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ORIGINAL ARTICLE

Muscle mechanical characteristics in fatigue and recovery from a marathon race in highly trained runners

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Abstract The aim of the present study was to examine muscle mechanical characteristics before and after a marathon race. Eight elite runners underwent a pre-test 1 week before the marathon and post-tests 30 min, two and five-day-post-marathon. Actual marathon race performance was 2:34:40 ± 0:04:13. Energy expenditure at marathon pace (EE_{Mpace}) was elevated 4% post-marathon (pre: 4,465 ± 91 vs. post 4,638 ± 91 J kg bodyweight^{-1} km^{-1}, $P < 0.05$), but was lowered by 6 and 9.5% two- and five-day-post-marathon compared to EE_{Mpace} pre-marathon. Counter-movement jump (CMJ) power decreased 13% post-marathon (pre: 21.5 ± 0.9 vs. post: 18.9 ± 1.2 W kg^{-1}; $P < 0.05$) and remained depressed two- (18%) and five-day (12%) post-marathon. CMJ force was unaltered across all four tests occasions. Knee extensor and plantar flexor maximal voluntary contraction (MVC) decreased from 176.6 ± 9.5 to 136.7 ± 16.8 Nm and 144.9 ± 8.7 to 119.2 ± 15.1 Nm post-marathon corresponding to 22 and 17%, respectively ($P < 0.05$). No significant changes were detected in evoked contractile parameters, except a 25% increase in force at 5 Hz, and low frequency fatigue was not observed. In conclusion, leg muscle power decreased acutely post-marathon race and recovered very slowly. The post-marathon increase in EE_{Mpace} might be attributed to a reduction in stretch shortening cycle efficiency. Finally, since MVC was reduced after the marathon race without any marked changes in evoked muscle contractile properties, the strength fatigue experienced by the subjects in this study seems to be related to central rather than peripheral mechanisms.

Keywords Marathon performance · Neuromuscular fatigue · Running economy · Endurance athletes

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Introduction

For many years exercise physiologists have studied the potential limiting factors for endurance performance in untrained and trained individuals. Many studies have focused on metabolic factors, primarily muscle and liver glycogen depletion, as a cause of fatigue in prolonged exercise such as the marathon event. Low glycogen levels in muscle have been found in fatigued subjects (Tzintzas et al. 1996; Sherman et al. 1983; Callow et al. 1986), and performance was markedly impaired when subjects were given a low-carbohydrate diet before exercise (Bergstrøm et al. 1967). However no studies have been able to establish a direct link between glycogen depletion and fatigue, and substantial quantities of glycogen remain in the muscles at exhaustion in highly trained athletes (Madsen et al. 1990, 1993; Noakes et al. 1988; Noakes 2001). Training status might play an important role since endurance trained athletes oxidize less carbohydrate and have substatial higher glycogen stores compared to sedentary subjects for the same absolute work due to a lower rate of glycogenolysis during exercise (Gollnick and Saltin 1988; Coggan and Williams 1995).

Therefore other factors seem to play a governing role in fatigue during prolonged exercise in well-trained athletes. More recent research on prolonged exercise has addressed the role of muscle and neuromuscular fatigue in relation to endurance performance. Neuromuscular fatigue can be

divided into central and peripheral fatigue. Central fatigue includes both supraspinal and spinal factors, whereas peripheral fatigue refers to muscle contractile properties and the transmission and conduction velocity of the action potential from the nerve endings across the sarcolemma (Gandevia 2001; Stein 1974).

Numerous studies have observed reduced contraction strength (MVC) for the knee extensor muscles (KE) in endurance trained athletes after prolonged running at distances from 30 to 65 km (Place et al. 2004; Millet et al. 2002, 2003; Davies and Thompson 1986; Nicol et al. 1991b). Both Millet et al. (2003) and Place et al. (2004) found a strong correlation between KE MVC loss and maximal voluntary activation (MVA) deficit, pointing in the direction of central fatigue (Gandevia 2001). Additionally alterations in electrically evoked muscle contractile properties after prolonged running have also been reported (Millet et al. 2003; Place et al. 2004; Skof and Strojnik 2006).

Prolonged running seems to induce a worsening in running economy (RE), which was suggested to be of both biomechanical and physiological origin (Nicol et al. 1991a; Kyrolainen et al. 2000; Guezennec et al. 1996). Nicol et al. (1991c) found that a worsening in RE after a marathon run was related to deterioration in the resistance to impact. Furthermore Byrne et al. (2004) suggested that a reduced neural activation in the eccentric phase of the stretch shortening cycle (SSC) resulted in impaired muscle-tendon elastic properties and thus caused mechanical efficiency to decrease. The above findings are well in accordance with early work by Cavagna et al. (1964) who suggested that a short transition from the breaking to the push-off phase is a necessity for efficient use of elastic recoil in human movement. Based on these findings one might speculate that there could be a direct link between neuromuscular and biomechanical efficiency during the running stride. Other physiological factors responsible for the worsening in RE include an increase in ventilation, greater reliance of fat as a utilization fuel, increase in core temperature and progressive type II muscle fiber recruitment (Saunders et al. 2004; Kyrolainen et al. 2000; Williams and Cavanagh 1987).

The recovery pattern after prolonged running has not been studied extensively. Avela et al. (1999) reported vastly different recovery patterns in some of the parameters tested, for instance, plantar flexor (PF) MVC had recovered two-day post-marathon, while landing impact forces in counter movement jump (CMJ) had not recovered six-day post-marathon. Other studies on intense SSC exercise show a prolonged recovery phase (up to 7 days) in both PF MVC and joint stiffness regulation (Kuitunen et al. 2002, 2004). Studying the fatigue recovery process in elite marathon runners enables to examine possible differences in physiological and biomechanical recovery patterns.

Based on previous studies we hypothesized that a reduction in both maximal muscle strength and maximal muscle power would occur after a marathon race and that a relationship would exist between the fatigue induced drop in strength/power KE/PF and running performance, respectively. In addition, it was hypothesized that EE would be worsened after a marathon race due to a decrease in neuromuscular efficiency.

Purpose

The aim of this study was to examine the magnitude of neuromuscular fatigue, i.e., central versus peripheral factors induced by elite marathon running. In addition, EE was examined pre and post the marathon run in order to find possible links between EE and neuromuscular fatigue variables. Finally, it was the aim to follow the recovery in neuromuscular capacity and EE in elite marathon runners in the days after a marathon race.

Methods

Subjects

Eleven male elite marathon runners volunteered to participate in this study. Three subjects failed to complete the race due to injury (one) and early fatigue (two), and were excluded from the study. Thus eight male experienced and competitive Danish national elite and subelite marathon runners with physical and performance characteristics as illustrated in Table 1 completed the study. All subjects gave their written consent to participate in the study. The study was approved by the local ethical committee (VF20040196) and was carried out in accordance with the Helsinki II Declaration.

Experimental design, overview

Subjects reported to the laboratory on four occasions: 7 days before, immediately after (20–30 min), 2 days after, and 5 days after the marathon race. In the lab each subject underwent VO_{2max} (only pre-test)—and $VO_{2submax}$ tests on a motor-driven treadmill. Isometric strength parameters (MVC) for PF and KE were measured in all occasions. Evoked muscle contractile properties were measured by cutaneous electrical stimulation of the m.vastus lateralis. To get a measure of the functional strength and power a countermovement jump (CMJ) on a force platform was performed.

The eight subjects were divided into two groups due to practical reasons. Each group followed the same order of tests on all four occasions in order to minimize the effects

Table 1 Anthropometric, training, and test data of the runners completing the study

Variables	Subjects (n = 8)	Range
Anthropometric data		
Age (years)	32.5 ± 2.2	24–44
Height (cm)	180.9 ± 3.1	167–196
Weight (kg)	67.2 ± 2.0	59.8–77.7
Percent fat (%)	9.6 ± 0.7	5.7–12.9
History of running		
Years of training (years)	9 ± 1	5–15
Average weekly training distance (km)	117 ± 9	68–140
Marathon completed (no.)	7 ± 2	1–21
Best marathon performance (hh:mm:ss)	02:31:29 ± 0:03:31	02:22:37–02:54:00
Test data		
VO_{2max} (ml O_2 min^{-1} kg^{-1})	71.7 ± 1.3	67.5–77.2
VE at VO_{2max} (l min^{-1})	166.3 ± 4.9	151–184
Max lactate (mmol l^{-1})	8.7 ± 1.4	7.4–12.9
Running economy, Pre-test (ml O_2 kg^{-1} km^{-1})	212.6 ± 3.9	196–226
Fractional utilization of VO_{2max} at Mpace (%)	82.9 ± 2.1	71–94

Values are mean ± SE of eight subjects

of possible influence from the various tests. Subjects were given the same amount of rest time between the test-setups on all four occasions. The experimental design is presented in Table 2.

Before each test occasion, a standardized warm-up of 15 min running at a self-chosen pace was conducted by each subject (except after the marathon run). In the breaks between each test-setup subjects were ordered to relax, but were allowed to eat and drink ad libitum. Since treadmill running was the first test, there should be no effect of energy intake on RER. After the marathon run, the subjects were given 1/2 liter of water. They were restricted from any further water or energy intake until after the treadmill test. During the marathon run there was no restriction on either water or energy intake.

Marathon race

The marathon race was the Danish Marathon Championships. A real-race situation was chosen in order to secure a high level of motivation and performance. Five subjects ran a personal best time for a marathon, while the remaining three subjects were 6.7, 1.9, and 8.3% slower than their personal best times. Split times were recorded for every runner at 10, 20, 21.1, 30, 40, and 42.2 km. In addition, the marathon was videotaped at 8, 18, 28, and 38 km. From these sequences both stride length and stride frequency was measured as an average of three strides. A zone of 1-m was outlined across the course at the relevant points. The runners were then videotaped with a stationary video camcorder as they passed through the zone. With the use of the software program (Dart Trainer, Dartfish USA, Alpharetta, GA, USA) it was possible to determine stride length and stride frequency.

Treadmill test

Before the running tests various anthropometric measures were obtained. Subjects underwent a sub-maximal treadmill test at their expected marathon pace (Mpace) to determine their RE based on VO_2 measured at Mpace (VO_{2Mpace}). This test was followed by a VO_{2max} test. Mpace was decided from the runners' own expectations regarding the race and our knowledge of the runners' capacity, and should mimick the pace the runner could maintain for more than 2 h.

Table 2 Test protocol

Pre-test		Post-test	+2 and 5 days of recovery
7 days before the race	Marathon race	30 min after the race	2 and 5 days after the race
15 min warm-up		Sub max test (VO_{2Mpace})	15 min warm up
Sub max test (VO_{2Mpace})			Sub max test (VO_{2Mpace})
VO_{2max} test			
Group 1		Group 1	Group 1
MVC PF		MVC PF	MVC PF
Elec.stim + MVC KE		Elec.stim + MVC KE	Elec.stim + MVC KE
CMJ		CMJ	CMJ
Group 2		Group 2	Group 2
Elec.stim + MVC KE		Elec.stim + MVC KE	Elec.stim + MVC KE
CMJ		CMJ	CMJ
MVC PF		MVC PF	MVC PF

VO_{2Mpace}

The subjects ran for 6 min at their individual Mpace which ranged between 15.4 and 18.1 km h^{-1}. The mean Mpace was 1.5% faster than mean marathon time. Blood samples were taken from the fingertip immediately after and analyzed for lactate concentration after 6 min of sub maximal running. Blood samples were also taken 1 and 3 min after the VO_{2Mpace} on days where the VO_{2max} test was not performed. Heart rate was monitored continuously throughout the 6 min (Polar, Vantaa, Finland). In addition step frequency was recorded during sub-maximal running and used for calculation of stride length. Due to severe fatigue only four subjects were able to complete the VO_{2Mpace} test post-marathon ($n = 4$). The subjects wore an airtight mask, and performed the test in the same clothes and shoes they wore in the marathon race. Oxygen consumption was measured online by analyzing the expired air with the Amis 2000 system (Jensen et al. 2002).

VO_{2max}

After a 2 min break, VO_{2max} was determined at a running speed 1 km^{-1} h^{-1} above the subject's Mpace. Every 2-min the inclination was raised by 2% until exhaustion (Jensen et al. 1999). A leveling off in oxygen consumption during the last 30 s of the VO_{2max} test was used as criterion for exhaustion. Heart rate was monitored continuously during the VO_{2max} test (Polar, Finland). Blood samples were taken from the fingertip and analyzed for lactate concentration 0, 1, and 3 min after completion of the VO_{2max} test.

Calculations: treadmill test

The oxygen consumption, ventilation and RER values were determined by taking the median of the last 5 measurements (30 s). When using the breath-by-breath method it is possible that a single measurement deviates largely from the rest, therefore the median was chosen as a measure of the aforementioned parameters.

RE is traditionally measured as ml O_2 kg bodyweight^{-1} km^{-1} where the speed typically is set equivalent to the competition pace. In order to exclude the influence of an altered metabolism on RE, for instance an increased reliance of fat post-exercise, the total energy expenditure (EE), calculated as J kg bodyweight^{-1} km^{-1}, might be considered a more precise measure of changes in efficiency (Kyrolainen et al. 2000; Billat et al. 2003). RE_{Mpace} was measured as ml O_2 kg bodyweight^{-1} km^{-1} at Mpace. In order to compensate for the influence of altered metabolism on RE, the total EE was calculated in each of the four test occasions. To calculate the EE_{Mpace}, an energy equivalent of 20.202 kJ l O_2^{-1} was applied when RER was 0.82. Changes of ±0.01 in RER-value relative to 0.82 were assumed to correspond to ±50 J changes in EE (McArdle et al. 1996, p. 147). Relative oxygen consumption ($VO_{2fractional}$) was calculated as follows: $VO_{2Mpace}/VO_{2max} \cdot 100\%$.

Force plate test

All vertical jumps were performed on a Kistler platform (Kistler 9281 B, $40 \times 60 \times 5$ cm^3) (Caserotti et al. 2001; Holsgaard Larsen et al. 2006). The vertical force signal (Fz) was fed from the Kistler amplifier to an A/D converter in a personal computer (dt28ez Data Translation) at a 1,000-Hz sampling rate for 5-s (Lab View, National Instruments, Austin, TX, USA).

Starting from a standing position, the subjects (who were wearing their racing shoes) were instructed to perform a CMJ consisting of a fast downward movement to about 90° knee flexion, immediately followed by a fast upward movement, and to jump as high as possible. Hands were kept on the hips to minimize any influence of the arms. Prior to the test, the jump was demonstrated to the subjects, who subsequently performed 2–3 sub-maximal trials. Three CMJ were performed interspersed with a 45-s break between each jump. All jumps were taped on a camcorder and analyzed with computer software (Dart Trainer, Dartfish USA). This enabled us to see, if there were major deviations in knee angles between the attempts of the subjects. CMJ with the highest jump height of the body center of mass (BCM) in each test occasion was selected and analyzed for mean vertical force and power in the concentric phase of the jump. Due to unexpected technical problems with the force plate in the post-marathon test session, force integration analysis could not be applied. The following formulas therefore were used to calculate mean Force and mean power in all four test occasions:

Jump height was calculated based on flight time (T_{flight})

$$h = 1/8\ g \cdot t^2. \quad (1)$$

$V_{takeoff}$ was calculated based on jump height (h).

$$1/2v^2 = g \cdot h \Rightarrow V_{takeoff} = \sqrt{(2 \cdot g \cdot h)}. \quad (2)$$

Mean vertical jump force (Fz) in the concentric jump phase (i.e., with BCM moving upwards) was calculated based on the time duration of the phase (T_{con}). T_{con} was estimated as a given fraction of T_{total}, where T_{total} was the time from the start of the CMJ until takeoff. T_{con} was accurately determined by Fz integration in tests 1, 3, and 4 (see Caserotti et al. 2001). By assuming that in each subject the individual ratio of T_{con} to T_{total} was the same in all test

occasions, the mean T_{con}/T_{total} ratio (R1) was calculated from tests 1, 3, and 4 in each subject, respectively. Subsequently, this enabled us to calculate T_{con} from the recording of T_{total} (namely $T_{con} = R1 \times T_{total}$). T_{total} was determined by graphically inspecting the raw Fz signal by a moving cursor.

Based on T_{con} it was possible to calculate mean concentric vertical jumping force (F) by the following formula

$$F \cdot \Delta t = \Delta(m \cdot v) = m \cdot \Delta V \Rightarrow F_{mean} = (m \cdot \Delta V)/\Delta t \quad (3)$$

in which ΔV = take-off velocity ($V_{take\text{-}off}$), $\Delta t = T_{con}$, and m = mass of the subject.

Based on F_{mean} the mean concentric jump power (P_{mean}) was calculated using the formula

$$P_{mean} = F_{mean} \cdot V_{power-mean} \quad (4)$$

where $V_{power\text{-}mean}$ was calculated from Eq. 5 below.

$V_{power\text{-}mean}$ was accurately determined by force integration in tests 1, 3, and 4 (see Caserotti et al. 2001). By assuming that in each subject the individual ratio of $V_{power\text{-}mean}$ to $V_{take\text{-}off}$ T_{total} was the same in all test occasions, the mean $V_{power\text{-}mean}/V_{take\text{-}off}$ ratio (R2) was calculated from tests 1, 3, and 4 for each subject, respectively. Subsequently, this enabled to calculate $V_{power\text{-}mean}$ from $V_{take\text{-}off}$

$$V_{power-mean} = R2 \cdot V_{take-off} \quad (5)$$

The validity of this approach was evaluated by comparing the estimated values for F_{mean} (Eq. 3) and P_{mean} (Eq. 4) with the corresponding values derived by force integration in the trials where the force plate signal was intact (i.e., tests 1, 3, and 4). Excellent agreement between estimated and actual values were observed as revealed by linear regression analysis (F_{mean} : $R = 0.97$, $r^2 = 0.95$, $P < 0.0001$; P_{mean} : $R = 0.96$, $r^2 = 0.93$, $P < 0.0001$; test 3). Further, coefficient of variation (CV) was below 3% in all cases.

Maximal muscle strength (PF)

Maximal isometric voluntary PF strength at the ankle-joint was measured by use of a Kin-Com dynamometer (Kinetic Communicator, Chattecx Corp, Chattanooga, TN, USA). The reliability and validity of the Kin-Com-system have been described previously (Farrell and Richards 1986). The data were sampled to a personal computer at a sampling rate of 1,000 Hz for 5 s. Subjects were placed in a sturdy chair with an adjustable seat. Subjects were positioned with their back at 45° to the ground in order to minimize influence of additional muscle groups on measurements. The knee ankle was kept at 90°. A support pad was placed under the knee to maintain this knee angle and a strap was wrapped around the distal thigh to insure that the thigh did not move during the test.

The ankle-joint was 90° (neutral position), which was insured by use of an angle measurement device. The left foot was firmly strapped to a footplate. The force output was measured by a dynamometer strain gauge load cell placed under the footplate. To ensure correct measurement of the torque output, the lateral maleoli was visually aligned to the rotational center of the dynamometer lever arm. During measurements subjects had the arms crossed over the chest while the non-involved leg and upper-body was kept still.

Prior to the MVC attempts, the subjects had a warm-up consisting of four to five sub-maximal dynamic PF contractions at speeds of 30° and 180° per second and four to five maximal attempts at the same speeds. The subject was verbally encouraged in every trial and instructed to keep pressing for as long as the verbal encouragement lasted (~2 s). On-line visual feed back of the force signals was provided to the subjects on a PC screen. Each subjects completed three MVC attempts from which the best attempt was selected for further analysis.

Evoked mechanical muscle properties (KE)

Subjects were seated in an adjustable upright chair with a rigid vertical back support. The subjects were strapped to the seat around the shoulders and hip to prevent accessory upper body movement. Identical individual chair setting was used at all test occasions. A strain gauge dynamometer was fixed around the right shank 4 cm above the medial maleoli.

Prior to stimulation the skin was shaved and cleaned with alcohol. Electrodes (5×9 cm^2, Pals Axelgaard, Fallbook, CA, USA), were placed over the m.vastus lateralis muscle belly (10 cm above the patella and 15 cm below the spina iliaca antero-superior.) The position of the electrodes was marked with a pencil to insure that the same position was obtained in all test occasions. The electrodes were connected to a stimulator (Digitimer DS7A, Digitimer Welwyn Garden City, UK). Stimulation and data sampling (at 1,000 Hz) was controlled from a personal computer using "Spike 2" Version 4.18 (Cambridge Electronic Design, Cambridge, UK). Data sampling and stimulation were done by a CED Micron 1401 II 16-bit AD-Converter (CED).

Maximal single twitch traces were determined in order to find the current for the tetanic stimulation. The maximal single twitch tension was found by increasing the current intensity in 50 mA increments. Starting current for each subject was 300 mA. The current required to elicit 25% of peak tension was determined and used in each test occasion. The muscle was stimulated with successive square

wave pulses (100 μs time width) at 5, 10, 20, 50 or 100 Hz stimulus rate, respectively, with a cut off voltage of 230 V (Nielsen et al. 2005). Maximal single twitches and time to twitch peak tension was determined at each test occasion. Tetanic stimulation of the m.vastus lateralis was conducted at 5, 10, 20, 50, and 100 Hz (1-s duration) separated by 2 s. Evoked force peak at each stimulation frequency was determined after filtering the data with a low-pass filter (4th order zero-lag Butterworth filter, 13.5 Hz cutoff frequency). The time to peak tension (TPT) was determined as the period from the start of the stimuli to peak twitch tension.

The 20:50 Hz ratio was used in order to assess electrically evoked muscle properties. The 20:50 Hz ratio has often been used as a non-invasive method to examine muscle contractile properties. A decrease in this ratio—also known as low frequency fatigue (LFF)—is thought to be associated with failure of the E–C coupling system and thus may be a good indicator of peripheral mechanical dysfunction (Skurvydas et al. 2003). Abrupt changes in metabolic transients and muscle damage have been suggested to play a role in LFF (Jones et al. 1989; Chin et al. 1997). It was previously demonstrated that LFF could be accurately assessed in the quadriceps muscle by transcutaneous muscle stimulation while compared to direct motor nerve stimulation (Martin et al. 2004).

Before and after each test occasion the strain gauge was calibrated. All tests were conducted at a room temperature of 22–23°C and humidity of 27–46%.

Statistics

Statistics were calculated using "*Stat View*" for Windows (SAS Institute, Cary, NC, USA, 1999). The data recorded during the four test-occasions were statistically tested using a one-way ANOVA with repeated measures. Student–Neuman–Keuls post hoc test was applied to locate differences. Regression analysis was used to determine correlation coefficients between selected parameters. Multiple regression analysis was used to determine the correlation between VO_{2max}, RE, $VO_{2fractional}$, and marathon performance (MP). Results were accepted as significant at $P < 0.05$. The results are presented as mean ± standard errors.

Results

Marathon running

Marathon run time was 2:34:40 ± 0:04:13 h:mm:ss for the eight subjects at the day of the actual competition. VO_{2max} and RE_{Mpace} (pre-test) was 71.7 ± 1.3 ml O_2 min^{-1} kg^{-1} and 212.6 ± 3.9 ml O_2 kg^{-1} km^{-1}, respectively. $VO_{2fractional}$ was 84 ± 3%. A strong correlation was found between VO_{2max}, $VO_{2fractional}$, RE_{Mpace}, and MP ($R = 0.93$, $P < 0.05$). There was no correlation between RE_{Mpace} and MP, but a strong trend toward a correlation between VO_{2max} and MP was observed ($R = 0.64$, $P = 0.06$).

Treadmill test

EE_{Mpace} was significantly elevated by 5.2% post-marathon (pre: 4,465 ± 91 vs. post 4,638 ± 91 J kg bodyweight^{-1} km^{-1} $P < 0.05$). Conversely, two- and five-day post-marathon EE_{Mpace} was significantly lowered compared to pre-marathon (pre: 4,465 ± 91 vs. 2 days: 4,232 ± 95 vs. 5 days: 4,101 ± 77 J kg bodyweight^{-1} km^{-1} $P < 0.05$) (Fig. 1).

RER_{Mpace} of the subjects was significantly lower post-marathon (pre: 0.94 ± 0.02 vs. post: 0.88 ± 0.01) ($P < 0.05$), but had recovered two- and five-day post-marathon. Ventilation was significantly different between post-marathon and five-day post (post: 122.3 ± 4.5 l min^{-1} vs. 5 days: 98.3 ± 6.2 l min^{-1}) ($P < 0.05$, $n = 4$), no significant difference was found from pre- to post-marathon, but there was a trend toward a higher ventilation post-marathon (pre 109.5 ± 5.1 l min^{-1} vs. post 122.3 ± 4.5 l min^{-1}) ($P = 0.06$, $n = 4$). VE/VO_2 was significantly higher post-marathon (pre: 26.3 ± 0.8 vs. post: 29.5 ± 0.7) ($P < 0.05$, $n = 4$). Lactate at VO_{2Mpace} was unaltered at all four test occasions (pre: 2.8 ± 0.3 vs. post: 2.7 ± 0.5 vs. 2 days: 3.3 ± 0.3 vs. 5 days: 2.9 ± 0.6 $mmol^{-1}$ l^{-1}, $n = 4$). Heart rate showed no significant changes (pre: 163 ± 2 vs. post: 170 ± 6 vs. 2 days: 162 ± 4 vs. 5 days: 163 ± 4 beats min^{-1}, $n = 4$).

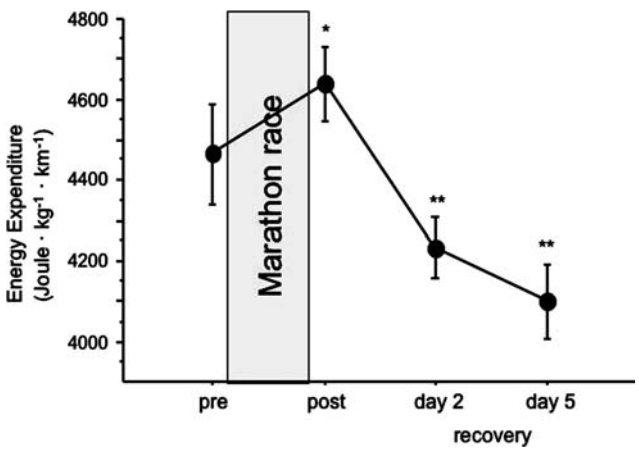

Fig. 1 Energy expenditure at the marathon pace (EE_{Mpace}), pre and post a marathon race, and after 2 and 5 days of recovery. *Single asterisk* denotes significantly higher than pre; *double asterisks* denotes significantly lower than pre ($P < 0.05$; $n = 4$)

Table 3 MVC for knee extensors (KE) and plantar flexors (PF), respectively

	MVC KE (Nm)	% Difference	MVC PF(Nm)	% Difference
Pre	176.6 ± 9.5		144.9 ± 8.7	
Post	136.7 ± 16.8*	–22.6 ± 7.1*	119.2 ± 15.1*	–17.7 ± 8.4*
2 days	165.5 ± 10.9	–6.3 ± 3.88	108.2 ± 10.0*	–25.4 ± 5.1*
5 days	179.8 ± 10.6	+1.8 ± 2.1	142.3 ± 7.0	–1.8 ± 4.3

Values are mean ± SE of eight subjects

*Significantly lower than pre ($P < 0.05$)

In the marathon race stride length decreased significantly with 14% from 8 to 38 km (8 km: 1.39 ± 0.05 m vs. 38 km: 1.19 ± 0.07 m) ($P < 0.01$, $n = 7$). Likewise, post-marathon stride length decreased significantly at Mpace (pre: 1.57 ± 0.05 m vs. post: 1.48 ± 0.07 m) ($P < 0.01$ $n = 4$). Two- and five-day post-marathon stride length had returned to pre-values.

Maximal voluntary contraction (MVC) for KE and PF muscles

MVC post-marathon was significantly lower for both KE and PF than pre (Table 3) ($P < 0.05$). From pre to two-day post-marathon there was no significant changes in KE MVC. In contrast, PF MVC remained depressed two-day post-marathon ($P < 0.05$). Five-day post-marathon both KE and PF MVC had recovered to pre-values. No significant correlation was observed between percent loss in time from the first to the second half marathon and percent force loss in neither KE MVC nor PF MVC.

Evoked mechanical muscle properties

Figure 2 shows changes in force at 5 and 50 Hz. While no significant differences in force were observed at 50 Hz evoked force increased post-marathon at 5 Hz (+25.3%, $P < 0.05$). The 20:50 Hz ratio remained unchanged during all test occasions. TPT, rate of force development (RFD), and rate of relaxation (RR) were unchanged across the test period. There were substantial inter-individual differences in the evoked force responses at 50 Hz. Three subjects showed an increase in force at 50 Hz of 1, 11, and 22%. For the remaining five subjects, force decreased from pre- to post-marathon with a span from 4 to 26%. A correlation was found to exist between Δ force at 50 Hz and ΔMVC ($R = 0.79$, $P < 0.05$) (Fig. 3).

Leg muscle power: CMJ

Post-marathon, mean concentric power in the CMJ was significantly reduced by 11% compared to pre (pre: 21.5 ± 0.9 vs. post: 18.9 ± 1.2 W kg^{-1}, $P < 0.01$). Five-day post-marathon mean CMJ power remained significantly suppressed compared to pre-marathon. There were no significant changes in mean CMJ force (Fig. 4).

The total time duration of a CMJ increased post-marathon and was significantly longer than pre (pre: 864.5 ± 65.3 ms vs. post: 1,040.8 ± 61.7 ms, $P < 0.01$). The eccentric phase increased significantly post-marathon (pre: 574.9 ± 49.0 ms vs. post: 725.3 ± 42.1 ms, $P < 0.01$) (Fig. 5). There was a trend toward a longer concentric phase post-marathon ($P = 0.067$) and also a trend toward a decreased relative duration of the concentric phase post-marathon ($P = 0.06$). Jump height post-marathon and 2 days post was significantly lower than pre-marathon (pre: 23.8 ± 1.1 cm vs. post: 21.7 ± 1.6 cm, 2 days post: 20.7 ± 1.1 cm) ($P < 0.05$).

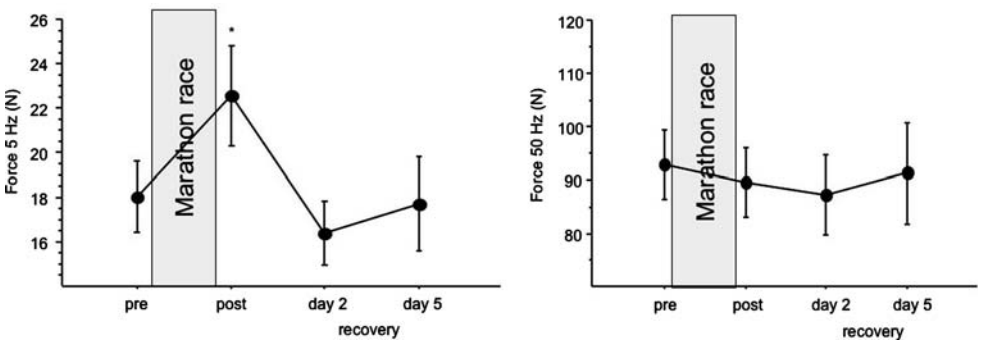

Fig. 2 Change in force at 5 Hz (*left*) and 50 Hz (*right*) in m.vastus lateralis pre and post a marathon race, and after 2 and 5 days of recovery. *Single asterisk* denotes significantly higher than pre ($P < 0.05$)

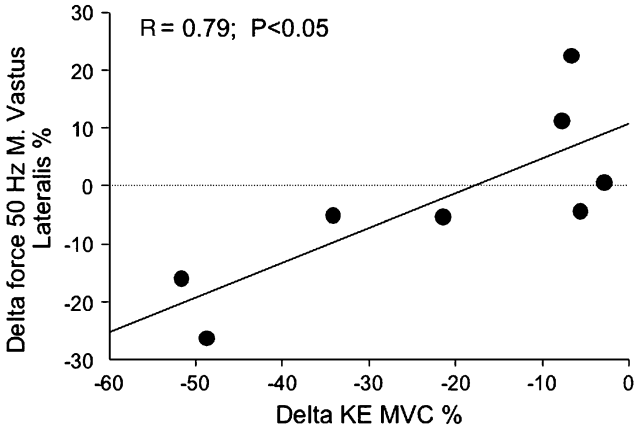

Fig. 3 Relationship between Δ electrically evoked tetanic force at 50 Hz on m.vastus lateralis and ΔKE MVC. Delta values are pre–post-marathon test ($R = 0.79$, $P < 0.05$)

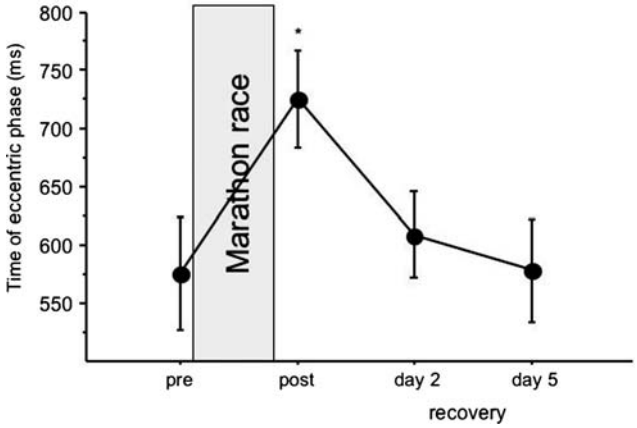

Fig. 5 The duration (in ms) of the eccentric phase in the countermovement jump test pre, post, 2- and 5-day post-marathon. *Single asterisk* denotes significantly higher than pre ($P < 0.05$)

Discussion

The main findings of this study were that in elite runners (1) EE at marathon pace was elevated by 4% after completing a marathon race, (2) a 25% decrease in KE MVC occurred without any signs of worsening in electrically evoked muscle contractile characteristics, (3) the strength recovery response differed between KE and PF muscles, and (4) mean SSC jumping power decreased without any changes in mean jumping force.

Neuromuscular changes

In the present study both PF and KE were examined due to potential difference in activation pattern during running. It can be speculated that PF is more active during running than KE, since studies have shown that glycogen depletion is far greater in PF than KE (Baldwin et al. 1973; Krssak et al. 2000). The decrease in PF MVC was 18%, and less than the 30% reduction reported by Avela et al. (1999). Surprisingly the reduction in KE MVC (25%) was larger than in PF MVC.

The post-marathon decrease in KE MVC is of the same magnitude found by Nicol et al. (1991a) (22%) who examined non-elite runners after a marathon. Other studies on prolonged running show decreases in KE MVC of the same magnitude (from 23 to 30%) (Nicol et al. 1991b; Millet et al. 2002, 2003; Place et al. 2004). However some important points regarding differences in study design needs to be addressed. Firstly, in our study the subjects had a break of 20–30 min before the post-marathon test, while subjects in other studies were tested immediately after the intervention (Millet et al. 2002, 2003; Place et al. 2004). This break could have resulted in recovery of KE and PF MVC force in our subjects, thus there is a possibility that KE and PF MVC would have been further depressed immediately after the marathon in our subjects, however Place et al. (2004) found no increase in KE MVC after a 30 min break compared with measurements made immediately after 5 h of running. Secondly, it is possible that differences in running surface can influence impact forces and thus the strain put on the KE muscles. For instance the subjects in the study of Place et al. (2004) ran on a motor-driven treadmill, while subjects in this study ran on

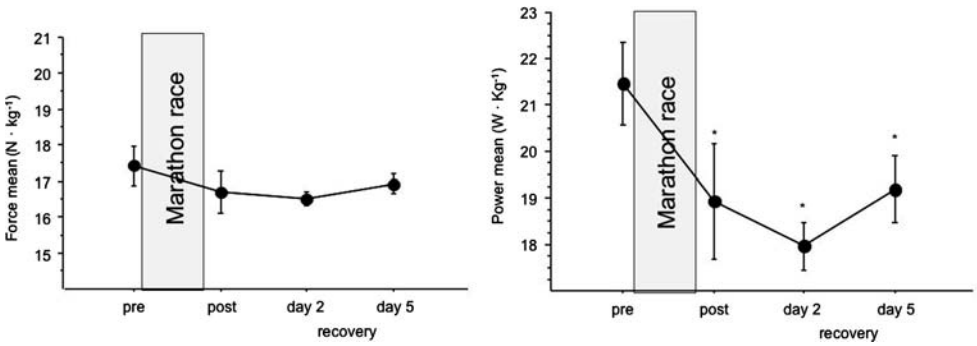

Fig. 4 *Left panel* illustrates the mean jumping force (F_{mean}) and right panel mean power (P_{mean}) in a countermovement jump pre, post, 2- and 5-day post-marathon. *Single asterisk* denotes significantly lower than pre ($P < 0.05$)

concrete road. Finally, several studies (Kyrolainen et al. 2000; Davies and Thompson 1986; Nicol et al. 1991a) have used a controlled setting, where running speed was pre-set. This could have resulted in a performance below the runners' maximum running capacity, which could have prevented fatigue.

While KE MVC had recovered to pre-marathon level, PF MVC further decreased by 9% from post-marathon to two-day post-marathon. Such large difference in the recovery pattern was unexpected, since reduction in post-marathon MVC strength for both PF and KE muscles were of a similar magnitude. We speculate that continuous prolonged high intensity exercise with long stride lengths could contribute to a greater active strain in the PF in the latter part of the stance phase, thereby leading to a greater amount of muscle and connective tissue damage. For example Lieber and Fridén (1993) reported that muscle damage in rabbit tibialis anterior muscle after eccentric work was a result of the magnitude of the active strain during muscle lengthening rather than high muscle forces. Furthermore we suggest that the significant decrease in PF MVC two-day post-marathon could be a result of an increased afferent feedback from nociceptors and metaboreceptors responding to accumulation of substrates involved in the inflammatory process.

Changes in energy expenditure

In order to compensate for potential differences in metabolism between the four VO_{2Mpace} tests (pre, post, 2 and 5 days), RE (ml O_2 min^{-1} kg^{-1}) was recalculated to EE as (J kg bodyweight^{-1} km^{-1}). The observed 4% increase in EE_{Mpace} post was of a smaller magnitude than the 12% increase reported by Kyrolainen et al. (2000). However the present subjects were better trained than the subjects in the study from Kyrolainen et al. (2000) which could explain some of the difference in the increase of EE_{Mpace}. The impaired EE_{Mpace} cannot be explained by one single factor, but is likely a product of several factors, reflecting both physiological and biomechanical alterations.

Some of the physiological explanations for the increase in EE_{Mpace} could be fluctuations in physiological parameters such as ventilation, core temperature, heart rate, and blood lactate (Franch et al. 1998; Saunders et al. 2004). A training study by Franch et al. (1998) found individual decrements in VE after a period of intense training that correlated with a reduced cost of running, potentially accounting for 25–70% of the decrease in aerobic demand. Metabolic products such as lactic acid might further play a role in the altered VE with fatigue. In the present study there was no sign of increased lactic acid levels post-marathon. Although there was no statistically significant increase in VE a strong trend toward an increase in VE was observed post-marathon ($P = 0.06$) and thus it seems plausible to assume that an increase in ventilation post-marathon could contribute to the elevation in EE_{Mpace}.

In SSC exercise like running it is commonly believed that the recoil of elastic energy by the muscle tendon system and muscle force potentiation will increase movement efficiency and thus reduce EE (Saunders et al. 2004; Van Ingen Schenau et al. 1997; Walshe et al. 1998). The length of the contact time might play a role in EE, since results from Paavolainen et al. (1999) indicate that shortened contact time occur concurrently with a lowering in the energy cost of running after a period with explosive resistance training.

In our study we did not examine the SSC during a running stride, but results from the CMJ test suggest that mechanical SSC output could have been affected post-marathon running and thus contributed to the elevated EE_{Mpace} in the present subjects. Power development in the CMJ test was significantly suppressed post-marathon with no significant decrease in mean takeoff force. The decrease in power development occurred concomitantly with an elongated eccentric phase (21%) post-marathon. We speculate that a prolonged eccentric phase will lead to less force potentiation (Walshe el al 1998; Van Ingen Schenau et al. 1997) and/or reduced conservation of elastic energy, since a short eccentric phase is a pre-requisite for stretch induced force enhancement (Saunders et al. 2004). Results from the CMJ test must be interpreted with caution, since there is no impact phase involved in a CMJ. However, Nicol et al. (1991a) found a reduced SSC efficiency in conjunction with a prolonged eccentric phase after SSC exercise with impact. In fact SSC performance is attenuated when SSC exercise is performed with impact forces (drop jumps, sledge drop jumps) compared to a SSC without impact (CMJ) (Nicol et al. 1991a).

Other mechanisms may act in order to counteract an elevation in EE_{Mpace} post-marathon such as an increase in muscle-tendon stiffness and electrically evoked force development at 5 Hz. Several studies have shown an increase in muscle-tendon stiffness in the days following eccentric work (Gleim et al. 1990; Craib et al. 1996; Chleboun et al. 1998; Harrison and Gaffney 2004). In addition the 25% increase in force at 5 Hz directly post-marathon may suggest a potentiation in intrinsic muscle fiber properties resulting in an improved $EE_{Mpace.}$ Muscle fiber potentiation at low Hz after endurance exercise lacks evidence, however potentiation at low frequency stimulation was previously reported by several authors after both isometric MVC's and tetanic stimulation of the m.vastus lateralis (Hamada et al. 2000; Rassier and McIntosh 2000; Sale 2002).

Central fatigue

A decrease in MVC can occur as a result of both central and peripheral factors. Data from the KE MVC was used in conjunction with results from the electrical stimulation, in order to elucidate whether the fatigue experienced in a MVC was of central or peripheral origin.

In the present study supraspinal fatigue could have occurred in our subjects post-marathon, since a depression in KE MVC without any concomitant worsening in electrically evoked forces was observed. However, distinctly different recovery patterns existed between KE MVC and PF MVC two-day post-marathon, disfavoring fatigue at the supraspinal level as this would be expected to affect all muscle groups to the same extent.

In the present study the decrease in KE MVC was larger than in PF MVC with no worsening in electrically evoked muscle contractile properties, thus it seems reasonable to speculate that fatigue mechanisms acutely post-marathon are predominantly influenced by spinal factors that are related to eccentric work. Such mechanisms could involve increased afferent input from nociceptors, since excessive eccentric work has been associated with muscle pain (Ebbeling and Clarkson 1989). Fatigue in the intrafusal fibers which could lead to reduced fusimotor support has also been mentioned as a cause of fatigue after SSC exercise (Avela and Komi 1998).

Peripheral fatigue

Peripheral fatigue is associated with changes in excitation–contraction (E–C) coupling, which could be due to a reduction in release and uptake of Ca^{2+} from the sarcoplasmatic reticulum (SR) (Allen et al. 1995). Modification of the E–C coupling system is also known to influence dynamic properties in the muscle such as the maximal RFD (Rassier and McIntosh 2000) and RR (Roberts and Smith 1989).

In our study force at both 20 and 50 Hz remained constant across all four tests occasions, as also the 20:50 Hz ratio remained constant, hence no LFF was noted. This result is rather surprising, since subjects in this study exercised at high submaximal intensities. In conjunction several studies have reported muscle damage after intensive SCC exercise (Kyrolainen et al. 1998, 2000; Strojnik and Komi 1998, 2000; Geronilla et al. 2003; Avela and Komi 1998; Kuitunen et al. 2004). There could be at least two possible explanations for the apparent lack of LFF in our subjects. Since electrically evoked muscle contractile properties were not tested immediately post-marathon, it must be acknowledged that recovery in these properties may have occurred. In a study by Avela et al. (2001) the authors showed a significant recovery in torque after 30 min at a stimulation frequency of 20 Hz after long-lasting mechanically and electrically elicited fatigue. However such a scenario even if present, does not favor occurrence of LFF due to muscle damage, rather the fast recovery in low frequency torque is likely a result of clearance of metabolic by-products. In addition PTP have been suggested to compensate for LFF in fatigued muscles immediately after a work intervention (Rassier and McIntosh 2000; Sale 2002), thereby disguising LFF, however in this study no LFF was found two-day post-marathon indicating that muscle damage was not present in our subjects (Rassier and McIntosh 2000)—at least not to an extent that electrically evoked muscle contractile properties were affected. To our knowledge no studies on prolonged running have been able to demonstrate a relationship between contractile failure and muscle damage, and therefore it seems questionable that a direct link between muscle damage and contractile failure in prolonged SSC exercise should exist and that training status should play a role in the resistance to muscle fatigue.

Another possible mechanism involved in peripheral fatigue could be alterations in the conduction of the nerve signal between nerve ending and motor endplate conduction. In studies by Millet et al. (2002, 2003) altered electrically evoked M-Wave peak-to-peak amplitude in KE muscles after prolonged running was observed, indicating a decline in sarcolemma excitability. The design of this study did not allow us to test peak-to-peak amplitude muscle properties in our subjects.

The statistical analysis might have disguised differences in individual responses in contractile parameters after the marathon run, since we saw large differences among the subjects. To evaluate this inter individual response range we determined the relationship between Δ force at 50 Hz and ΔMVC, and found a strong correlation ($R = 0.79$, $P < 0.05$). Apparently some of the subjects experienced peripheral fatigue at the neuromuscular junction, which contributed to the large force loss in the KE MVC detected in those subjects. To our knowledge no other study has been able to determine such a relationship. Notably, the force loss at 50 Hz is considered a marker of attenuated sarcolemma excitability, which has been associated with depletion of Na^+ and accumulation of K^+ in the extracellular spaces impairing membrane depolarization especially at high frequencies (Strojnik and Komi 1998; Enoka and Stuart 1992).

Conclusion

In summary the present data indicate that the fatigue experienced by elite runners following a marathon race is of central rather than peripheral origin. Thus, no LFF was observed and voluntary KE MVC decreased without any

signs of worsening in intrinsic muscle properties. EE_{Mpace} was significantly elevated after completing a marathon, but was lowered 2 and 5 days after the marathon compared to pre-test values. Recovery pattern for KE and PF MVC showed different behavior as KE MVC recovered to pre-values after 2 days, while PF MVC needed 5 days to fully recover. Functional jumping power assessed in a CMJ remained depressed 5 days after completion of a marathon race indicating a prolonged depression of powerful mechanical muscle function.

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