified. Muscle thickness was determined as the distance from the adipose tissue-muscle interface to the intermuscular interface. The muscle fiber pennation angle was measured as the angle between the fiber fascicle and the deep aponeuroses. From the muscle thickness and fiber pennation angle, the fiber fascicle length across the deep and superficial aponeuroses was estimated as follows: fiber fascicle length = muscle thickness·sin (fiber pennation angle)$^{-1}$. The physical characteristics of the subjects in the age groups are shown in Table 1.

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 $MgCl_2$, 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 skinning 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 desucrosed 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,

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

Variable	Age Group					*P*
	18–33 yr	40–49 yr	50–59 yr	60–69 yr	70–84 yr	
N	16	16	18	21	20	
Age, yr	24.3±1.0	44.0±0.9	53.9±0.6	65.8±0.6	75.3±0.9	
Height, cm	178.0±1.1[d,e]	180.5±1.9[c,d,e]	175.5±1.1[b]	172.7±0.9[a,b]	171.1±1.2[a,b]	<0.001
Body mass, kg	77.2±1.4[e]	79.7±1.9[d,e]	74.3±1.4	71.2±0.9[b]	69.8±2.0[a,b]	<0.001
Body fat, %	16.5±0.9	13.3±1.0	14.9±0.9	13.6±1.0	15.0±1.0	NS
Thigh length, cm	45.0±0.5	47.1±0.6	46.1±0.6	45.0±0.4	45.1±0.7	NS
Thigh circumference, cm	55.9±0.6[c,d,e]	54.5±0.7[d,e]	52.2±0.5[a,e]	50.7±0.5[a,b]	48.7±0.8[a,b,c]	<0.001
VL thickness, cm	2.61±0.08[c,d,e]	2.35±0.09[e]	2.08±0.11[a]	2.10±0.09[a]	1.96±0.08[a,b]	<0.001
VL fascicle length, cm	7.91±0.49	7.90±0.35	7.71±0.41	7.99±0.27	7.38±0.27	NS

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 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 groups, respectively. NS, not significant.

they were not included in the analyses. Figure 1*A* 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 IIAB, 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 Protean 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. 1*B*. 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.

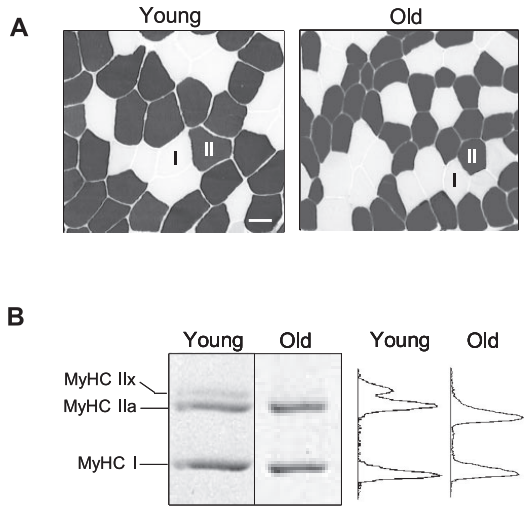

Fig. 1. Examples of enzyme histochemistry and gel electrophoresis of muscle samples from runners aged 26 (Young) and 71 yr (Old). *A*: cryosections stained for myofibrillar ATPase after preincubation at pH 10.30 showing slow type I (I) and fast type II fibers (II). Scale bar, 50 μm. Note the larger difference between the younger and older muscle in the area of type II compared with type I fibers. *B*: portion of a 6% SDS-polyacrylamide gel with densitometric scans used for determination of myosin heavy chain (MyHC) isoform content of the muscle homogenates. The sample from the younger subject shows all 3 MyHC isoform bands, whereas no IIx MyHC was detected in the older muscle.

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 2.66–2.85 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 (P_o) 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 Mg^{2+}, 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 Ca^{2+} concentrations were 10^{-9} M (relaxing solution) and $10^{-4.5}$ M (maximum activation solution) and are expressed as pCa ($-\log [Ca^{2+}]$). Apparent stability constants for Ca^{2+}-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 Ca^{2+}-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 (V_o) 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 V_o for that fiber. P_o 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 V_o 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 P_o changed more than 10% from first to final activation, or if SL during isometric

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 wore 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° = full extension). On a verbal command, the subjects performed a maximal isometric leg extension as fast as possible over 2.5–4 s. Each testing session consisted of two practice contractions followed by three to four maximum-effort trials with 1- to 1.5-min rest periods.

The force signal was recorded on a computer (486 DX-100) and subsequently digitized and analyzed by a Codas computer system (Data Instruments). Maximal isometric force (F_{max}) was defined as the highest force value recorded during the contraction. The entire force-time curve was analyzed according to the guidelines of Viitasalo et al. (72, 73). In the force-time curves, the times taken to increase force from contraction onset to the levels of 100, 250, 500, 750, 1,000, 1,500 and 2,000 N (absolute scale), and the times needed to increase force from the start of contraction to 10–90% of F_{max} in 10% increments (relative scale) were calculated. The maximal rate of force development (RFD) was determined as the greatest increase in force in a given 50-ms time period. Normalized maximal rate of force development (normalized RFD), a measure of the slope of the force-time curve when normalized with respect to maximal force, was obtained by dividing the absolute RFD by the F_{max} for the subject (expressed as % of F_{max} per second). The test-retest *r* values of the two best efforts were within the range 0.96–0.99 for F_{max} and 0.85–0.98 for RFD, and the CVs were between 2.7–4.2% for F_{max} and 4.9–9.0% for RFD in the different age groups.

Dynamic explosive strength was evaluated by means of a vertical countermovement 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. 3*B* 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. 2*A*). 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, IIAB/I, and IIB/I fiber area ratios with age (Fig. 3*A*). 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*

Variable	Age Group					*P*
	18–33 yr	40–49 yr	50–59 yr	60–69 yr	70–84 yr	
Training						
Years of sprint training	13.2±1.3[c,d,e]	28.1±2.0	28.8±2.8[a]	35.1±4.2[a]	34.3±4.9[a]	0.001
Training hours, h/wk	11.5±0.6[b,c,d,e]	6.7±0.7[a]	7.6±0.7[a]	6.1±0.5[a]	5.9±0.7[a]	<0.001
Frequency, sessions/wk	5.9±0.3b[c,d,e]	4.9±0.4	4.4±0.2[a]	4.2±0.3[a]	4.0±0.3[a]	<0.001
Strength training, h/wk	5.2±0.4[b,c,d,e]	2.2±0.5[a,d]	1.5±0.3[a]	0.8±0.1[a,b]	0.9±0.2[a]	<0.001
Performance†						
60-m sprint, s	7.00±0.03[b,c,d,e]	7.62±0.09[a,c,d,e]	8.03±0.09[a,b,d,e]	8.61±0.08[a,b,c,e]	9.53±0.19[a,b,c,d]	<0.001
Vertical jump, cm	52.5±1.62[b,c,d,e]	42.0±0.97[a,c,d,e]	33.1±0.89[a,b,d,e]	28.3±0.91[a,b,c,e]	22.7±0.99[a,b,c,d]	<0.001
Isometric force, N	3,865±233[c,d,e]	3,307±187[d,e]	2,872±186[a]	2,440±120[a,b]	2,310±175[a,b]	<0.001

Values are means ± SE. ANOVA *P* values and location of significant differences are shown. [a,b,c,d,e]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. †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.

Table 3. *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*

	Type I			Type I/IIa			Type IIa			Type IIax			Type IIx		
Age group	Area, μm²	ST, N/cm²	V_o, ML/s	Area, μm²	ST, N/cm²	V_o, ML/s	Area, μm²	ST, N/cm²	V_o, ML/s	Area, μm²	ST, N/cm²	V_o, ML/s	Area, μm²	ST, N/cm²	V_o, ML/s
18–33 yr (N = 8)	4,300±170 (n = 35)	26.4±1.8	0.63±0.03	2,400 (n = 1)	22.0	0.83	4,700±140 (n = 24)	30.4±3.3	1.75±0.15	3,700±190 (n = 4)	29.9±2.7	2.44±0.99	3,200±680 (n = 2)	33.1±1.5	4.21±1.32
53–77 yr (N = 9)	3,200±170 (n = 36)	32.5±2.2	0.48±0.04	4,500 (n = 1)	41.1	0.84	3,000±240 (n = 29)	36.1±2.8	1.67±0.13	4,200±510 (n = 7)	37.7±4.8	3.37±0.59	2,800±160 (n = 5)	22.5±1.9	3.43±0.77
P	<0.05	NS	<0.05				<0.05	NS	NS						

Values are means ± SE. P values of level of significance are given. Statistical analyses have been restricted to muscle fibers expressing the type I or IIa MHC isoform, because of the small number of fibers expressing other isoforms or combination of isoforms. N, number of subjects; n, number of fibers tested; ST, specific tension; V_o, maximum velocity of unloaded shortening; ML/s, muscle lengths per second; NS, not significant.

type IIA (6.7%/decade), IIAB (7.5%/decade), or IIB (11.3%/decade) fibers. When the cross-sectional area of fast and slow fibers was compared within the age groups, type I fibers were smaller than type IIA and/or IIAB fibers in the three youngest age groups but were larger than type IIAB and IIB in the two oldest groups (Fig. 3*B*). Among the fast fiber population, the area of type IIA fiber was larger than that of type IIAB and type IIB in all age groups ($P < 0.001$–0.01), whereas a difference between type IIAB and type IIB was observed in the 40- to 49-yr ($P < 0.01$) and 50- to 59-yr ($P < 0.05$) groups (data not shown).

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 IIB 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

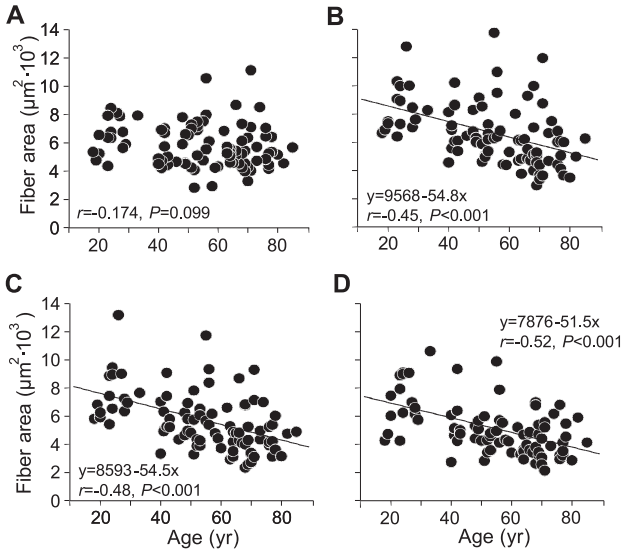

Fig. 2. Relationship between age and mean cross-sectional area of type I (*A*) and different type II muscle fibers [IIA(*B*), IIAB(*C*), and IIB (*D*)]. Regression lines are shown for cross-sectional areas of type II fibers, which declined significantly with age. The slope of the regression line among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

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)→I/IIa→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.

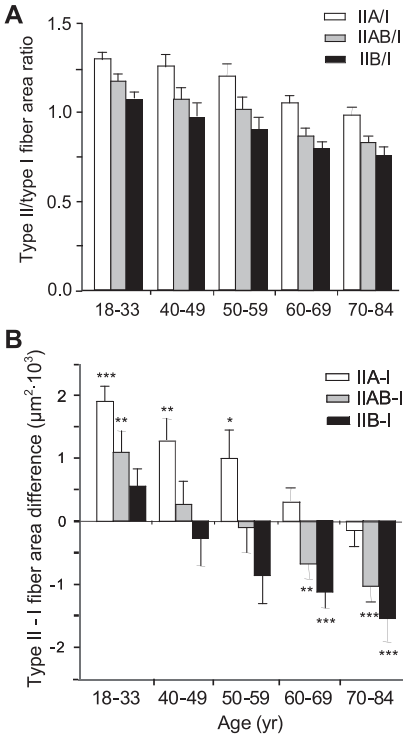

Fig. 3. *A*: ratio of type II-to-type I fiber mean cross-sectional areas of vastus lateralis muscle in different age groups. There was a significant decline ($P < 0.001$) in type IIA/I, IIAB/I and IIB/I fiber area ratios with age. *B*: difference between the mean cross-sectional area (μm^2) of each type II and type I fibers in different age groups. ***$P < 0.001$, **$P < 0.01$, *$P < 0.05$ compared with type I fiber.

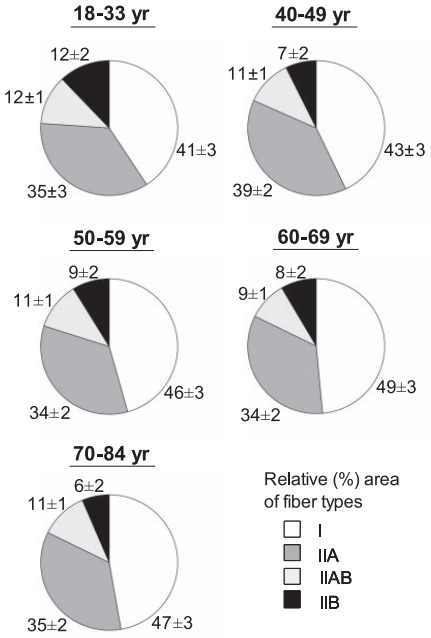

Fig. 4. Proportion (%) of overall muscle fiber cross-sectional area occupied by different fiber types in the vastus lateralis muscle of subjects in different age groups. Relative area of type I fibers increased with age ($P < 0.05$) whereas that of type IIB showed aging-associated decline ($P = 0.05$).

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. 6*A*). Correspondingly, there was an aging-associated decline ($P < 0.001$) in the absolute RFD. The loss of RFD from 25,270 ± 1,576 N/s in the youngest group to 13,216 ± 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 ± 4 ms (18–33 yr) to 104 ± 12 ms (70+ 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_{max}, with the significance of association being strongest at force levels between 10–50% of the maximum (Fig. 6*B*). Moreover, adjustment for F_{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_{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_{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_{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-

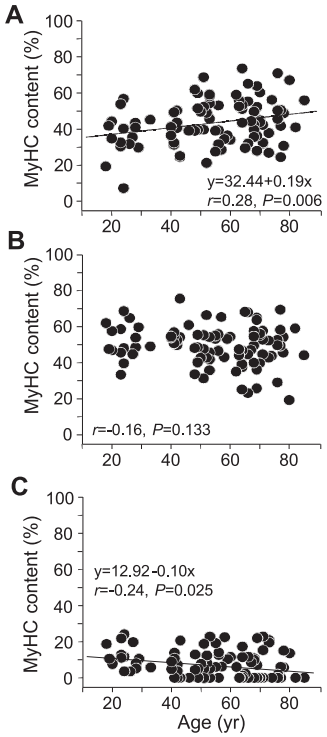

Fig. 5. Relationship between age and relative content of MyHC I (*A*), IIa (*B*), and IIx (*C*) isoforms. Lines in *A* and *C* are linear regressions.

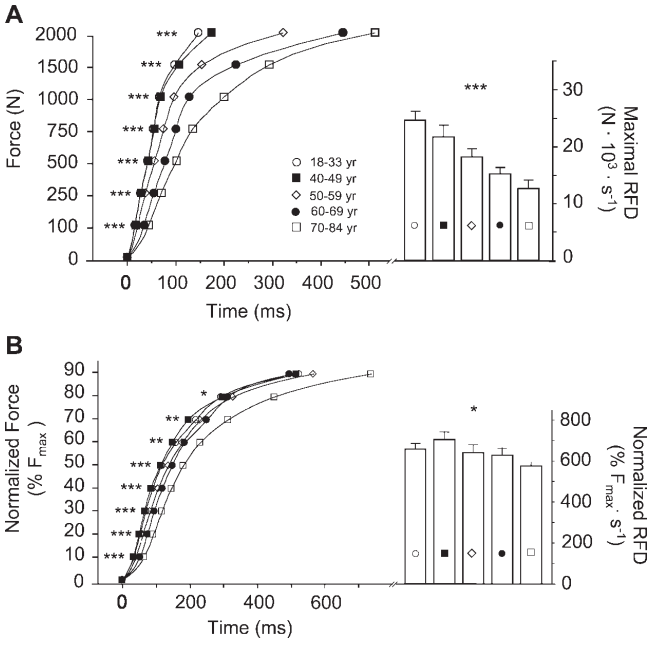

Fig. 6. Force-time curves (up to 2,000 N) and maximal rate of force development (RFD) calculated for absolute values (*A*), and normalized force-time curves and RFD calculated as a percentage of maximal force (F_{max}) developed (*B*) in fast isometric contraction in different age groups. RFD was obtained from the slope of force-time curve (Δforce/Δtime). Asterisks at the side of force-time curve and above the bars indicate significant age effect obtained by the regression analyses: ***$P < 0.001$; **$P < 0.01$; *$P < 0.05$.

V

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. The V_o was significantly slower in type I but not in type IIa MHC fibers from the older subjects. *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. 7*A*, 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).

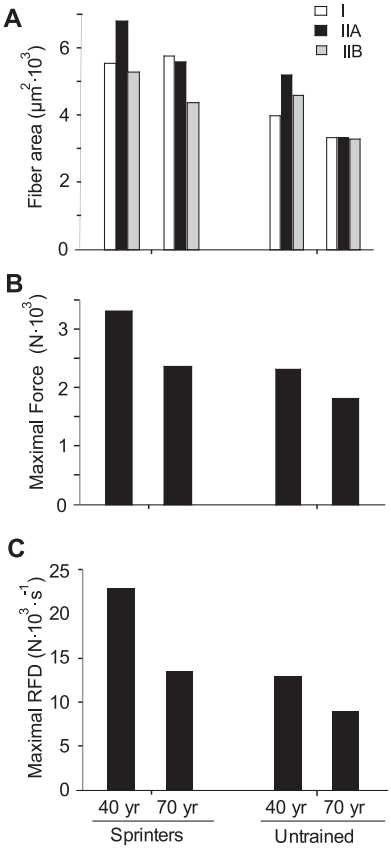

Fig. 7. Cross-sectional area of type I, IIA, and IIB fibers of vastus lateralis (*A*), and maximal force (*B*) and rate of force development (*C*) in fast isometric contraction for selected groups of middle-aged and older sprinters and untrained men of comparable age. Data for untrained men were obtained using identical measurements and analyses by Häkkinen et al. (22–26).

MHC isoform expression. 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 (28, 36, 60) and endurance-trained people (36) 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. 5*C*). Previous research on young subjects (1, 2) 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 β/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. 6*A*). 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. 6*B*). 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 F_{max}) 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 F_{max} 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 F_{max} 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 V_o 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.

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REFERENCES

1. **Andersen JL and Aagaard P.** Myosin heavy chain IIX overshoot in human skeletal muscle. *Muscle Nerve* 23: 1095–1104, 2000.
2. **Andersen JL, Klitgaard H, and Saltin B.** Myosin heavy chain isoforms in single fibres from m. vastus lateralis of sprinters: influence of training. *Acta Physiol Scand* 151: 135–142, 1994.
3. **Avigad G, Kniep A, and Bailin G.** Reaction of rabbit skeletal myosin with D-glucose 6-phosphate. *Biochem Mol Biol Int* 40: 273–284, 1996.
4. **Balagopal P, Rooyackers OE, Adey DB, Ades PA, and Nair KS.** Effects of aging on in vivo synthesis of skeletal muscle myosin heavy-chain and sarcoplasmic protein in humans. *Am J Physiol Endocrinol Metab* 273: E790–E800, 1997.
5. **Bergström J.** Muscle electrolytes in man. Determination by neutron activation analysis on needle biopsy specimens. A study on normal

subjects, kidney patients and patients with chronic diarrhoea. *Scand J Clin Lab Invest* 14 (*Suppl* 68): 1–110, 1962.
6. **Bosco C and Komi PV.** Influence of aging on the mechanical behavior of leg extensor muscles. *Eur J Appl Physiol* 45: 209–219, 1980.
7. **Brooke MH and Kaiser KK.** Muscle fiber types: how many and what kind? *Arch Neurol* 23: 369–379, 1970.
8. **Brown MR and Knull HR.** Effects of nonenzymatic glycation of subfragment-1 of myosin on interactions with actin. *Biochem Cell Biol* 70: 617–622, 1992.
9. **Clarkson PM, Kroll W, and Melchionda AM.** Age, isometric strength, rate of tension development and fiber type composition. *J Gerontol A Biol Sci Med Sci* 36: 648–653, 1981.
10. **Coggan AR, Spina RJ, King DS, Rogers MA, Brown M, Nemeth PM, and Holloszy JO.** Histochemical and enzymatic comparison of the gastrocnemius muscle of young and elderly men and women. *J Gerontol A Biol Sci Med Sci* 47: B71–B76, 1992.
11. **D'Antona G, Pellegrino MA, Adami R, Rossi R, Carlizzi CN, Canepari M, Saltin B, and Bottinelli R.** The effect of ageing and immobilization on structure and function of human skeletal muscle fibres. *J Physiol* 552: 499–511, 2003.
12. **Edman KA.** The velocity of unloaded shortening and its relation to sarcomere length and isometric force in vertebrate muscle fibres. *J Physiol* 291: 143–159, 1979.
13. **Fabiato A.** Computer programs for calculating total from specified free or free from specified total ionic concentrations in aqueous solutions containing multiple metals and ligands. *Methods Enzymol* 157: 378–417, 1988.
14. **Fauteck SP and Kandarian SC.** Sensitive detection of myosin heavy chain composition in skeletal muscle under different loading conditions. *Am J Physiol Cell Physiol* 268: C419–C424, 1995.
15. **Frontera WR, Hughes VA, Krivickas LS, Kim SK, Foldvari M, and Roubenoff R.** Strength training in older women: early and late changes in whole muscle and single cells. *Muscle Nerve* 28: 601–608, 2003.
16. **Frontera WR and Larsson L.** Contractile studies of single human skeletal muscle fibers: a comparison of different muscles, permeabilization procedures, and storage techniques. *Muscle Nerve* 20: 948–952, 1997.
17. **Frontera WR, Suh D, Krivickas LS, Hughes VA, Goldstein R, and Roubenoff R.** Skeletal muscle fiber quality in older men and women. *Am J Physiol Cell Physiol* 279: C611–C618, 2000.
18. **Galler S, Hilber K, Gohlsch B, and Pette D.** Two functionally distinct myosin heavy chain isoforms in slow skeletal muscle fibres. *FEBS Lett* 410: 150–152, 1997.
19. **Goldspink G.** Selective gene expression during adaptation of muscle in response to different physiological demands. *Comp Biochem Physiol B Biochem Mol Biol* 120: 5–15, 1998.
20. **Greaser ML, Moss RL, and Reiser PJ.** Variations in contractile properties of rabbit single muscle fibres in relation to troponin T isoforms and myosin light chains. *J Physiol* 406: 85–98, 1988.
21. **Grimby G, Danneskiold-Samsoe B, Hvid K, and Saltin B.** Morphology and enzymatic capacity in arm and leg muscles in 78–81 year old men and women. *Acta Physiol Scand* 115: 125–134, 1982.
22. **Häkkinen K, Alen M, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Mälkiä E, Kraemer WJ, and Newton RU.** Muscle CSA, force production, and activation of leg extensors during isometric and dynamic actions in middle-aged and elderly men and women. *JAPA* 8: 232–247, 1998.
23. **Häkkinen K, Kallinen M, Izquierdo M, Jokelainen K, Lassila H, Mälkiä E, Kraemer WJ, Newton RU, and Alen M.** Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. *J Appl Physiol* 84: 1341–1349, 1998.
24. **Häkkinen K, Kraemer WJ, Newton RU, and Alen M.** Changes in electromyographic activity, muscle fibre and force production characteristics during heavy resistance/power strength training in middle-aged and older men and women. *Acta Physiol Scand* 171: 51–62, 2001.
25. **Häkkinen K, Kraemer WJ, Pakarinen A, Triplett-McBride T, McBride JM, Häkkinen A, Alen M, McGuigan MR, Bronks R, and Newton RU.** Effects of heavy resistance/power training on maximal strength, muscle morphology, and hormonal response patterns in 60–75-year-old men and women. *Can J Appl Physiol* 27: 213–231, 2002.
26. **Häkkinen K, Newton RU, Gordon SE, McCormick M, Volek JS, Nindl BC, Gotshalk LA, Campbell WW, Evans WJ, Häkkinen A, Humphries BJ, and Kraemer WJ.** Changes in muscle morphology, electromyographic activity, and force production characteristics during progressive strength training in young and older men. *J Gerontol A Biol Sci Med Sci* 53: B415–B423, 1998.
27. **Häkkinen K, Pastinen UM, Karsikas R, and Linnamo V.** Neuromuscular performance in voluntary bilateral and unilateral contraction and during electrical stimulation in men at different ages. *Eur J Appl Physiol* 70: 518–527, 1995.
28. **Hameed M, Orrell RW, Cobbold M, Goldspink G, and Harridge SD.** Expression of IGF-I splice variants in young and old human skeletal muscle after high resistance exercise. *J Physiol* 547: 247–254, 2003.
29. **Harridge S, Magnusson G, and Saltin B.** Life-long endurance-trained elderly men have high aerobic power, but have similar muscle strength to non-active elderly men. *Aging (Milano)* 9: 80–87, 1997.
30. **Harridge SD, Bottinelli R, Canepari M, Pellegrino MA, Reggiani C, Esbjörnsson M, and Saltin B.** Whole-muscle and single-fibre contractile properties and myosin heavy chain isoforms in humans. *Pflügers Arch* 432: 913–920, 1996.
31. **Harridge SD, White MJ, Carrington CA, Goodman M, and Cummins P.** Electrically evoked torque-velocity characteristics and isomyosin composition of the triceps surae in young and elderly men. *Acta Physiol Scand* 154: 469–477, 1995.
32. **Henneman E and Mendell LM.** Functional organization of motoneuron pool and its inputs. In: *Handbook of Physiology. The Nervous System*. Bethesda, MD: Am. Physiol. Soc., 1981, sect. 1, vol. 2, pt. 1, chapt. 11, p. 423–507.
33. **Höök P, Li X, Sleep J, Hughes S, and Larsson L.** In vitro motility speed of slow myosin extracted from single soleus fibres from young and old rats. *J Physiol* 520: 463–471, 1999.
34. **Höök P, Sriramoju V, and Larsson L.** Effects of aging on actin sliding speed on myosin from single skeletal muscle cells of mice, rats, and humans. *Am J Physiol Cell Physiol* 280: C782–C788, 2001.
35. **Hughes SM, Cho M, Karsch-Mizrachi I, Travis M, Silberstein L, Leinwand LA, and Blau HM.** Three slow myosin heavy chains sequentially expressed in developing mammalian skeletal muscle. *Dev Biol* 158: 183–199, 1993.
36. **Klitgaard H, Mantoni M, Schiaffino S, Ausoni S, Gorza L, Laurent-Winter C, Schnohr P, and Saltin B.** Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. *Acta Physiol Scand* 140: 41–54, 1990.
37. **Korhonen MT, Mero A, and Suominen H.** Age-related differences in 100-m sprint performance in male and female master runners. *Med Sci Sports Exerc* 35: 1419–1428, 2003.
38. **Krivickas LS, Suh D, Wilkins J, Hughes VA, Roubenoff R, and Frontera WR.** Age- and gender-related differences in maximum shortening velocity of skeletal muscle fibers. *Am J Phys Med Rehabil* 80: 447–455, 2001.
39. **Kubo K, Kanehisa H, Azuma K, Ishizu M, Kuno SY, Okada M, and Fukunaga T.** Muscle architectural characteristics in women aged 20–79 years. *Med Sci Sports Exerc* 35: 39–44, 2003.
40. **Larsson L and Ansved T.** Effects of ageing on the motor unit. *Prog Neurobiol* 45: 397–458, 1995.
41. **Larsson L, Grimby G, and Karlsson J.** Muscle strength and speed of movement in relation to age and muscle morphology. *J Appl Physiol* 46: 451–456, 1979.
42. **Larsson L, Höök P, and Pircher P.** Regulation of human muscle contraction at the cellular and molecular levels. *Ital J Neurol Sci* 20: 413–422, 1999.
43. **Larsson L, Li X, and Frontera WR.** Effects of aging on shortening velocity and myosin isoform composition in single human skeletal muscle cells. *Am J Physiol Cell Physiol* 272: C638–C649, 1997.
44. **Larsson L and Moss RL.** Maximum velocity of shortening in relation to myosin isoform composition in single fibres from human skeletal muscles. *J Physiol* 472: 595–614, 1993.
45. **Lexell J, Taylor CC, and Sjöström M.** What is the cause of the ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15- to 83-year-old men. *J Neurol Sci* 84: 275–294, 1988.
46. **Li X and Larsson L.** Maximum shortening velocity and myosin isoforms in single muscle fibers from young and old rats. *Am J Physiol Cell Physiol* 270: C352–C360, 1996.
47. **Lionikas A, Li M, and Larsson L.** Human skeletal muscle myosin function at physiological and non-physiological temperatures. *Acta Physiol Scand* 186: 151–158, 2006.

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48. **Lowe DA, Surek JT, Thomas DD, and Thompson LV.** Electron paramagnetic resonance reveals age-related myosin structural changes in rat skeletal muscle fibers. *Am J Physiol Cell Physiol* 280: C540–C547, 2001.
49. **Metzger JM and Moss RL.** Calcium-sensitive cross-bridge transitions in mammalian fast and slow skeletal muscle fibers. *Science* 247: 1088–1090, 1990.
50. **Moisescu DG and Thieleczek R.** Calcium and strontium concentration changes within skinned muscle preparations following a change in the external bathing solution. *J Physiol* 275: 241–262, 1978.
51. **Moss RL.** Sarcomere length-tension relations of frog skinned muscle fibres during calcium activation at short lengths. *J Physiol* 292: 177–192, 1979.
52. **Oertel G.** Changes in human skeletal muscles due to ageing. Histological and histochemical observations on autopsy material. *Acta Neuropathol (Berl)* 69: 309–313, 1986.
53. **Pearson SJ, Young A, Macaluso A, Devito G, Nimmo MA, Cobbold M, and Harridge SD.** Muscle function in elite master weightlifters. *Med Sci Sports Exerc* 34: 1199–1206, 2002.
54. **Pereira Sant'Ana JA, Ennion S, Sargeant AJ, Moorman AF, and Goldspink G.** Comparison of the molecular, antigenic and ATPase determinants of fast myosin heavy chains in rat and human: a single-fibre study. *Pflügers Arch* 435: 151–163, 1997.
55. **Prochniewicz E, Thomas DD, and Thompson LV.** Age-related decline in actomyosin function. *J Gerontol A Biol Sci Med Sci* 60: 425–431, 2005.
56. **Proctor DN, Sinning WE, Walro JM, Sieck GC, and Lemon PW.** Oxidative capacity of human muscle fiber types: effects of age and training status. *J Appl Physiol* 78: 2033–2038, 1995.
57. **Ramamurthy B, Höök P, Jones AD, and Larsson L.** Changes in myosin structure and function in response to glycation. *FASEB J* 15: 2415–2422, 2001.
58. **Saltin B and Gollnick PD.** Skeletal muscle adaptability: significance for metabolism and performance. In: *Handbook of Physiology. Skeletal Muscle*. Bethesda, MD: Am. Physiol. Soc., 1983, sect. 10, chapt. 19, p. 555–631.
59. **Sato T, Akatsuka H, Kito K, Tokoro Y, Tauchi H, and Kato K.** Age changes in size and number of muscle fibers in human minor pectoral muscle. *Mech Ageing Dev* 28: 99–109, 1984.
60. **Short KR, Vittone JL, Bigelow ML, Proctor DN, Coenen-Schimke JM, Rys P, and Nair KS.** Changes in myosin heavy chain mRNA and protein expression in human skeletal muscle with age and endurance exercise training. *J Appl Physiol* 99: 95–102, 2005.
61. **Sipilä S, Koskinen SO, Taaffe DR, Takala TE, Cheng S, Rantanen T, Toivanen J, and Suominen H.** Determinants of lower-body muscle power in early postmenopausal women. *J Am Geriatr Soc* 52: 939–944, 2004.
62. **Sjöström M, Johansson C, and Lorentzon R.** Muscle pathomorphology in m. quadriceps of marathon runners. Early signs of strain disease or functional adaptation? *Acta Physiol Scand* 132: 537–541, 1988.
63. **Staron RS.** Correlation between myofibrillar ATPase activity and myosin heavy chain composition in single human muscle fibers. *Histochemistry* 96: 21–24, 1991.
64. **Staron RS, Hagerman FC, Hikida RS, Murray TF, Hostler DP, Crill MT, Ragg KE, and Toma K.** Fiber type composition of the vastus lateralis muscle of young men and women. *J Histochem Cytochem* 48: 623–629, 2000.
65. **Staron RS, Karapondo DL, Kraemer WJ, Fry AC, Gordon SE, Falkel JE, Hagerman FC, and Hikida RS.** Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. *J Appl Physiol* 76: 1247–1255, 1994.
66. **Syrovy I and Hodny Z.** Non-enzymatic glycosylation of myosin: effects of diabetes and ageing. *Gen Physiol Biophys* 11: 301–307, 1992.
67. **Thelen DG, Schultz AB, Alexander NB, and Ashton-Miller JA.** Effects of age on rapid ankle torque development. *J Gerontol A Biol Sci Med Sci* 51: M226–M232, 1996.
68. **Thompson LV and Brown M.** Age-related changes in contractile properties of single skeletal fibers from the soleus muscle. *J Appl Physiol* 86: 881–886, 1999.
69. **Tomonaga M.** Histochemical and ultrastructural changes in senile human skeletal muscle. *J Am Geriatr Soc* 25: 125–131, 1977.
70. **Trappe S, Gallagher P, Harber M, Carrithers J, Fluckey J, and Trappe T.** Single muscle fibre contractile properties in young and old men and women. *J Physiol* 552: 47–58, 2003.
71. **Widrick JJ, Trappe SW, Blaser CA, Costill DL, and Fitts RH.** Isometric force and maximal shortening velocity of single muscle fibers from elite master runners. *Am J Physiol Cell Physiol* 271: C666–C675, 1996.
72. **Viitasalo JT and Komi PV.** Force-time characteristics and fiber composition in human leg extensor muscles. *Eur J Appl Physiol* 40: 7–15, 1978.
73. **Viitasalo JT, Saukkonen S, and Komi PV.** Reproducibility of measurements of selected neuromuscular performance variables in man. *Electromyogr Clin Neurophysiol* 20: 487–501, 1980.

PAPER VI

EFFECTS OF COMBINED STRENGTH AND SPRINT TRAINING ON REGULATION OF MUSCLE CONTRACTION AT THE WHOLE-MUSCLE AND SINGLE-FIBRE LEVELS IN ELITE MASTER SPRINTERS

by

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Effects of combined strength and sprint training on regulation of muscle contraction at the whole-muscle and single-fibre levels in elite master sprinters

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Abstract

Aim: This study aims at examining the effects of progressive strength and sprint training on regulation of muscle contraction at the whole-muscle and single-fibre levels in older sprint-trained athletes.

Methods: Eleven men (52–78 years) were randomized to a training (EX, $n = 7$) or control (CTRL, $n = 4$) group. EX participated in a 20-week programme that combined sprint training with heavy and explosive strength exercises, while CTRL maintained their usual run-based training schedules.

Results: EX improved maximal isometric and dynamic leg strength, explosive jump performance and force production in running. Specific tension and maximum shortening velocity of single fibres from the vastus lateralis were not altered in EX or CTRL. Fibre type and myosin heavy chain isoform distributions remained unchanged in the two groups. There was a general increase in fibre areas in EX, but this was significant only in IIa fibres. The 10% increase in squat jump in EX was accompanied by a 9% increase in the integrated EMG (iEMG) of the leg extensors but the 21–40% increases in isometric and dynamic strength were not paralleled by changes in iEMG.

Conclusion: Adding strength training stimulus to the training programme improved maximal, explosive and sport-specific force production in elite master sprinters. These improvements were primarily related to hypertrophic muscular adaptations.

Keywords ageing, fibre types, hypertrophy, master athletes, neural activity, skinned fibres.

It has been established that ageing-related impairments in maximal and explosive muscle strength can be partially reversed by progressive resistance training. The exercise-induced improvement of muscle function can be explained by muscular or neural mechanisms, or a combination of both. Adaptations to exercise in older subjects have been reported at different levels, such as (1) hypertrophic muscle adaptation with an increase in the size of type I and type II fibres (Larsson 1982, Frontera *et al.* 1988, Hakkinen *et al.* 2002), (2) altered

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regulation of muscle contraction at the cellular level with effects on shortening velocity (Trappe *et al.* 2000), maximum force (Trappe *et al.* 2000, Frontera *et al.* 2003), maximum force normalized to cross-sectional area (CSA; specific force) (Frontera *et al.* 2003) and power (Trappe *et al.* 2000), or (3) a dominant impact of neural adaptations indicated by a large increase in electromyographic activity of the agonist muscles with relatively small hypertrophic changes in muscle volume in response to strength training (Moritani & deVries 1980, Hakkinen *et al.* 1998).

The physical activity level of the subject determines the nature and magnitude of strength training-induced adaptations in the neuromuscular system. Research into the impact of strength training in old age has focused on subjects with a low physical activity level. To our knowledge, no attempts have been made to investigate the effects of strength training in older athletes who have a previous training background. Physically active master athletes represent a valuable model for training studies as the physiological adaptations in this group should reflect the impact of training *per se* rather than the unspecific effect in response to increased physical activity that may apply to previously untrained subjects. Further insight could be obtained into the adaptive capacity of the ageing neuromuscular system by studying the influence of strength training on muscular and neural characteristics among older athletes. Moreover, given the importance of muscle strength and power for many athletic movements, it is of interest to examine whether strength training can result in increased sports performance in older athletes.

The purpose of this study, therefore, was to carry out a comprehensive examination of whole-muscle, single-fibre and neural adaptations to simultaneous strength and sprint training in highly competitive master sprinters.

Materials and methods

Subjects

The study was part of a larger investigation of the effects of age and long-term sprint training on muscle structure and function (Korhonen *et al.* 2006). Of the seventy-two 40- to 84-year-old male sprint athletes participating in the baseline measurements and subsequently randomized into an experimental (EX, $n = 40$) and a control (CTRL, $n = 32$) group, a subset of twelve 52- to 78-year-old elite sprinters with no background of intensive strength training were chosen for the present study, which involved single-fibre analysis of muscle biopsy samples. Of these subjects, seven in the EX group and four in the CTRL group completed the study. The subjects had achieved world-class results in 100–400 m sprint events and were all continuing to participate in major national and/or international competitions. The running times of the 60-m sprint trial performed at the beginning of this study (see the test description below) were on average 104 ± 2% of the age-based world record times. Selected physical and training characteristics of the subjects, measured as described previously (Korhonen *et al.* 2006), are presented in Table 1. All subjects had been sprint training for several years. During the preceding year, the subjects reported very similar training programmes. These consisted mainly of speed, speed endurance and plyometric exercises. In addition, the amount of resistance training (strength endurance type) was approx. 1 h per week in both groups.

Table 1 Physical characteristics of subjects

	Experimental group ($n = 7$)	Control group ($n = 4$)
Age (years)	66 ± 3	71 ± 5
Training background (years)	32 ± 7	24 ± 4
Training (h year^{-1})	355 ± 46	310 ± 45
Strength training (h week^{-1})	0.9 ± 0.4	1.0 ± 0.3
Height (cm)	173 ± 2	171 ± 2
Weight (kg)		
Before	71.3 ± 2.5	69.6 ± 3.6
After	71.5 ± 2.3	70.9 ± 3.1
Body fat (%)		
Before	15.4 ± 1.4	17.5 ± 1.7
After	15.9 ± 0.8	17.5 ± 1.9

Values are mean ± SE.

The subjects were healthy and free of cardiovascular diseases as assessed by their medical histories and by a focused medical examination based on resting electrocardiograms and blood pressure measurements. Subjects reported that they were not taking any medication or nutritional supplements that could influence physical performance or skeletal muscle. There were no significant between-group differences in the selected subject characteristics either before or after training (Table 1). A written consent was obtained from all the participating subjects, after they had been fully informed of the procedures, potential risks and benefits associated with taking part in the study. The study was approved by the Ethics Committee of the University of Jyväskylä and conformed to the principles of the Declaration of Helsinki.

Experimental protocol

The 20-week experimental period took place during the indoor season from late December to early May. The main laboratory measurements of neuromuscular characteristics were performed before and after the exper-

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imental period and lasted 2 days. The first day included tests of the subject's anthropometry, maximal running and strength performance, and measures of muscle activation during isometric and dynamic contractions with surface electromyography. On the second day, fasting blood samples for the determination of serum hormones and muscle biopsies were taken from each subject. To obtain feedback on the athlete's training status and degree of progress during the intense training period, the study included field tests (weeks 5, 10, 15) for running performance and muscle power (data not shown).

Periodized training programme

The combined strength and sprint training programme was designed by researchers and coaches in collaboration and utilized knowledge obtained from earlier studies in young adult athletes (Joch 1992, Delecluse 1997, Kraemer & Häkkinen 2002). In order to reduce the potential for overtraining and to optimize the adaptation, attention was paid to the proper periodization of training. The planned programme and relative volumes of the different modes of training during the course of the study are summarized in Figure 1 and described in the Appendix.

Subjects completed training logs describing all their training parameters (number of repetitions, sets, loads, distances, times of exercises) to monitor progress and to provide motivation for maximal effort during the study. The logs were collected every fifth week during the field testing sessions. The overall training adherence rate in EX, calculated as the percentage of training sessions successfully completed, was 86 ± 4% for strength training and 83 ± 6% for sprint training across the 20-week study period. In EX, the average training hours and frequency over the training period were 2.1 ± 0.2 h and 1.6 ± 0.1 times per week for strength training and 2.1 ± 0.3 h and 1.6 ± 0.1 times per week for sprint training. Other exercises (ball games, aerobic running, skiing) were performed 0.5 ± 0.2 h and 0.6 ± 0.2 times per week. The controls maintained their previous run-based training schedules.

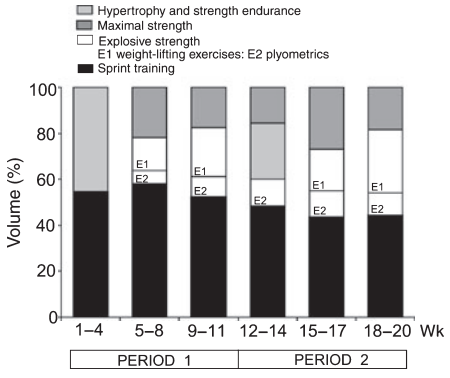

Figure 1 The planned training programme and relative volumes of different training modes during the 20-week study. The training programme consisted of two 11- and 9-week periods that were further divided into three 3- to 4-week phases with variations in training intensity, volume and type. Wk, weeks.

Measurements

Muscle strength. Isometric torque of the knee extensors and flexors was measured on the dominant leg using a David 200 dynamometer (David Fitness and Medical Ltd, Outokumpu, Finland) (Hakkinen *et al.* 1998). The subject was in a sitting position with knee and hip angles of 90° and 110°, respectively, and on a verbal command exerted maximal force for a period of ~4.0 s. Three to four trials were recorded until there was no further improvement in peak torque. Concentric force of the leg extensors was measured with the one-repetition maximum method (1-RM) using a half squat exercise in the Smith machine (Hakkinen *et al.* 2002). The test involved the subject bending the knees to 90° with a loaded bar on the shoulders (controlled with an auditory signal), maintaining the position for ~1 s, and then extending up on a command. The highest load lifted was determined as the subject's 1-RM. Two subjects in the EX group and one control subject declined to perform the squat test because of fear of possible injury.

Explosive strength capability was assessed by various types of jumping tests. The squat jump required performing a vertical jump on the force platform from a static squat with a knee angle of ~90° (Asmussen & Bonde-Petersen 1974). Three to five trials were recorded and the highest vertical displacement value evaluated from the flight time was used in the analyses. For the triple jump test, carried out on a long jump track, the subjects began by standing on a plate (height 5 cm) with toes over the edge (Mero *et al.* 1981). Using arm swings at the start the subjects performed three successive maximal jumps with alternative left- and right-leg contacts, landing on two legs on the sand after the final jump. The reactive jump test was performed two to three times and involved a series of vertical jumps for ~5 s on a contact mat keeping the legs as extended as possible to emphasize the use of the ankle plantar flexors (Bret *et al.* 2002). The subjects were instructed to jump as high as possible while minimizing the contact times. The contact and flight times of each jump were measured to determine mechanical power per kg body weight (Bosco *et al.* 1983), and the mean of the two best consecutive jumps was included in the analysis.

Force production of running. The subjects ran two maximal 60 m sprints from a standing start on an indoor synthetic track with spiked running shoes. Vertical and horizontal ground reaction forces, contact times and stride rates were measured during the maximal speed phase (from 30 m) using a special 9.4-m long force platform system (Paavolainen *et al.* 1999). The force platform signals were sampled at 1000 Hz and stored on a microcomputer via an AT Codas A/D converter card (Dataq Instruments, Akron, OH, USA). The 10-m running velocity over the force platform and the 60-m trial times were obtained by using double-beam photocell gates (starting line 0.7 m behind the first photocell gates). The average stride length was calculated by dividing the 10-m running velocity by the stride rate.

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The ground reaction force variables were analysed by custom-built software (University of Jyväskylä). The transition point from a negative to positive value in the horizontal force–time curve was used to divide the contact time and vertical and horizontal force components into the braking and propulsion phases (Mero & Komi 1986). The vertical and horizontal forces were integrated with respect to time phases and then combined to obtain the average resultant force separately for the braking and propulsive phases. The amplitudes of the average resultant forces were normalized to body weight ($N\ kg^{-1}$). The average rate of force development (RFD) for the braking and propulsive phases was calculated by dividing the average resultant forces by the respective contact times. The first four contacts of the fastest trial were averaged and used in the final analysis.

EMG activity. EMG activity during isometric knee extension, dynamic squat 1-RM and squat jump tests was recoded from the vastus lateralis (VL), vastus medialis (VM) and biceps femoris (BF) of the dominant leg using previously described procedures (Hakkinen *et al.* 1998) with slight modifications. Briefly, bipolar surface electrodes (Beckman miniature skin electrodes; 2 cm interelectrode distance; Beckman Instruments, IL, USA) were attached longitudinally to the belly of the muscles on the motor point areas (Hermens *et al.* 1999). The positions of the electrodes were marked with small ink dots to ensure consistency in electrode placement in the pre- and post-measurements. The integrated EMG activity (iEMG, in $\mu V\ s^{-1}$) was analysed for the peak force phase of the isometric knee extension (over a period of 1 s around the peak torque) and for the whole concentric phases of the dynamic half squat 1-RM and squat jump exercises. The iEMG was calculated for VL and VM separately and then averaged for the final analyses. The iEMGs of the VL and VM during dynamic actions were also expressed as a percentage of their maximum isometric activity during knee extension to evaluate neural adaptation (dynamic vs. isometric) without comparison of absolute EMG values. The iEMG of the BF from the knee flexion exercise was analysed as described for VL and VM in the isometric knee extension. To evaluate the degree of antagonist BF coactivation during isometric knee extension, squat 1-RM and squat jump exercises, the iEMG of the BF was expressed relative to that measured in maximal isometric knee flexion contraction. The BF iEMG data had to be excluded from two subjects in the EX group because of distorted EMG signals.

Muscle biopsy. Details of the experimental procedure have been described earlier (Korhonen *et al.* 2006). Briefly, muscle samples were taken from the middle portion of the VL of the dominant leg using a needle biopsy technique (Bergstrom 1962) with suction. 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 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 myosin heavy chain (MyHC) analyses. The third part was split in several small bundles of ∼25–50 fibres that were chemically permeabilized and saved for the single-fibre analysis (Frontera & Larsson 1997, Korhonen *et al.* 2006).

Homogenate gel electrophoresis. The MyHC isoform content of the biopsy samples was determined by sodium dodecyl sulphate-polyacrylamide gel electrophoresis (SDS-PAGE) according to previously described methods (Andersen & Aagaard 2000) with slight modifications (Korhonen *et al.* 2006). It has been shown previously that the slowest migration protein band in humans is analogous to the MyHC IIx, not the MyHC IIb, isoform in rats (Pereira Sant'Ana *et al.* 1997) and therefore the MyHC IIx nomenclature is used in this study.

Myofibrillar ATPase histochemistry. The myofibrillar ATPase histochemical analysis was performed as described in detail earlier (Korhonen *et al.* 2006). Relative fibre type distribution was calculated from an average of 486 ± 85 and 357 ± 87 fibres per biopsy in the pre- and post-measurements respectively. The determination of fibre CSA comprised an average of 216 ± 46 and 146 ± 29 type I, 161 ± 33 and 112 ± 27 type IIA, 57 ± 12 and 31 ± 7 type IIAB, and 52 ± 15 and 68 ± 37 type IIB fibres per biopsy in the pre- and post-measurements respectively.

Single-fibre contractile measurements. The experimental procedure has been described in detail elsewhere

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(Larsson & Moss 1993). The skinned muscle fibre used in this work had an average segment length of 2.02 ± 0.51 mm (mean ± SD, range 1.05–3.60 mm) exposed to the solution between the connectors of the force transducer. The sarcomere length (SL) of the single-fibre segment was set to 2.79 ± 0.01 μm (range 2.72–2.84 μm) by adjusting the overall segment length. Fibre SL was measured routinely in the fibres during maximal activation. Fibre depth was measured by recording the vertical displacement of the microscope nosepiece while focusing on the top and bottom surfaces of the fibre. Fibre CSA was calculated from the width and depth, assuming an elliptical circumference. Every width and depth value represented the average of three different measurements. The accuracy of the measurements carried out by the same observer was verified by comparing 200 average values for depth calculated at two different SL ($r^2 = 0.99$). Specific tension (ST) was calculated as maximum tension (P_o) normalized to CSA, and was corrected for the 20% swelling that is known to occur during skinning (Moss 1979).

Relaxing and activating solutions were prepared as previously described (Larsson & Moss 1993), and the apparent stability constants for Ca^{2+} -EGTA were corrected for temperature and ionic strength (Fabiato 1988).

Maximum unloaded shortening velocity (V_o) was measured by the slack-test procedure (Edman 1979). Fibres 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 fibre. 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 fibre was re-extended while relaxing in order to minimize non-uniformity of SL. A straight line was fitted to a plot of ΔL vs. Δt, using a least-squares regression, and the slope of the line normalized to muscle fibre length was recorded as V_o for that fibre. Maximum active tension (P_o) 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 V_o value was based on linear regressions including four or more data points, and data were discarded if r for the fitted line was less than 0.97, if P_o changed more than 10% from first to final activation, or if SL during isometric tension development changed by more than 0.10 μm compared with SL when the fibre was relaxed (Moss 1979).

Single-fibre gel electrophoresis. After mechanical measurements, each fibre was placed in SDS sample buffer in a plastic microfuge tube and stored at −20 °C for up to 1 week or at −80 °C if the gels were run later. The MyHC composition was determined by SDS-PAGE. The total acrylamide and bis concentrations were 4% (w/v) in the stacking gel and 6% in the running gel, and the gel matrix included 30% glycerol. The ammonium per sulphate concentrations were 0.04% and 0.029% in the stacking and separation gels, respectively, and the gel solutions were degassed (<100 mtorr) for 15 min at 18 °C. Polymerization was subsequently activated by adding TEMED to the stacking (0.1%) and separation gels (0.07%). Sample loads were kept small to improve the resolution of the MyHC bands and electrophoresis was performed at 120 V for 22–24 h with a Tris-glycine electrode buffer (pH 8.3) at 10 °C (SE 600 vertical slab gel unit; Hoefer Scientific Instruments, San Francisco, CA, USA) (for details, see Larsson & Moss 1993).

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Statistical analyses

Mean values and standard errors were calculated from individual values by using standard procedures. Linear regression analysis was used to assess the reliability of CSA depth measurements on single fibres. Because the data were not normally distributed, the Wilcoxon signed-ranks test was used to assess differences in single-fibre CSA, P_o/CSA and V_o, histochemical fibre characteristics, MyHC, muscle performance and iEMG before and after training within the study groups. The Mann–Whitney test was used to test for differences in changes between EX and CTRL. Differences were considered significant at $P < 0.05$ for all analyses.

Results

Muscle strength

Maximum isometric and dynamic strength before and after the 20-week period are shown in Figure 2. In EX, the maximal isometric torque during unilateral knee extension and knee flexion exercises increased by 21% ($P < 0.05$) and 40% ($P < 0.01$) respectively. Maximum dynamic strength, as evaluated by bilateral concentric 1-RM squat, increased by 27% ($P < 0.001$, $n = 5$). Significant increases were also observed in the squat jump (10%, $P < 0.01$), triple jump (4%, $P < 0.01$) and the mechanical power of reactive jump test (29%, $P < 0.01$). In CTRL, no significant changes were found in any of these parameters. When comparing percentage changes over the intervention period, all jump test variables were found to be increased significantly more for the EX group than for the CTRL group ($P < 0.05$–0.01).

Force production during running

The selected ground reaction force characteristics of maximal running are presented in Figure 3. In EX, the

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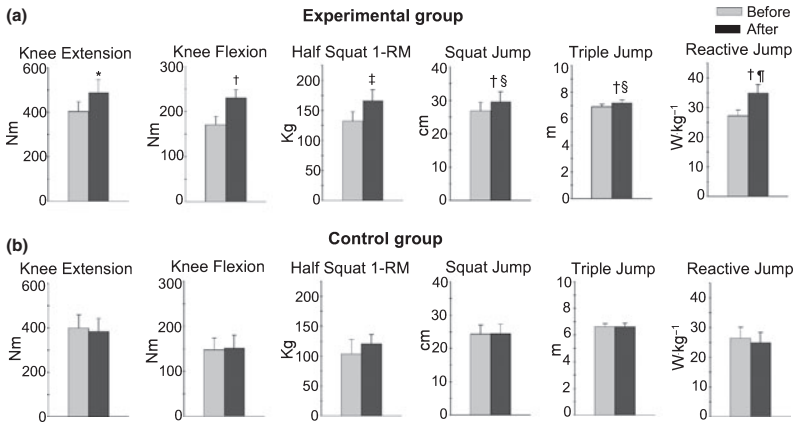

Figure 2 Isometric and dynamic force parameters in the experimental (a) and control (b) groups. Values are mean ± SE. $^{*}P < 0.05$, $^{\dagger}P < 0.01$, $^{\ddagger}P < 0.001$ for the comparison with the corresponding value before training; $^{\S}P < 0.05$, $^{\P}P < 0.01$ for the comparison of the percentage change from before training with that in the control group.

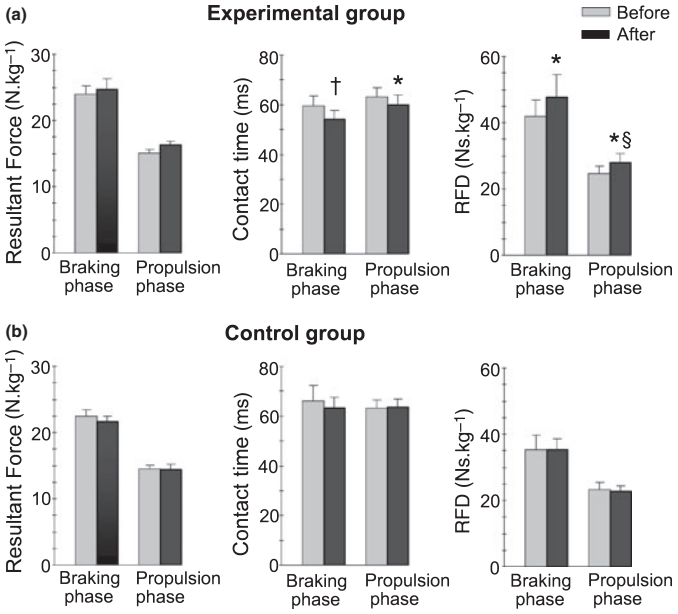

Figure 3 The selected ground reaction force characteristics of maximal running in the experimental (a) and control (b) groups. Values are mean ± SE. $^{*}P < 0.05$, $^{\dagger}P < 0.01$ for the comparison with the corresponding value before training; $^{\S}P < 0.05$ for the comparison of the percentage change from before training with that in the control group.

average resultant force of the braking phase of contact did not change (2%, $P = 0.18$), but an 8% increase ($P < 0.05$) was observed in the propulsive phase of the contact. The ground contact times of the braking and propulsive phases decreased by 9% ($P < 0.01$) and 5% ($P < 0.05$) respectively. As a consequence of these changes there was a significant increase in the rate of resultant force development (RFD) in both the braking

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(12%, $P < 0.05$) and propulsive (14%, $P < 0.05$) phases. In addition, maximum 10-m running speed improved from 7.47 ± 0.28 to 7.74 ± 0.31 m s^{-1} (4%, $P < 0.01$) and 60-m sprint time from 8.69 ± 0.30 to 8.52 ± 0.29 s (2%, $P < 0.01$). Stride length of the maximum speed phase increased from 1.79 ± 0.06 to 1.85 ± 0.08 m (3%, $P < 0.05$), but no significant change occurred in stride rate (from 4.19 ± 0.10 to 4.22 ± 0.13 Hz). In CTRL, no significant changes were observed either in the ground reaction force characteristics (Fig. 3b) or in any of the other studied parameters of running. When comparing percentage changes over the intervention period, the propulsive phase RFD ($P < 0.05$), stride length ($P < 0.05$), maximum 10-m speed ($P < 0.05$) and overall 60-m sprint time ($P < 0.01$) differed significantly between the two groups.

EMG activity

The absolute and normalized iEMG activity levels before and after the 20-week period are shown in Figure 4. In EX, a 9% increase ($P < 0.05$) was observed in the iEMG of the VL and VM muscles in the squat jump, but no significant changes were observed in the maximum iEMGs of the VL and VM in the concentric half squat 1-RM or during isometric knee extension. The maximum iEMGs of the BF during the isometric knee flexion remained unaltered. The percentage increase in the squat jump iEMG by the EX group was significantly greater than the percentage change by the CTRL ($P < 0.05$).

Statistically nonsignificant increases were observed when the iEMGs of the VL and VM obtained in the dynamic squat 1-RM and squat jump actions were normalized to their maximum isometric activity during knee extension. In addition, no change was observed in the magnitude of antagonist BF iEMG coactivity (relative to maximum agonist values of the BF) during the isometric knee extension (28 ± 8% before; 25 ± 5% after), squat 1-RM (48 ± 15% before; 47 ± 10% after) or squat jump (55 ± 8% before; 53 ± 9% after) exercises. In CTRL, no significant alterations were observed in any of the muscle activity parameters.

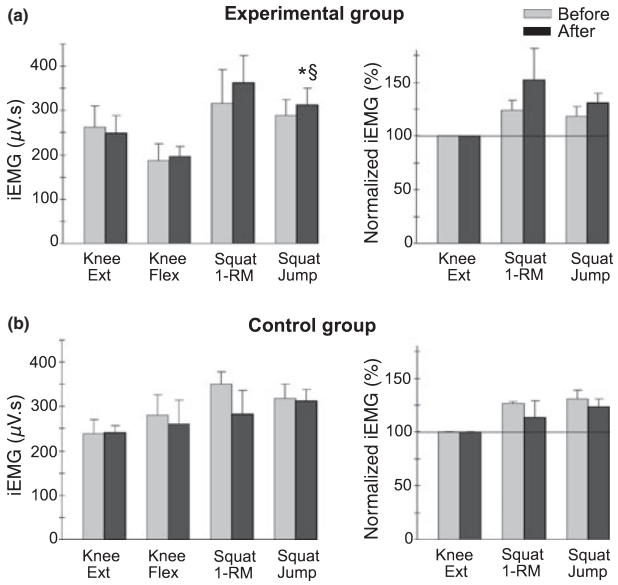

Figure 4 Absolute and normalized maximum integrated EMG activity (iEMG) in the experimental (a) and control (b) groups. iEMG was averaged for the vastus lateralis and vastus medialis (VL + VM)/2 in unilateral isometric knee extension (Knee Ext), bilateral one-repetition maximum half squat (Squat 1-RM) and squat jump. The unilateral isometric knee flexion (Knee Flex) value represents the iEMG activity from the biceps femoris. In the normalized iEMGs the activity of the VL and VM during dynamic actions is expressed as a percentage of their maximum isometric activity during knee extension. Values are mean ± SE. *$P < 0.05$ for the comparison with the corresponding value before training; §$P < 0.05$ for the comparison of the percentage change from before training with that in the control group.

Table 2 Histochemical fibre type distribution, fibre cross-sectional areas (CSA) and myosin heavy chain (MyHC) isoform composition from the vastus lateralis muscle before and after the 20-week training period

	Experimental group		Control group	
	Before	After	Before	After
Fibre type (%)				
I	43.3 ± 2.5	45.6 ± 6.3	39.9 ± 7.7	43.4 ± 8.0
IIA	34.8 ± 3.4	34.6 ± 3.3	28.3 ± 7.3	28.2 ± 5.4
IIAB	11.2 ± 2.2	7.7 ± 2.3	15.1 ± 3.1	9.2 ± 1.1
IIB	10.7 ± 2.9	12.1 ± 5.3	16.7 ± 6.2	19.2 ± 6.4
CSA (10^2 μm^2)				
all	45.1 ± 3.5	54.2 ± 6.0*	46.8 ± 3.5	40.1 ± 2.6
I	49.2 ± 3.9	58.0 ± 7.5	45.8 ± 6.1	38.6 ± 1.5
II	42.2 ± 3.3	49.3 ± 4.4*§	47.0 ± 3.1	41.6 ± 4.8
IIA	44.0 ± 3.5	52.8 ± 5.0*§	53.2 ± 6.2	45.5 ± 4.1
IIAB	35.9 ± 2.6	41.6 ± 5.0	45.7 ± 4.9	43.2 ± 6.0
IIB	40.8 ± 4.5	45.8 ± 4.6	39.7 ± 1.7	36.1 ± 5.1
MyHC (%)				
I	46.2 ± 4.1	48.3 ± 6.6	36.8 ± 5.4	31.6 ± 5.7
IIa	47.9 ± 4.0	42.9 ± 4.4	51.2 ± 6.3	55.2 ± 3.3
IIx	6.0 ± 2.5	8.8 ± 3.6	12.0 ± 5.4	13.3 ± 5.2

Values are mean ± SE.
*$P < 0.05$ for the comparison with the corresponding value before training.
§$P < 0.05$ for the comparison of the percentage change from before training with that in the CTRL group.

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Myofibrillar ATPase histochemistry

The relative distribution and the CSAs of the histochemically identified fibre types are shown in Table 2. The fibre type distribution remained unaltered during the training period in EX, but the mean CSA of all fibres in EX increased by 20% ($P < 0.05$). In enzyme-histochemically classified fibre types, a statistically significant change was only observed in type II and type IIA fibres, demonstrating an increase of 17 and 20%, respectively ($P < 0.05$). Larger CSAs were also observed in type I (19%), IIAB (17%) and type IIB (33%) fibres in EX, but these changes were not statistically significant ($P = 0.3$–0.1). In CTRL, no significant changes were observed in either muscle fibre type distribution or CSAs ($P = 0.6$–0.3). The percentage changes over the experimental period in the CSA of type II and type IIA fibres were significantly ($P < 0.05$) different between EX and CTRL. The type II-to-type I fibre area ratio remained unchanged at the end of the training period in both the EX (0.87 ± 0.05 before; 0.88 ± 0.06 after) and CTRL groups (1.09 ± 0.18 before; 1.09 ± 0.16 after).

MyHC isoform expression

The relative content of type I, IIa and IIx MyHC isoforms in the muscle homogenates of the biopsies did not change over the 20-week period in either EX or CTRL (Table 2). The IIx MyHC isoform was not detected in pre- or post-biopsies in three subjects in the EX and in one subject in the CTRL group.

Single-fibre properties

A total of 219 single membrane permeabilized muscle fibre segments out of 470 fibres fulfilled the criteria for acceptance for contractile measurements. No significant differences in the relative number of accepted single-fibre preparations were observed between EX (48%) and CTRL (44%).

Muscle fibre CSA was measured in all 470 fibre segments in EX ($n = 312$) and CTRL ($n = 158$) at a fixed SL assuming an elliptical cross section of the fibre segment. In EX, the average CSA of all fibres was 25% higher after (3660 ± 348 μm^2, $n = 154$) than before (2940 ± 130 μm^2, $n = 158$) the 20-week training programme, but this difference was not statistically significant ($P = 0.075$). In CTRL, average CSA was 3330 ± 373 μm^2 ($n = 74$) and 3410 ± 399 μm^2 ($n = 84$) before and after the experimental period respectively. Fibre type-specific analyses on CSA were restricted to fibres expressing the type I and IIa MyHC isoform; the low number of type IIx MyHC fibres ($n = 11$) and fibres co-expressing the type I and IIa MyHC isoforms ($n = 20$) did not permit statistical analyses. A total of 61 fibres co-expressed type IIa and IIx MyHCs, but these fibres were only observed in two EX subjects both before and after the training programme. In EX, a 40% increase ($P < 0.05$) in CSA was

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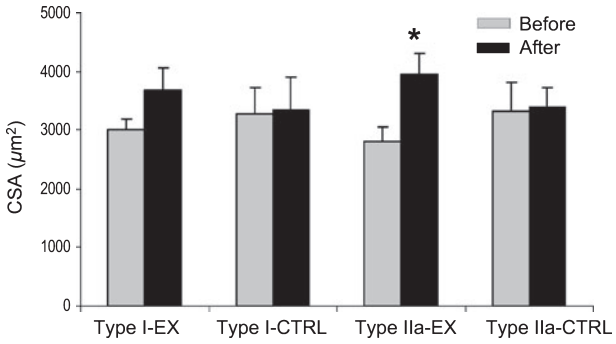

Figure 5 Single fibre cross-sectional area (CSA) of the vastus lateralis muscle. The CSA values within the EX and CTRL groups before and after the 20-week period. *$P < 0.05$ for the comparison of the change within the group.

observed in muscle cells expressing the type IIa MyHC isoform (Figure 5, Table 3). The 22% larger CSA of type I MyHC fibres after training was not statistically significant (Table 3). In CTRL, CSAs were unchanged in the type I and IIa MyHC fibres. Similarly, the difference in change in type IIa fibres between EX and CTRL did not reach statistical significance ($P = 0.097$). The type II-to-type I area ratio of single fibres did not differ significantly in response to the 20-week period in either EX (0.96 ± 0.07 before; 1.14 ± 0.17 after) or CTRL (1.04 ± 0.09 before; 1.06 ± 0.13 after).

In EX, the 27 and 58% increases in maximum force (P_o) in cells expressing the type I (630 ± 49 μN, $n = 45$ vs. 800 ± 155 μN, $n = 45$) and IIa (640 ± 137 μN, $n = 21$ vs. 1010 ± 187 μN, $n = 28$) MyHC isoforms, respectively, did not reach statistical significance. The ST, i.e. maximum force normalized to fibre CSA, in single type I and IIa MyHC fibres did not change in EX or CTRL (Table 3). In CTRL, P_o in fibres expressing the type I (630 ± 31 μN, $n = 15$ vs. 620 ± 122 μN, $n = 12$) and type IIa (740 ± 77 μN, $n = 19$ vs. 720 ± 103 μN, $n = 15$) MyHC isoforms was not affected during the 20-week period. The maximum unloaded shortening velocity (V_o) of type I and IIa MyHC fibres remained unaffected at the end of the training period in both groups (Table 3).

Discussion

In this study we examined the effects of a 20-week combined strength and sprint training programme on muscular and neural characteristics in male master sprinters. The main findings were as follows: (1) the EX group showed significant increases in maximal isometric and dynamic leg strength, explosive jump performances and force production in running, (2) in EX significant hypertrophy occurred in type II and IIa fibres according to the histochemical and single-fibre data analyses, (3) the type II-to-type I fibre area ratio was not influenced by the training programme, (4) the maximum velocity of unloaded shortening and the force generation capacity (ST) in single muscle fibres expressing the type I and IIa MyHC isoforms were not affected by the training period, (5) the relative content of MyHC isoforms and proportions of fibre types remained unchanged during the training programme, (6) in EX a 9% increase in the iEMG of agonist VL and VM accompanied the 10% increase in the squat jump, but no significant changes were observed in iEMG, despite increases of 21–40% in maximal isometric and dynamic leg strength.

Whole-muscle performance

Resistance training is an effective method of improving muscle strength qualities in previously untrained subjects. The present study showed that incorporating weight training exercises into the overall training led to improvements in maximal and explosive strength in world-class master sprinters (Fig. 2). Part of the explanation for the substantial improvements in muscular strength may be due to the specific form of strength training used, in which heavy resistance exercises were combined with explosive types of weight training and plyometric exercises. This is in accordance with previous studies demonstrating that the combination of heavy and explosive weight training method is an efficient strategy to improve strength performance in both young (Harris *et al.* 2000, Newton *et al.* 2002) and older (Hakkinen *et al.* 1998, Izquierdo *et al.* 2001, Newton *et al.* 2002) subjects. However, the large strength gains may also reflect successful periodization of training to maintain overall training loading within the normal physiological range.

Although many studies have shown that strength training can improve sprinting speed in young adult athletes (Cadefau *et al.* 1990, Andersen *et al.* 1994, Delecluse *et al.* 1995, Harris *et al.* 2000), to our knowledge, no attempts have been made to investigate the manner in which strength gains are transferred to force production in running. Our results showed that

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Table 3 Cross-sectional area (CSA), specific tension (ST), and maximum velocity of unloaded shortening (V_o) in skinned single muscle fibres expressing different types of MyHC isoforms in the experimental (EX) and control (CTRL) group before and after the 20-week training period

	Type I			Type I/IIa			Type IIa			Type IIax			Type IIx		
	CSA (μm^2)	ST (N cm^{-2})	V_o (ML s^{-1})	CSA (μm^2)	ST (N cm^{-2})	V_o (ML s^{-1})	CSA (μm^2)	ST (N cm^{-2})	V_o (ML s^{-1})	CSA (μm^2)	ST (N cm^{-2})	V_o (ML s^{-1})	CSA (μm^2)	ST (N cm^{-2})	V_o (ML s^{-1})
EX before	3000 ± 190 ($n = 90$)	30.86 ± 3.44	0.50 ± 0.06 ($n = 45$)	2790 ± 270 ($n = 7$)			2810 ± 240 ($n = 44$)	33.88 ± 5.23	1.74 ± 0.19 ($n = 21$)	2850 ± 330 ($n = 14$)	34.49 ± 2.93	2.72 ± 0.47 ($n = 4$)	2610 ± 60 ($n = 3$)	18.65 25.73	1.50 3.82
EX after	3670 ± 390 ($n = 85$)	30.19 ± 3.32	0.61 ± 0.05 ($n = 45$)	3490 ± 780 ($n = 6$)	44.80 ± 7.32	0.92 ± 0.20 ($n = 3$)	3950 ± 360* ($n = 47$)	37.96 ± 3.98	1.83 ± 0.26 ($n = 28$)	3170 ± 730 ($n = 16$)	15.5	2.05 ($n = 1$)	1860 4510		
CTRL before	3280 ± 440 ($n = 25$)	32.63 ± 1.23	0.57 ± 0.10 ($n = 15$)				3320 ± 500 ($n = 31$)	33.03 ± 5.86	1.58 ± 0.14 ($n = 19$)	3634 ± 460 ($n = 6$)			2790 ± 415 ($n = 4$)	20.32 28.13	2.34 3.68
CTRL after	3350 ± 560 ($n = 28$)	30.31 ± 6.64	0.57 ± 0.16 ($n = 12$)	2980 ± 570 ($n = 7$)	32.95 ± 5.40	0.92 ± 0.20 ($n = 3$)	3380 ± 340 ($n = 31$)	32.01 ± 6.61	1.66 ± 0.33 ($n = 15$)	4140 ± 610 ($n = 25$)	28.07 ± 4.20	2.59 ± 0.86 ($n = 4$)	4360 4370		

Values are mean ± SE. n, number of fibres tested
(for ST number of fibres same as for V_o); ML, muscle length.
*$P < 0.05$ for the comparison with the corresponding value before training.

older athletes were able to produce higher ground forces with shorter contact times in response to the training programme, leading to an increase in the RFD (Fig. 3). It is possible that the gain in muscular strength improved the stretch-shortening cycle capacity in running by allowing the leg extensor muscles to withstand greater impact loads and enhancing the force performance potentiation in the propulsion phase of contact.

Muscular adaptations

The quantitative loss in muscle CSA with ageing is a major contributor to the decrease in muscle strength seen with advancing age in sedentary people (Frontera *et al.* 2000). Resistance training protocols have proved efficient in stimulating muscle fibre hypertrophy, with subsequent increases in strength and myofibrillar protein turnover rate (Frontera *et al.* 1988). At the single-fibre level, significant increases have been reported in CSA and P_o in both young (Widrick *et al.* 2002, Shoepe *et al.* 2003) and older (Trappe *et al.* 2000) subjects. However, the hypertrophic effects of resistance training vary between studies, and it can be speculated that the mode, intensity and duration of the training, as well as differences in the age, sex and habitual activity level of the subjects may be factors that decide the size of the hypertrophic effects of training (Hakkinen *et al.* 1985, Frontera *et al.* 1988, Trappe *et al.* 2000, Aagaard *et al.* 2001, Widrick *et al.* 2002, Shoepe *et al.* 2003). The 20-week training programme, which included sprint and heavy resistance and explosive strength training exercises, was accompanied by muscle fibre hypertrophy, with significant increases in the CSA of type II and IIa fibres. These findings support the effectiveness of this specific training regimen and also point out the important contribution of hypertrophic factors to maximal and explosive strength development in physically very active older men.

An ageing-related preferential decrease in fast-twitch fibre CSA, resulting in a decreased type II-to-type I fibre area ratio, has been repeatedly documented in human skeletal muscle from sedentary men since Tomonaga's pioneering study 30 years ago (Tomonaga 1977, Larsson *et al.* 1978, Yu *et al.* 2007). The type II-to-type I fibre area ratio observed in older master sprinters in this study was similar to previous observations in sedentary older men (Larsson *et al.* 1978, Yu *et al.* 2007). The CSA of type II (IIA) and type I muscle fibres increased markedly in EX, although it did not reach statistical significance. Consequently, the type II-to-type I fibre area ratio did not increase significantly and remained lower in older sprinters than in sedentary young men [1.20 ± 0.07 measured from skinned single fibres (Yu *et al.* 2007), 1.24 ± 0.05 measured from biopsy cross-sections (Larsson *et al.* 1978)] and in young adult sprinters [1.21 ± 0.04 measured from biopsy cross sections (Korhonen *et al.* 2006)]. Training-induced muscle fibre growth is known to be influenced by satellite cell function (Hawke & Garry 2001, Thornell *et al.* 2003). Further research is needed to understand whether years of intensive training in master athletes leads to changes in satellite cell properties (number, activation, proliferation, chemotaxis, fusion) and hence influence the fibre type-specific hypertrophic adaptations.

The histochemical fibre type distribution and the relative MyHC isoform content remained unaffected, suggesting that the combination of strength and sprint training does not cause a change in muscle fibre or MyHC isoform composition in older male sprinters (Table 2). The two published studies on young sprint athletes have reported divergent results concerning the effects of simultaneous strength and sprint training on muscle fibre distribution. Cadefau *et al.* (1990) reported an increase in the percentage of type I fibres in response to 8 months of training in 16- to 18-year-old male and female sprinters, while Andersen *et al.* (1994) found bidirectional transformation towards IIa fibres and MyHC isoform composition ($I \rightarrow IIa \leftarrow IIb/IIx$) with 3 months of intensive training in 20- to 27-year-old male sprinters. The reason for the discrepancies between our and the previous findings is unclear but could be accounted for by differences in age of subjects, total training duration, and the type, intensity and relative amount of strength and sprint exercises in the training programmes.

Ageing is known to affect both the ST and maximum velocity of unloaded shortening in single fibres expressing the type I and IIa MyHC isoforms in untrained people (Larsson *et al.* 1997, D'Antona *et al.* 2003, Ochala *et al.* 2007). Our previous cross-sectional study has provided evidence that while systematic sprint training alone can maintain the ST of type I and IIa MyHC fibres, it is unable to prevent the ageing-related atrophy of type II fibres and the slowing of type I fibres (Korhonen *et al.* 2006). The results of the present follow-up study show that the addition of a high-intensity strength training stimulus to overall training did not produce significant adaptations in the ST and maximum velocity of unloaded shortening at the single muscle fibre level, although a trend towards higher V_o of type I MyHC fibres was observed in response to the training. The lack of a significant training effect on the ST and V_o agrees with the strength (Widrick *et al.* 2002, Shoepe *et al.* 2003) and sprint training (Harridge *et al.* 1998) studies in young men, but is contradicted by some studies in older sedentary people that have shown increases in the ST of type I fibres (Frontera *et al.* 2003) and V_o of type I and IIa fibres (Trappe *et al.* 2000) after 12-week strength training. It could be hypothesized that

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these differential training effects reflect various physical activity histories and that the improved single-fibre function in sedentary older subjects is partially explained by change from inactivity to active usage of muscles.

On the basis of recent research, resistance training can influence the qualitative properties of the fast myosin isoform in young and older subjects (Canepari *et al.* 2005). The ST generated at the single-fibre level is dependent on the number of active cross-bridges and the tension generated by each cross-bridge. The absence of a change in ST seems to suggest that the 20-week intensive training programme did not lead to further increases in the number or force-generating capacity of the cross-bridges in older sprint athletes in the present study.

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Neural adaptations

Several studies have shown an increase in muscle EMG with resistance training, especially during the earlier weeks, and this is regarded as evidence for enhanced neural drive to a muscle (Moritani & deVries 1980, Narici *et al.* 1989, Hakkinen & Hakkinen 1995, Reeves *et al.* 2005). In the present study, the 10% improvement in squat jump performance in EX was accompanied by a significant 9% increase in the iEMG of the agonist leg extensor muscles, and this was significantly greater than in CTRL (Fig. 4). Within the limitations of absolute surface EMG measurements, the increased iEMG response in the initial movement phase of the squat jump suggests adaptations in the rapid neural activation of motor units. Increased EMG development at the onset of a muscular contraction has been observed in previous strength training studies in young (Hakkinen *et al.* 1985, Van Cutsem *et al.* 1998, Aagaard *et al.* 2002) and older adults (Hakkinen & Hakkinen 1995, Hakkinen *et al.* 1998, Barry *et al.* 2005). This may reflect adaptations in the motor unit recruitment pattern, for instance, earlier motor unit activation, increased firing frequency and brief interspike intervals (doublets) in the EMG burst (Van Cutsem *et al.* 1998).

The training-induced increases in maximal isometric knee extension and knee flexion torque as well as dynamic 1-RM squat in the EX group were not accompanied by a significant change in the maximum iEMGs of the agonist muscles (Fig. 4), suggesting that the addition of strength training stimulus in already sprint-trained athletes may not lead to increases in neural drive during maximal slow concentric and isometric contractions. It is important to emphasize that despite thorough control of the measurement procedures, EMG recording is always liable to various errors (Farina *et al.* 2004) and we recognize the difficulties in data interpretation on the basis of changes in surface EMG. Further studies using electrical stimulation and intramuscular EMG recording techniques are needed to complement the findings of the present study.

Among older people, poor muscle force production is a significant risk factor for mobility disability and incidence of falls (Bean *et al.* 2003, Moreland *et al.* 2004). Thus, a better understanding of the mechanisms underlying strength deficits and ways of maintaining or augmenting muscular function during ageing is of significant importance. The present training study in elite master athletes provides new information, independent of deconditioning effect, on the plasticity of the neuromuscular system in older age. We found improved strength along with fibre hypertrophy in older muscle in response to increased strength training employing periodization procedures. This suggests that the adaptability of the skeletal muscle to altered functional demands and the concepts of progressive overload and specificity of training also apply to older habitually trained men. Strength training containing both heavy resistance and high-power exercises may be effective in preventing fast fibre atrophy and loss of explosive force production, which are critical changes in the normal ageing process.

Methodological considerations

For technical reasons, the muscle biopsies for single-fibre measurements could only be obtained from a small sample of 12 athletes. As a result of the original randomization that was done after the baseline measurements, and exclusion of one control subject, the present study comprised seven experimental and four control subjects. Due to the sample size, the statistical power of this study was limited to detecting differences in changes between groups in the outcome variables. It may be argued whether a single needle biopsy is representative of the whole muscle in spite of the fact that care was taken to achieve a consistent biopsy location in pre- and post-biopsies in all subjects. Small variations in the location of the muscle sample will not influence measurements of regulation of muscle contraction at the single muscle fibre level in muscle fibres expressing specific MyHC isoforms. However, it cannot be completely ruled out that it might affect fibre CSA and fibre type proportions, although this appears unlikely. Furthermore, differences in training background may affect the magnitude of training-induced adaptations. However, the subjects in this study represent a homogeneous group of elite sprinters with no background in heavy strength training, making differences in baseline training status an unlikely bias in this study.

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Conclusions

Trained master athletes represent a valuable model for estimating the effects of resistance training in older age. Combined strength and sprint training programme at high intensities have proved useful for the development of the maximal and explosive force. Muscular adaptive mechanisms, primarily hypertrophy of muscle fibres expressing fast MyHC isoforms, appear to be the dominant adaptation to this type of training in highly trained and motivated older subjects. Improvements in dynamic explosive force production during more complex movements may also be associated with increased rapid neural activation of motor units.

Conflict of interest

There is no conflict of interest.

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References

Aagaard, P., Andersen, J.L., Dyhre-Poulsen, P., Leffers, A.M., Wagner, A., Magnusson, S.P., Halkjaer-Kristensen, J. & Simonsen, E.B. 2001. A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. *J Physiol* **534**, 613–623.

Aagaard, P., Simonsen, E.B., Andersen, J.L., Magnusson, P. & Dyhre-Poulsen, P. 2002. Increased rate of force development and neural drive of human skeletal muscle following resistance training. *J Appl Physiol* **93**, 1318–1326.

Andersen, J.L. & Aagaard, P. 2000. Myosin heavy chain IIX overshoot in human skeletal muscle. *Muscle Nerve* **23**, 1095–1104.

Andersen, J.L., Klitgaard, H. & Saltin, B. 1994. Myosin heavy chain isoforms in single fibres from m. vastus lateralis of sprinters: influence of training. *Acta Physiol Scand* **151**, 135–142.

Asmussen, E. & Bonde-Petersen, F. 1974. Storage of elastic energy in skeletal muscles in man. *Acta Physiol Scand* **91**, 385–392.

Barry, B.K., Warman, G.E. & Carson, R.G. 2005. Age-related differences in rapid muscle activation after rate of force development training of the elbow flexors. *Exp Brain Res* **162**, 122–132.

Bean, J.F., Leveille, S.G., Kiely, D.K., Bandinelli, S., Guralnik, J.M. & Ferrucci, L. 2003. A comparison of leg power and leg strength within the InCHIANTI study: which influences mobility more? *J Gerontol A Biol Sci Med Sci* **58**, 728–733.

Bergstrom, J. 1962. Muscle electrolytes in man. Determination by neutron activation analysis on needle biopsy specimens. A study on normal subjects, kidney patients and patients with chronic diarrhoea. *Scand J Clin Lab Invest* (Suppl. 68), 1–110.

Bosco, C., Luhtanen, P. & Komi, P.V. 1983. A simple method for measurement of mechanical power in jumping. *Eur J Appl Physiol Occup Physiol* **50**, 273–282.

Bret, C., Rahmani, A., Dufour, A.B., Messonnier, L. & Lacour, J.R. 2002. Leg strength and stiffness as ability factors in 100 m sprint running. *J Sports Med Phys Fitness* **42**, 274–281.

Cadefau, J., Casademont, J., Grau, J.M., Fernandez, J., Balaguer, A., Vernet, M., Cusso, R. & Urbano-Marquez, A. 1990. Biochemical and histochemical adaptation to sprint training in young athletes. *Acta Physiol Scand* **140**, 341–351.

Canepari, M., Rossi, R., Pellegrino, M.A., Orrell, R.W., Cobbold, M., Harridge, S. & Bottinelli, R. 2005. Effects of resistance training on myosin function studied by the in vitro motility assay in young and older men. *J Appl Physiol* **98**, 2390–2395.

D'Antona, G., Pellegrino, M.A., Adami, R., Rossi, R., Carlizzi, C.N., Canepari, M., Saltin, B. & Bottinelli, R. 2003. The effect of ageing and immobilization on structure and function of human skeletal muscle fibres. *J Physiol* **552**, 499–511.

Delecluse, C. 1997. Influence of strength training on sprint running performance. Current findings and implications for training. *Sports Med* **24**, 147–156.

Delecluse, C., Van Coppenolle, H., Willems, E., Van Leemputte, M., Diels, R. & Goris, M. 1995. Influence of high-resistance and high-velocity training on sprint performance. *Med Sci Sports Exerc* **27**, 1203–1209.

Edman, K.A. 1979. The velocity of unloaded shortening and its relation to sarcomere length and isometric force in vertebrate muscle fibres. *J Physiol* **291**, 143–159.

Fabiato, A. 1988. Computer programs for calculating total from specified free or free from specified total ionic concentrations in aqueous solutions containing multiple metals and ligands. *Methods Enzymol* **157**, 378–417.

Farina, D., Merletti, R. & Enoka, R.M. 2004. The extraction of neural strategies from the surface EMG. *J Appl Physiol* **96**, 1486–1495.

Frontera, W.R. & Larsson, L. 1997. Contractile studies of single human skeletal muscle fibers: a comparison of different muscles, permeabilization procedures, and storage techniques. *Muscle Nerve* **20**, 948–952.

Frontera, W.R., Meredith, C.N., O'Reilly, K.P., Knuttgen, H.G. & Evans, W.J. 1988. Strength conditioning in older men: skeletal muscle hypertrophy and improved function. *J Appl Physiol* **64**, 1038–1044.

Frontera, W.R., Hughes, V.A., Fielding, R.A., Fiatarone, M.A., Evans, W.J. & Roubenoff, R. 2000. Aging of skeletal muscle: a 12-yr longitudinal study. *J Appl Physiol* **88**, 1321–1326.

Frontera, W.R., Hughes, V.A., Krivickas, L.S., Kim, S.K., Foldvari, M. & Roubenoff, R. 2003. Strength training in older women: early and late changes in whole muscle and single cells. *Muscle Nerve* **28**, 601–608.

Hakkinen, K. & Hakkinen, A. 1995. Neuromuscular adaptations during intensive strength training in middle-aged and elderly males and females. *Electromyogr Clin Neurophysiol* **35**, 137–147.

Hakkinen, K., Komi, P.V. & Alen, M. 1985. Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. *Acta Physiol Scand* **125**, 587–600.

Hakkinen, K., Kallinen, M., Izquierdo, M., Jokelainen, K., Lassila, H., Malkia, E., Kraemer, W.J., Newton, R.U. & Alen, M. 1998. Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. *J Appl Physiol* **84**, 1341–1349.

Hakkinen, K., Kraemer, W.J., Pakarinen, A., Triplett-Mcbride, T., Mcbride, J.M., Hakkinen, A., Alen, M., Mcguigan, M.R., Bronks, R. & Newton, R.U. 2002. Effects of heavy resistance/power training on maximal strength, muscle morphology, and hormonal response patterns in 60-75-year-old men and women. *Can J Appl Physiol* **27**, 213–231.

Harridge, S.D., Bottinelli, R., Canepari, M., Pellegrino, M., Reggiani, C., Esbjornsson, M., Balsom, P.D. & Saltin, B. 1998. Sprint training, in vitro and in vivo muscle function, and myosin heavy chain expression. *J Appl Physiol* **84**, 442–449.

Harris, G.R., Stone, M.H., O'Bryant, H.S., Proulx, C.M. & Johnson, R.L. 2000. Short-term performance effects of high power, high force, or combined weight-training methods. *J Strength Cond Res* **14**, 14–20.

Hawke, T.J. & Garry, D.J. 2001. Myogenic satellite cells: physiology to molecular biology. *J Appl Physiol* **91**, 534–551.

Hermens, H.J., Freriks, B., Merletti, R., Stegeman, D., Blok, J., Rau, G., Disselhorst-Klug, C. & Hagg, G. 1999. SENIAM-European Recommendations for Surface Electromyography. Roessingh Research and Development, Enschede, the Netherlands.

Izquierdo, M., Hakkinen, K., Ibanez, J., Garrues, M., Anton, A., Zuniga, A., Larrion, J.L. & Gorostiaga, E.M. 2001. Effects of strength training on muscle power and serum hormones in middle-aged and older men. *J Appl Physiol* **90**, 1497–1507.

Joch, W. (ed.) 1992. *Rahmentrainingsplan für das Aufbautraining Sprint*. Meyer & Meyer Verlag, Aachen, Germany.

Korhonen, M.T., Cristea, A., Alen, M., Hakkinen, K., Sipila, S., Mero, A., Viitasalo, J.T., Larsson, L. & Suominen, H. 2006. Aging, muscle fiber type, and contractile function in sprint-trained athletes. *J Appl Physiol* **101**, 906–917.

Kraemer, W.J. & Häkkinen, K. (eds) 2002. *Strength Training for Sport*. Blackwell Science, Oxford, UK.

Larsson, L. 1982. Physical training effects on muscle morphology in sedentary males at different ages. *Med Sci Sports Exerc* **14**, 203–206.

Larsson, L. & Moss, R.L. 1993. Maximum velocity of shortening in relation to myosin isoform composition in single fibres from human skeletal muscles. *J Physiol* **472**, 595–614.

Larsson, L., Sjodin, B. & Karlsson, J. 1978. Histochemical and biochemical changes in human skeletal muscle with age in sedentary males, age 22–65 years. *Acta Physiol Scand* **103**, 31–39.

Larsson, L., Li, X. & Frontera, W.R. 1997. Effects of aging on shortening velocity and myosin isoform composition in single human skeletal muscle cells. *Am J Physiol* **272**, C638–C649.

Mero, A. & Komi, P.V. 1986. Force-, EMG-, and elasticity-velocity relationships at submaximal, maximal and supramaximal running speeds in sprinters. *Eur J Appl Physiol* **55**, 553–561.

Mero, A., Luhtanen, P., Viitasalo, J.T. & Komi, P.V. 1981. Relationships between the maximal running velocity, muscle fiber characteristics, force production and force relaxation in sprinters. *Scand J Sports Sci* **3**, 16–22.

Moreland, J.D., Richardson, J.A., Goldsmith, C.H. & Clase, C.M. 2004. Muscle weakness and falls in older adults: a systematic review and meta-analysis. *J Am Geriatr Soc* **52**, 1121–1129.

Moritani, T. & deVries, H.A. 1980. Potential for gross muscle hypertrophy in older men. *J Gerontol* **35**, 672–682.

Moss, R.L. 1979. Sarcomere length-tension relations of frog skinned muscle fibres during calcium activation at short lengths. *J Physiol* **292**, 177–192.

Narici, M.V., Roi, G.S., Landoni, L., Minetti, A.E. & Cerretelli, P. 1989. Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *Eur J Appl Physiol Occup Physiol* **59**, 310–319.

Newton, R.U., Hakkinen, K., Hakkinen, A., Mccormick, M., Volek, J. & Kraemer, W.J. 2002. Mixed-methods resistance training increases power and strength of young and older men. *Med Sci Sports Exerc* **34**, 1367–1375.

Ochala, J., Frontera, W.R., Dorer, D.J., Van Hoecke, J. & Krivickas, L.S. 2007. Single skeletal muscle fiber elastic and contractile characteristics in young and older men. *J Gerontol A Biol Sci Med Sci* **62**, 375–381.

Paavolainen, L., Hakkinen, K., Hamalainen, I., Nummela, A. & Rusko, H. 1999. Explosive-strength training improves 5-km running time by improving running economy and muscle power. *J Appl Physiol* **86**, 1527–1533.

Pereira Sant'Ana, J.A., Ennion, S., Sargeant, A.J., Moorman, A.F. & Goldspink, G. 1997. Comparison of the molecular, antigenic and ATPase determinants of fast myosin heavy chains in rat and human: a single-fibre study. *Pflugers Arch* **435**, 151–163.

Reeves, N.D., Maganaris, C.N. & Narici, M.V. 2005. Plasticity of dynamic muscle performance with strength training in elderly humans. *Muscle Nerve* **31**, 355–364.

Shoepe, T.C., Stelzer, J.E., Garner, D.P. & Widrick, J.J. 2003. Functional adaptability of muscle fibers to long-term resistance exercise. *Med Sci Sports Exerc* **35**, 944–951.

Thornell, L.E., Lindstrom, M., Renault, V., Mouly, V. & Butler-Browne, G.S. 2003. Satellite cells and training in the elderly. *Scand J Med Sci Sports* **13**, 48–55.

Tomonaga, M. 1977. Histochemical and ultrastructural changes in senile human skeletal muscle. *J Am Geriatr Soc* **25**, 125–131.

Trappe, S., Williamson, D., Godard, M., Porter, D., Rowden, G. & Costill, D. 2000. Effect of resistance training on single muscle fiber contractile function in older men. *J Appl Physiol* **89**, 143–152.

Van Cutsem, M., Duchateau, J. & Hainaut, K. (1998) Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. *J Physiol* **513**(Pt 1), 295–305.

Widrick, J.J., Stelzer, J.E., Shoepe, T.C. & Garner, D.P. 2002. Functional properties of human muscle fibers after short-term resistance exercise training. *Am J Physiol Regul Integr Comp Physiol* **283**, R408–R416.

Yu, F., Hedstrom, M., Cristea, A., Dalen, N. & Larsson, L. 2007. Effects of ageing and gender on contractile properties in human skeletal muscle and single fibres. *Acta Physiol (Oxf)* **190**, 229–241.

Appendix

The training program consisted of two 11- and 9-week periods that were further divided into three 3–4 week phases with variations in training intensity, volume and type. The aim of the strength training was to increase maximal and explosive strength and promote muscle hypertrophy. The first 4 weeks of training involved low intensity and high volume strength endurance/hypertrophy exercises (3–4 sets × 8–12 repetitions at 50–70% of 1-RM) to prepare the muscles for more intensive training in the following phases. In the second and third phases, maximal strength (2-3 × 4–6 repetitions at 70–85%), and explosive-type weightlifting (2–3 × 4–6 repetitions at 35–60%) and plyometric exercises (2–3 × 3–10 repetitions) were undertaken and alternated within a week to allow recovery from different types of exercise stress. During the latter half of the training programme, the three-phase protocol was repeated with a slight progressive increase in training intensity aimed at inducing a further overload stimulus and peak maximal and explosive strength at the end of the training period. Maximal strength and plyometric exercises had already been included in the first phase (weeks 12–14) in the second training period. Strength training was performed two times per week on non-consecutive days and each session lasted 50–90 min.

The strength training focused on the leg extensor and hamstring muscle groups and the main exercises that were performed at the beginning of the training sessions included leg press and/or half squat on machines, clean pull (from knee height) and/or stiff leg deadlift (Romanian lift) using free weights. The supplementary dynamic exercises (using whole range of motion) were hip extension, hip flexion, knee flexion, knee extension and ankle plantar flexion on machines. In addition, each training session included two to four exercises for the other main muscle groups of the body (trunk extension, trunk flexion, bench press, push press, and sprinting arm movements with and without hand weights). Plyometric exercises, utilized as a part of explosive strength training, progressed over the training period from low intensity vertical jumps to horizontal bounding exercises. These exercises were performed at the beginning of the speed training sessions.

The aim of the sprint training was to increase acceleration and maximum speed abilities in running. In general, it followed the athletes' usual training regimen but the overall volume was decreased when strength exercises were incorporated into the programme. The schedule for sprint training was similar in the first and second half of the training period. Sprint training was started with a combination of low intensity, high volume speed-endurance intervals (3–5 × 200–250 m at 75–85% of max. speed) and acceleration practices from the standing start position (4 × 30 m at 80%) to develop the requisite muscular and metabolic base for subsequent training. In the second and third phases, maximum speed exercises were added and intensified gradually up to almost competitive pace (2–3 × 30–80 m at 90–98%) while the total running distance covered was decreased. In addition, exercises for explosive starting and high acceleration from starting blocks (2–4 × 30 m at 90–98%) were included. Each sprint training session included drills to improve coordination and running technique. Speed training was performed two times weekly on non-consecutive days and each session lasted 50–90 min.

