



MATURATION-RELATED DIFFERENCES IN NEUROMUSCULAR FATIGUE AFTER A SHORT-TERM MAXIMAL RUN

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ABSTRACT

Purpose. This study investigated maturation-related differences in neuromuscular fatigue after a short-term maximal run.

Methods. Eight male children, eight adolescents, and eight adults performed a maximal ca. 50-s run (300/350/400 m, respectively). Mechanisms of neuromuscular fatigue were assessed through isometric plantar flexor tests, electrical stimulation of the posterior tibial nerve, soleus electromyography, and blood tests.

Results. All the groups showed a decrease in the running speed (children: $-12.2 \pm 6.5\%$; adolescents: $-9.8 \pm 5.1\%$; adults: $-12.2 \pm 3.1\%$), but only adults revealed a decline in the maximal isometric plantar flexor torque ($-16.1 \pm 13.0\%$). On the other hand, the relative pre- to post-fatigue change in the maximal isometric plantar flexor torque differed only between adults and adolescents. The peak torque in the passive twitch test decreased in adolescents ($-19.2 \pm 12.2\%$) and adults ($-23.7 \pm 13.7\%$). Moreover, post-fatigue minimum blood pH (children: 7.18 ± 0.03 ; adolescents: 7.14 ± 0.07 ; adults: 6.97 ± 0.06) differed between the groups. No changes were observed in the neural drive or mechanisms at the spinal level.

Conclusions. Despite the loss of running speed, children showed no post-exercise fatigue, whereas adolescents and adults demonstrated fatigue at peripheral sites. Central fatigue could not be established for the studied groups.

Key words: children, maturation, neuromuscular fatigue, high-intensity exercise

Introduction

Speed endurance sports, such as a 400-m run, are characterized by an explosive, nearly all-out, start and a subsequent struggle against fatigue due to the prolonged sprint distance. Because of the high power output, the main sources of energy involve the immediate adenosine triphosphate (ATP) and phosphocreatine (PCr) stores and anaerobic glycolysis, which is why the ability to cope with neuromuscular fatigue is a critical factor for the overall performance [1]. The loss of running speed during a competitive 400-m run, for instance, ranges from 13 to 23% [e.g. 2].

Neuromuscular (or muscle) fatigue can be defined as ‘any exercise-induced reduction in the ability of a muscle to generate force or power’ [3, p. 1733]. Fatigue can be caused by impairments in central and peripheral processes [3]. In the context of high-intensity running or jumping exercise, fatigue has mainly been attributed to the peripheral processes, including the excitation-con-

traction (E-C) coupling and cross-bridge cycling [4–7], while central factors seem to play a minor role [6, 7]. Unfortunately, studies of this type have so far focused mainly on grown-up subjects.

It has, however, been well-established that children experience a lower degree of fatigue [8] and need a shorter time to recover [9] than adults during high-intensity exercise. These differences could be explained by maturation-related development of force/power generation capacity [10], anaerobic capacity [11, 12], and glycolytic enzyme system [13]. Moreover, children also seem to benefit more from oxidative capacity as compared with adults [12, 14].

The aforementioned maturation-related differences in anaerobic performance compared with adults give a reason to expect that mechanisms underlying fatigue are also dependent on the stage of maturation. Owing to their lower anaerobic capacity, children’s performance is less disturbed by accumulation of metabolites, such as hydrogen (H^+) ions [15, 16]. This suggests that children

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develop less peripheral fatigue than adults.

The knowledge of how maturation affects the role of central processes under neuromuscular fatigue during dynamic high-intensity whole-body exercise remains very limited. Children have demonstrated a lower degree of central fatigue than adults during maximal isokinetic and isometric contractions [8, 17]. On the other hand, children showed a greater reduction in the level of voluntary activation, as well as a smaller decrease in peripheral force generation capacity during a 2-min maximal isometric knee extension effort [18]. Hence, the available results are controversial.

The purpose of the study is to investigate maturation-related differences concerning the relative contributions of central and peripheral factors underlying neuromuscular fatigue in a short-term maximal run. Our first hypothesis states that owing to their greater anaerobic capacity, neuromuscular fatigue is mainly caused by peripheral mechanisms in adults. Our second hypothesis states that in children central factors are involved as well.

Material and methods

Participants

The total of 24 male volunteers participated in the study. On the basis of their age, the subjects were divided into 3 groups (Table 1): children ($n = 8$; 11–13 years), adolescents ($n = 8$; 14–16 years), and adults ($n = 8$; 18–24 years). The adults were represented by good national level athletes in the 400-m flat and 400-m hurdles (400-m record: 49.54 ± 1.80 s), whereas the subjects in the 2 other groups included active, although not yet specialized young runners. All the subjects (or, if younger than 18 years, their caretakers) signed an informed consent and were aware of the protocol, benefits, and possible risks of the study. They were also advised of their rights to retire from the study at any time. The study was performed according to the Declaration of Helsinki, and was approved by the Ethical Committee of the local University.

Experimental protocol

The experiments were undertaken on a 200-m indoor track, during the period between the end of the

indoor season and the beginning of the outdoor season. The subjects were advised to rest or exercise lightly the day before the test and fast for 10 h before the first blood sampling in the morning of the test day. After the first blood sampling and light breakfast, electrodes were fixed, each subject was familiarized with the measurement equipment and protocol, and the ankle dynamometer was adjusted to the pre-determined joint angles.

The initial preparations were followed by recording of the recruitment curve of the H-reflex, the second resting blood sample, and a normal 60-min racing warm-up. One maximal sprint (80–100 m) was performed by the subjects after the 45-min warm-up and 15 min before the test run.

After the warm-up session, pre-fatigue neuromuscular tests were performed in the ankle dynamometer. The third blood sample was taken 2–4 min before the maximal run started. Children, adolescents, and adults ran all-out 300-, 350-, and 400-m sprints, respectively (Table 1). The runners were advised to pace the running for optimal time over the whole run. Group-specific distances were used in order to equalize the duration of the run. Intermediate times were recorded at 100-, 200-, and 300-m splits for children and adults, and after 50-, 150-, and 250-m splits for adolescents.

The post-fatigue neuromuscular tests were performed 6–12 minutes after the run. It took several minutes to walk from track to the laboratory and make the preparation for the next measurements. The test set was identical with the pre-fatigue one, except that two additional maximal M-waves were elicited immediately at the beginning of the tests. The post-fatigue blood samples were taken 5, 6, 11, 14, 30, and 60 min after the run. A schematic illustration of the protocol is presented in Figure 1.

Equipment

Ankle dynamometer

The plantar flexor torque was measured from the left leg with a custom-made ankle dynamometer (University of Jyväskylä, Finland). The subjects were firmly attached to the seat (joint angles: ankle 90° , knee 180° , and hip 120°) by belts at the shoulders and thighs, and the left foot was firmly attached to the pedal of the force transducer. The torque signal was amplified ($\times 100$, Kis-

Table 1. Subject characteristics and running performance

Group	<i>n</i>	Age (years)	Height (m)	Weight (kg)	Distance (m)	Time (s)	Speed (m/s)
Children	8	11.9 ± 1.4	1.61 ± 0.10	47.9 ± 7.1	300	53.6 ± 5.7	5.65 ± 0.54
Adolescents	8	14.9 ± 1.1	1.80 ± 0.03	63.9 ± 4.2	350	53.5 ± 2.3	6.57 ± 0.27
Adults	8	21.3 ± 3.3	1.82 ± 0.07	73.2 ± 7.0	400	52.1 ± 2.1	7.68 ± 0.30

Data presented as means \pm SD. There were significant ($p < 0.001$) differences between the groups in all variables except running time.

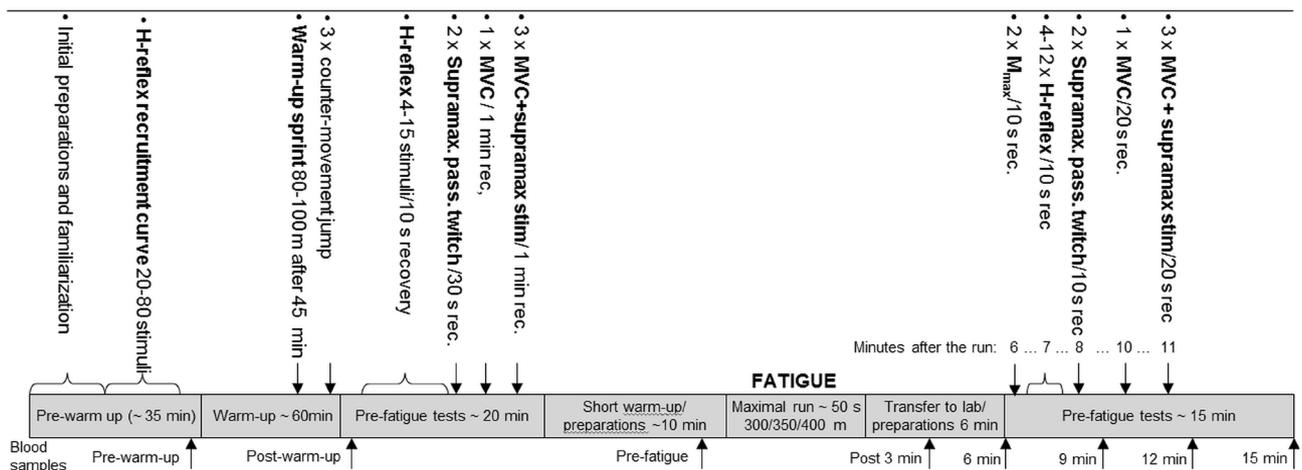


Figure 1. Test protocol

tlar Charge Amplifier, type 5011), supplied to the A/D-converter (Power1401, CED Ltd., Cambridge, UK), and analysed with the Spike 5.14 software (CED Ltd., Cambridge, UK).

Electromyography

Self-made bipolar circular surface electromyography (EMG) electrodes (Ag-AgCl, interelectrode distance: 2 cm) were attached over the soleus and medial gastrocnemius following the SENIAM recommendations [19]. The ground electrode was placed over the tibia. All the skin-electrode contact sites were prepared with the use of common skin preparation methods (shaving, abrasion with sandpaper, cleaning with alcohol) so that the impedance was below 10 k Ω . The electrode fixations were ensured by taping and strapping with gauze. The data were recorded and filtered (sampling rate: 1500 Hz; bandwidth: 10–500 Hz; gain \times 2) with a wireless EMG transmitter/receiver system (Noraxon 2400T/R, Scottsdale, USA). The surface EMG electrodes were connected to the transmitter device through pre-amplified leads (gain \times 500). The amplified and filtered signals were digitized with the A/D converter (Power1401, CED Ltd., Cambridge, UK) and analysed with the Spike 5.14 software (CED Ltd., Cambridge, UK).

Electrical stimulations

The posterior tibial nerve was stimulated electrically with single rectangular (200 μ s) pulses (Digitimer DS7[®], Hertfordshire, UK). A self-adhesive cathode (1.5 \times 1.5 cm, Unomedical Ltd., UK) was placed and fixed in the popliteal fossa. The anode, a large oval-shaped (5 \times 8 cm) self-adhesive electrode (Mettler Electronics Corp. Anaheim, CA, USA), was placed over the upper edge of the patella tendon. The placement of the fixed cathode was determined in a standing position with the use of

a hand-held cathode electrode. The site that produced the greatest M-wave response at the soleus was marked to the skin and the stimulation electrode was fixed to this place by tape and gauze.

Blood lactate and blood pH

The blood lactate concentration was analysed from a fingertip blood sample (20 μ l) with a Biosen C-Sport analyser (EKF Industrie, Elektronik GmbH, Barleben, Germany). The level of blood pH was determined from a whole blood sample (200 μ l) collected from an antecubital vein (GEM Premier 3000, Instrumentation Laboratory, Lexington, MA, USA).

Measurements

H_{max}/M_{max} ratio

Before the warm-up session, the recruitment curve of the H-reflex was recorded while the subject was sitting in a relaxed position in the ankle dynamometer bench, with the target leg in an extended position. The stimulation intensity was increased from the initial 5–10 mA in 1–5 mA steps (pulse duration: 200 μ s; stimulus interval: 10 s) until the H-reflex disappeared. Thereafter, the intensity was further increased in 10 mA steps until the increase in the M-wave levelled off. The H_{max}/M_{max} ratio was determined from the recruitment curve [20].

Maximal voluntary contraction

For the assessment of the overall degree of fatigue, the subjects performed four plantar flexor maximal voluntary contraction (MVC) efforts (2–3-s) with 60-s (pre-fatigue test) and 20-s (post-fatigue test) rest periods. The shorter rest periods in the post-fatigue test were

used in order to minimize the time to recover from the sprint. The effort that produced the greatest torque was selected for further analyses. The subjects were verbally instructed and encouraged to produce an all-out effort. In order to analyse the V/M_{\max} ratio, supramaximal stimuli (at 150% intensity of the maximal M_{\max} response) were delivered to the tibial nerve over the last three MVC efforts [21].

EMG activity

The extent of maximal EMG activity (aEMG) was recorded from soleus during the four MVC efforts (see: *Maximal voluntary contraction*). The greatest torque response was analysed. The aEMG was obtained by rectifying and averaging the EMG signal over a 200-ms period, starting from the onset of the torque plateau. In addition, the normalized values of aEMG/ M_{\max} and aEMG/MVC ratio [22] were analysed.

Passive twitch

Two passive twitch responses were produced and recorded from the relaxed calf muscles in the same sitting position by delivering supramaximal single stimuli (rectangular pulse: 200 μ s; 150 and 180% of the intensity required to attain the maximal M_{\max} response) to the tibial nerve with 30-s (pre-fatigue test) and 10-s (post-fatigue test) rest periods [23]. Peak torque (PT), contraction time (CT), and half-relaxation time (HRT) were analysed from the greatest of the two torque outputs. Moreover, the maximum rates of torque development (MRTD) and relaxation (MRTR), as well as the average rates of torque development (ARTD) and relaxation (ARTR) were analysed as averages of the two trials.

M_{\max}

The maximal peak-to-peak amplitude of the M-wave response was analysed for the soleus. The pre-fatigue values were determined from the H-reflex recruitment curve data. The post-fatigue value was expressed as the mean of the two supramaximal stimuli delivered at 10-s intervals immediately at the beginning of the post-fatigue tests (6 min after the run). The stimulus intensity was 150 (1st stimulus) and 180% (2nd stimulus) of the M_{\max} intensity.

H_{20}

The H-reflex response was recorded from the soleus during the pre- and post-fatigue tests while the subject was positioned in the ankle dynamometer bench [21]. In order to eliminate peripheral effects, the stimulus intensity was continuously monitored and manu-

ally adjusted so that the peak-to-peak amplitude of the concurrent M-wave response remained within the limits of 10–30% of the peak-to-peak M_{\max} . The stimulus was triggered manually every 10th second until 4 valid responses were recorded, except in the post-fatigue test, in which no more than 12 attempts were allowed. Since no significant differences were observed in the peak-to-peak amplitude of the control M-wave responses between the groups in the pre- (children: $19.0 \pm 4.1\%$ of the peak-to-peak M_{\max} ; adolescents: $18.8 \pm 4.2\%$; adults: $18.7 \pm 5.3\%$) or post-tests (children: $20.7 \pm 6.7\%$; adolescents: $21.9 \pm 6.1\%$; adults: $17.7 \pm 5.1\%$), the H-reflex responses were considered comparable. The post-fatigue test failed with one child and one adult. The sample mean of 4 successful responses was analysed for each subject.

V/M_{\max}

The magnitude of the efferent motoneuronal output during voluntary muscle activation was assessed from the EMG responses evoked by superimposed twitches (150% of the intensity required to attain the maximal M_{\max} response) during the three MVC efforts. In order to eliminate the effects of peripheral changes, the peak-to-peak amplitude of V-wave was normalized to M_{\max} [21]. The mean of the three trials were calculated.

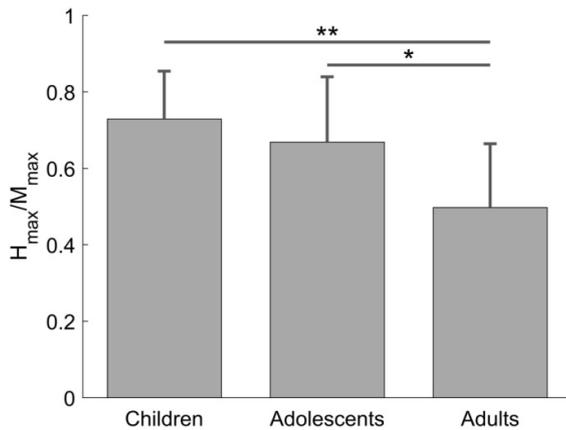
Statistical analyses

All the statistical analyses were performed with the IBM SPSS Statistics software (version 19). The results are presented group-wise as sample means and standard deviations. Differences between the pre- and post-fatigue measurements were analysed with the paired-samples *t*-tests. The between-group differences in the relative changes between the pre- and post-values were analysed with the one-way ANOVA method, the least significant difference post-hoc analysis. The normality and homogeneity of the distributions were checked with the Shapiro-Wilk and the Levene's tests, respectively. In the case of non-normality, independent samples Kruskal-Wallis and repeated samples Wilcoxon sign-rank tests were applied. Linear relationships between the metabolic variables and the relative changes in the neuromuscular measurements were analysed with the Pearson product moment correlation coefficients. The significance level was at $p < 0.05$.

Results

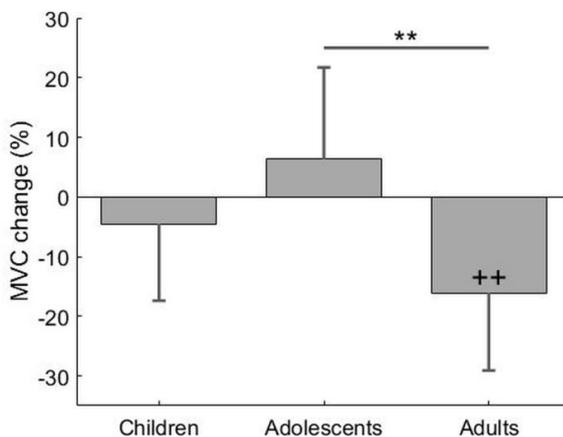
Running performance

The running times were similar in the groups (Table 1). The average running speed was 16.9% and 35.9% higher ($p < 0.001$) in adults compared with adolescents and



* $p < 0.05$, ** $p < 0.01$

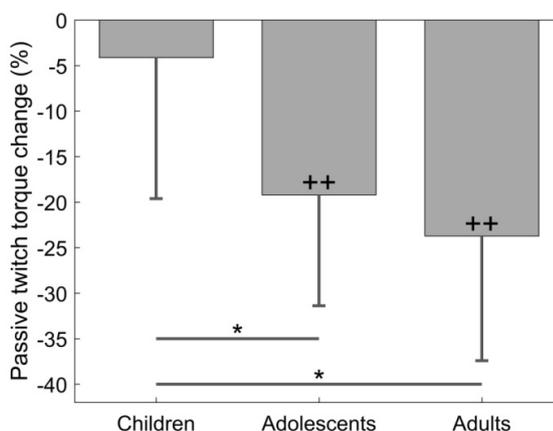
Figure 2. H_{\max}/M_{\max} ratio measured before the run



** $p < 0.01$ (significant difference between groups),

** $p < 0.01$ (significant difference between pre- and post-fatigue values)

Figure 3. The relative change in maximal voluntary contraction (MVC) between pre- and post-fatigue measurements



* $p < 0.05$ (significant difference between groups)

** $p < 0.01$ (significant difference between pre- and post-fatigue values)

Figure 4. Relative changes in passive twitch torque between pre- and post-fatigue measurements

children, respectively, and 16.3% higher in adolescents compared with children ($p < 0.001$). The running speed decreased from the fastest 100-m split to the last 100-m split by $12.2 \pm 6.5\%$ ($p < 0.01$), $9.8 \pm 5.1\%$ ($p < 0.001$), and $12.2 \pm 3.1\%$ ($p < 0.001$) in children, adolescents, and adults, respectively.

H_{\max}/M_{\max} ratio

Children and adolescents demonstrated a 46.6% ($p < 0.01$) and 34.3% ($p < 0.05$), respectively, higher H_{\max}/M_{\max} ratio compared with adults (Figure 2).

MVC

The MVC torque decreased only in adults ($-16.1 \pm 13.0\%$; $p < 0.01$) (Figure 3) and their change was greater ($p < 0.01$) than that in adolescents.

Passive twitch characteristics

Passive twitch decreased by $19.2 \pm 12.2\%$ and $23.7 \pm 13.7\%$ ($p < 0.01$) in adolescents and adults, respectively (Figure 4). These changes were significantly greater compared with children ($p < 0.05$). All the groups showed equal decreases in CT and HRT (Table 2). MRTD decreased in adults ($-23.5 \pm 23.7\%$; $p < 0.05$) and adolescents ($-34.4 \pm 30.1\%$; $p < 0.05$) and both these changes differed from that in children ($p < 0.05$ and $p < 0.01$, respectively). Although MRTR did not change significantly in any of the groups, there was a significant difference ($p < 0.05$) between children ($+10.8 \pm 25.74\%$; NS) and adults ($-16.4 \pm 23.1\%$; NS). ARTD decreased only in adults ($-15.2 \pm 16.6\%$; $p < 0.05$) and the relative change was greater than in children ($p < 0.05$), who showed a non-significant increase in ARTD ($6.4 \pm 19.5\%$; NS).

aEMG, aEMG/ M_{\max} , aEMG/MVC ratio, H_{20} , M_{\max} , V/M_{\max}

In these parameters, there were neither within-group differences between pre- and post-fatigue measurements nor between-group differences in the relative changes (Table 2).

Blood metabolites

The blood pH decreased after the run from the resting values to 7.18 ± 0.03 , 7.14 ± 0.07 , and 6.97 ± 0.06 ($p < 0.001$ for all changes) in children, adolescents, and adults, respectively (Figure 5). The post-fatigue minimum blood pH differed between adults and children ($p < 0.001$), as well as between adults and adolescents ($p < 0.001$).

The peak of blood lactate after the run reached 10.2 ± 1.1 , 13.3 ± 3.7 , and 17.4 ± 1.8 mmol/l in children, adolescents, and adults, respectively ($p < 0.05$ between the groups).

Table 2. Pre- and post-fatigue measurements and relative differences

	Group	Pre	Post	Difference (%)
M_{max} (mV)	children	3.95 ± 1.52	3.56 ± 1.33	-3.4 ± 13.0
	adolescents	3.42 ± 1.10	3.22 ± 1.01	-5.0 ± 12.0
	adults	5.57 ± 1.93	4.78 ± 1.01	-10.7 ± 13.5
H_{20} (mV)	children	0.51 ± 0.13	0.60 ± 0.22	+20.7 ± 9.1
	adolescents	0.39 ± 0.21	0.42 ± 0.24	+8.8 ± 36.5
	adults	0.37 ± 0.20	0.36 ± 0.11	+5.7 ± 35.3
V/M_{max}	children	0.18 ± 0.10	0.28 ± 0.13	+131.7 ± 207.6
	adolescents	0.23 ± 0.17	0.26 ± 0.12	+57.5 ± 131.9
	adults	0.19 ± 0.11	0.24 ± 0.31	+9.6 ± 63.5
aEMG (µV)	children	64 ± 22	65 ± 21	+5.4 ± 29.0
	adolescents	62 ± 33	73 ± 38	+19.1 ± 38.6
	adults	83 ± 32	74 ± 20	+1.0 ± 10.4
aEMG/ M_{max} (10^{-3})	children	17 ± 5	18 ± 7	+4.3 ± 23.1
	adolescents	21 ± 13	25 ± 22	+19.7 ± 46.1
	adults	19 ± 7	19 ± 10	+0.3 ± 21.1
aEMG/MVC (10^{-4})	children	3.03 ± 1.16	3.24 ± 1.13	+8.9 ± 16.0
	adolescents	1.88 ± 0.62	2.10 ± 0.83	+9.9 ± 17.7
	adults	2.00 ± 0.77	2.10 ± 0.68	+19.1 ± 22.1
CT (ms)	children	106 ± 9	95 ± 6**	-9.4 ± 5.8
	adolescents	110 ± 8	99 ± 9**	-9.4 ± 7.4
	adults	105 ± 4	95 ± 3***	-9.8 ± 3.4
HRT (ms)	children	68 ± 8	58 ± 6**	-13.2 ± 8.4
	adolescents	66 ± 7	58 ± 6**	-12.3 ± 5.9
	adults	71 ± 9	62 ± 5*	-11.3 ± 9.3

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ (significant difference from the pre-value)

M_{max} – the maximum peak-to-peak amplitude of the M-wave, H_{20} – the peak-to-peak amplitude of the H-reflex response produced at the intensity corresponding to 10–30% of M_{max} , V/M_{max} – the peak-to-peak to amplitude of V-wave normalized to peak-to-peak amplitude of M_{max} , aEMG – average of the rectified amplitude of maximal EMG response, aEMG/ M_{max} – average of the rectified amplitude of maximal EMG response normalized to the peak-to-peak amplitude of maximal M-wave, aEMG/MVC – average of the rectified amplitude of maximal EMG response normalized to the MVC torque, CT – contraction time of the passive twitch torque, HRT – half-relaxation time of the passive twitch torque

Data presented as means ± SD

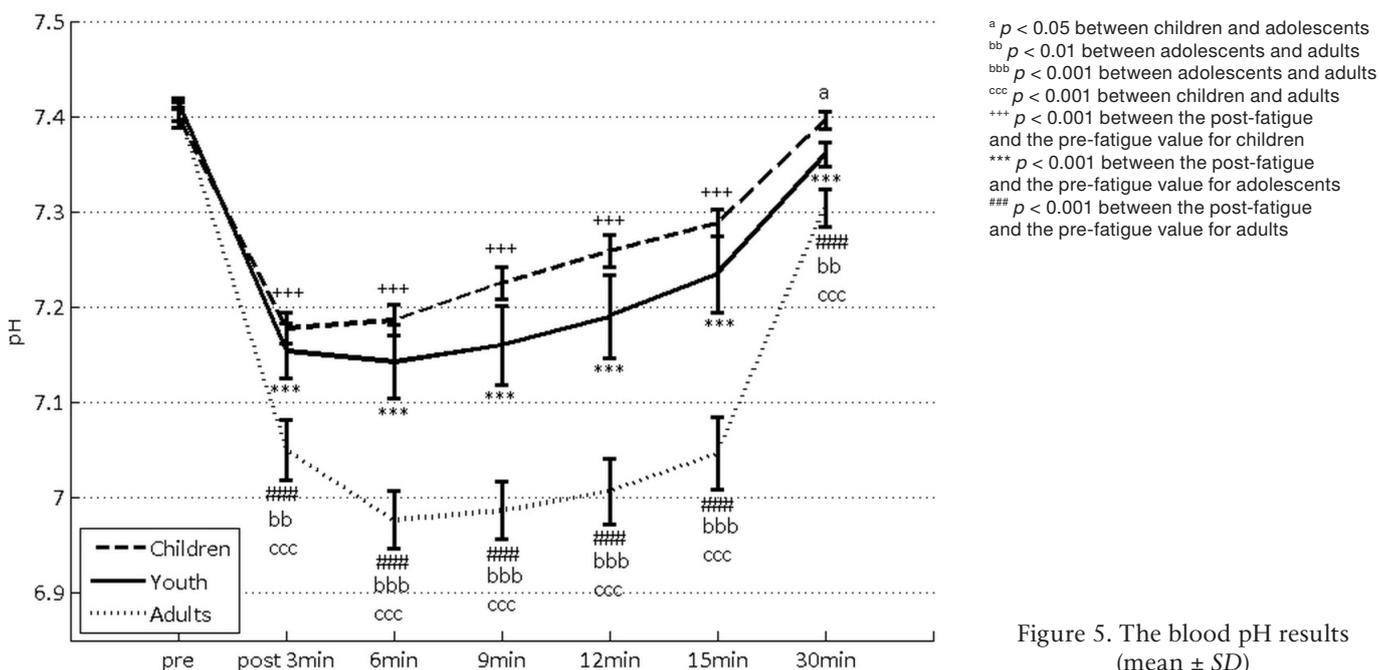


Figure 5. The blood pH results (mean ± SD)

Discussion

The purpose of the study was to investigate maturation-related differences concerning the relative contributions of central and peripheral factors underlying neuromuscular fatigue in a short-term maximal run.

Main findings

The main finding was that regardless of the stage of maturation the predominant site of muscle fatigue after the maximal ca. 50-s sprint was peripheral. We did not observe any neural impairment after the run.

The loss of running speed, -12.2% , -9.8% , and -12.2% in children, adolescents, and adults, respectively, was comparable with previous studies [2, 24]. The speed deceleration and the post-exercise blood lactate and blood pH levels indicate that the subjects made an all-out effort. In line with previous studies [6, 7], adults showed a decrease in MVC torque (-16.1%) after the run. Children and adolescents presented no change in MVC despite the loss of running speed, which might be due to faster recovery before the post-fatigue tests.

Peripheral fatigue

Previous studies addressing neuromuscular fatigue in adults have consistently shown significant reductions (from -35 to -8%) in the plantar flexor / knee extensor PT after short-term high-intensity running efforts [4–7]. Accordingly, both adolescents and adults presented peripheral fatigue in this study. Interestingly, even though the MVC torque was unchanged in the adolescents, they still demonstrated an almost equal decrease (-19.2%) with adults (-23.7%) in PT. This somewhat conflicting finding might be explained by the additional 2-min delay between the passive twitch and the MVC tests, which may have allowed the recovery of peripheral processes among adolescents. These findings concerning peripheral fatigue raise two questions: (1) Which are the underlying mechanisms causing the peripheral fatigue in adolescents and adults? (2) Why did children show no peripheral fatigue despite the significant loss of running speed?

Both the present and the previous studies [4, 7] suggest that the M-wave remains unchanged after a short-term maximal run. Although depressed M-waves have been observed after very short repeated sprint (12×40 m) [6] and maximal jumping exercise [22], it may be that the neuromuscular junction and the sarcolemma retain their functionality during continuous anaerobic long-sprint distances.

Thus, in line with the recent study by Tomazin et al. [7], the peripheral fatigue could have been caused by the processes beyond the sarcolemma. Consistently with earlier studies [4, 6], both adolescents and adults showed a decrease in MRTD that is limited by the rate of cross-

bridge transition from the weakly to strongly bound state [25]. The inhibition of the cross-bridge system has been associated with increased levels of metabolic by-products, such as H^+ , inorganic phosphate (P_i), and adenosine diphosphate (ADP) [25, 26].

An increased H^+ concentration has been classically considered as the major cause of fatigue [e.g. 25]. The reduced level of the post-fatigue blood pH is a consequence of extensive regeneration of ATP through anaerobic glycolysis during intensive exercise lasting 30–120 s [1]. This may partly explain the impairments observed at the level of cross-bridges [23], even though the overall effects of H^+ on muscle performance may have previously been over-estimated [26]. It has also been suggested that instead of directly inhibiting contractile mechanics, low pH rather limits the performance by disturbing metabolic processes [27], e.g. through glycolytic enzymes [25]. Since the muscle power output recovers from maximal 30-s exercise while muscle pH still remains at a low level [28], there must be also other factors explaining peripheral fatigue during high-intensity exercise [29]. More recent studies indicate that other metabolites, such as P_i , have a significant contribution to fatigue at the level of the contractile system [26]. However, the relative contributions of various metabolites remain debatable [25, 26] and more definite conclusions on their roles are not possible on the basis of the methodology applied in this study.

Interestingly, children experienced a loss of running speed, even though they did not show any sign of neuromuscular fatigue during the post-fatigue tests. This suggests that the 6-min delay before the post-fatigue tests was long enough for the complete restoration of their peripheral muscle performance. The higher and earlier peak of blood pH indicates that metabolic stress was milder at the end of the run in children compared with adolescents and adults. Consequently, children may have been less fatigued owing to their more immature anaerobic characteristics and the lower power generation capacity compared with the other two groups [11, 15]. The low anaerobic capacity in childhood has been hypothetically associated, for instance, with the dimensional growth of glycolytic fast twitch fibres and testosterone levels [15, 30].

On the other hand, the lower the accumulation of H^+ ions at the end of exercise, the shorter the time to restore the level of muscle PCr [31], which is an important factor for the recovery of power output after maximal exercise [28]. It has been shown that children are capable of resynthesizing PCr even twice faster than adults [14], which suggest that children may not only experience less fatigue, but also recover faster.

In the presented study, we also observed that the H_{max}/M_{max} ratio was significantly higher in children than in adolescents and adults. Assuming that the bigger H_{max}/M_{max} ratio is associated with the greater percentage of low-threshold motor units [32], the observed post-

fatigue differences in the metabolic and neuromuscular characteristics between the studied groups could be explained to some extent by the maturation-related decrease in the relative proportion of more aerobic low-threshold muscle fibres [33]. Overall, the present results support the previous outcomes that because of their lower anaerobic capacity children are less capable of pushing their neuromuscular system to the same degree as more matured athletes [34]. This appears as smaller changes in peripheral factors after maximal running exercise. On the other hand, children experienced a loss of running speed comparable with adults and adolescents during the maximal run and, hence, the faster rate of recovery after the maximal exercise may also contribute to the observed differences in the peripheral factors.

Central fatigue

Neural impairments, i.e. central fatigue, could not be observed in any of the studied group. The V-wave, the H-reflex, and the maximal aEMG activity in the soleus remained unchanged. Since the muscle torque output fluctuated to some extent during the MVC efforts, the V-waves should be considered with some caution.

The present findings are in good agreement with the previous outcomes that have mainly shown minor changes in neural factors after high-intensity running and jumping exercise [4–7, 22]. Overall, the identification of exercise-induced neural changes is challenging because the neural processes require only a few minutes to recover from maximal or prolonged submaximal contractions [35, 36]. The common 2–8-min delay in a running exercise protocol may therefore be too long for revealing neural changes. On the other hand, Tomazin et al. [7] interestingly observed a sign of delayed central fatigue 5 min after a maximal 400-m run. The authors hypothesized that slow post-exercise accumulation of metabolites, e.g., NH_3 and H^+ [28], may lead to central fatigue within the brain as the metabolites permeate the blood-brain barrier and perturb neurotransmitter metabolism within the brain [37]. Nevertheless, this study does not provide further evidence for this hypothesis.

Conclusion

Even though the used methodology does not allow unconditional exclusion of central fatigue, the present study indicates that peripheral mechanisms are the predominant cause of post-exercise muscle fatigue in a short-term maximal run. The results are also in good agreement with previous studies on adults [38] and, moreover, suggest that the relative contributions of central and peripheral mechanisms do not significantly differ between the groups representing various stages of maturation. Probably neural adjustments, particularly group

III and IV afferent inhibition and disfacilitation of spindle support, are more inherent in prolonged running exercise upwards of 30 min [39] or exhaustive jumping exercise [40] than short-term sprinting. From the practical point of view, the results emphasize running speed and aerobic training in children and adolescents as a good basis for the adult phase. A more thorough investigation of central fatigue during continuous short-term maximal running requires that measurements are carried out in an online fashion or immediately after the run.

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