Different Metabolic Responses during Incremental Exercise Assessed by Localized ^{31}P MRS in Sprint and Endurance Athletes and Untrained Individuals

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Key words
- ^{31}P MRS
- CSI
- PCr recovery
- muscle function
- exercise training

Abstract

Until recently, assessment of muscle metabolism was only possible by invasive sampling. ^{31}P magnetic resonance spectroscopy (^{31}P MRS) offers a way to study muscle metabolism non-invasively. The aim of the present study was to use spatially-resolved ^{31}P MRS to assess the metabolism of the quadriceps muscle in sprint-trained, endurance-trained and untrained individuals during exercise and recovery. 5 sprint-trained (STA), 5 endurance-trained (ETA) and 7 untrained individuals (UTI) completed one unlocalized ^{31}P MRS session to measure phosphocreatine (PCr) recovery, and a second session in which spatially-resolved ^{31}P MR spectra were obtained. PCr recovery time constant (τ) was significantly longer in STA (50 ± 17 s) and UTI (41 ± 9 s) than in ETA (30 ± 4 s), ($P < 0.05$). PCr changes during exercise differed between the groups, but were uniform across the different components of the quadriceps within each group. pH during recovery was higher for the ETA than for the UTI ($P < 0.05$) and also higher than for the STA ($P < 0.01$). Muscle volume was greater in STA than in UTI ($P < 0.05$) but not different from ETA. Dynamic ^{31}P MRS revealed considerable differences among endurance and sprint athletes and untrained people. This non-invasive method offers a way to quantify differences between individual muscles and muscle components in athletes compared to untrained individuals.

Introduction

Skeletal muscle shows remarkable plasticity in adapting to different exercise training regimes. It is well known that athletes with a background of endurance training have better oxidative metabolism than sprint-trained athletes [34], resulting from cardiovascular adaptations, enhanced capillarity and increased mitochondrial density [25] as a result of training-specific adaptations to high-volume load. In sprint athletes, at the other end of the continuum, increased force-production capacity and buffering capacity support high power output and running speed [38]. According to their demand, muscles of untrained individuals seem to reside between those extremes [46].

It has long been a goal of sports science to characterize performance-related characteristics of single muscles, but until relatively recently this required ex vivo measurements on biopsy specimens. Classification of muscle fibres depends on the technique used. Histochemical analysis of biopsy specimens has been widely employed to characterize fibres into type I and type II according to their ATPase activity. ^{31}P nuclear magnetic resonance spectroscopy (^{31}P MRS) has been used to evaluate metabolic properties of skeletal muscle non-invasively. It is well established that ^{31}P MRS measurements of post-exercise phosphocreatine (PCr) recovery (a purely oxidative process) can be used as a measure of muscle mitochondrial oxidative capacity, which correlates well with various in vitro measurements of mitochondrial numbers and function [15,20,26,30].

Dynamic ^{31}P MRS measurements obtained during exercise and recovery using an MR-compatible ergometer have been used to evaluate progress in training [23] and identify differences between differently specialised athletes [28]. Differences between sprinters and long-distance runners have also been detected in PCr/ATP and PCr/Pi ratios in resting muscle, which are thought to reflect different fibre-type composition [2]. However, human muscle shows considerable variation in biochemical properties, perfusion, and fibre type composition [19,32]. Most human biopsy data, especially in trained individuals, comes from vastus lateralis or rectus femoris,

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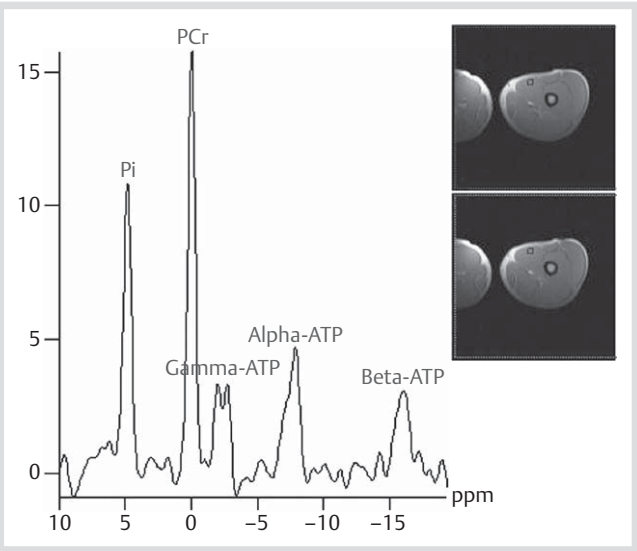

Fig. 1 Localized dynamic ^{31}P spectra of the medialis muscle during exercise.

and much of what is known about other muscles comes from autopsy material [9,16]. There is some evidence that fibre-type distribution among different parts of the quadriceps is uniform in untrained subjects [12], at least in vastus lateralis and rectus femoris. The well-known shifts towards type I fibres with endurance training and type II with strength/speed training have been mostly studied in vastus lateralis [36,44]. It is not clear, though, whether heterogeneity of skeletal muscle structure, composition, and capillarity results in local differences in metabolic function between different muscle regions in athletes or untrained subjects. ^{31}P MRS can differentiate between the athletes based on their skeletal muscle properties. Whether or not the other muscle groups of the quadriceps follow the same pattern remains unknown. Although findings of differences in PCr recovery kinetics between sprinters and endurance athletes are not new [18], results are often based on measurements of the bulk of the muscle. We wanted to investigate this in the different component muscles of the quadriceps and also incorporate a group of untrained individuals. In this respect we hypothesised that the metabolic situation in the main locomotor muscle and its component muscles is homogenous within a specific group, but will be different and highly dependent on activity level and training status.

We therefore studied aerobic performance in the muscle of untrained individuals and athletes specializing in the 2 extremes of sprint and long-distance events. Using unlocalized ^{31}P MRS, we took (i) the time constant (τ) of PCr resynthesis after submaximal exercise in the quadriceps as an inverse measure of whole-muscle oxidative capacity. We also used ^{31}P chemical shift imaging (CSI) to assess (ii) spatial metabolic variation in different compartments of the quadriceps muscle. The results throw light on the spatial homogeneity of adaptive changes in muscle of a variously active population.

Materials and Methods

We studied 7 male untrained individuals and 10 male athletes with 7±2 years (mean±SD) of national or international competitive experience, who had undergone specific training programs: the sprint-trained group (STA: n=5, age 23±2 years, height 179±6 cm, weight 77±7 kg) were sprinters of national and international level with a competitive distance of 100–400 m, while the endurance-trained group (ETA: n=5, age 27±9 years, height 176±4 cm, weight 65±6 kg) were endurance athletes of national level, comprising 4 runners with competitive distances 3 000–5 000 m and 1 road cyclist. The untrained individuals (UTI: n=7, age 30±5 years, height 179±6 cm, weight 77±7 kg) did not engage in any regular physical activity or sport (less than 2 h activity per week) and were therefore classified as sedentary.

The present study meets the ethical standards of the journal [14] and was approved by the ethics committee of Innsbruck Medical Universtiy (AN3433 271/4.12). After giving written informed consent, subjects performed MR measurements of the quadriceps muscle at the Department of Radiology, Innsbruck Medical University in a 1.5 T whole-body MR scanner (Magnetom Avanto, Siemens, Erlangen, Germany) using a circular surface coil (Siemens, Erlangen, Germany) double-tuned to ^{1}H at 63.5 MHz and ^{31}P at 25.8 MHz. Subjects lay prone in the bore, strapped to the table over upper legs, buttocks and lower back to avoid movement artefacts during measurement. With their non-dominant leg (defined as the leg opposite to that preferred in jumping activities) they performed repetitive dynamic single-leg extensions on an MR-compatible ergometer using a pedal connected to a valve-adjustable air pressure system, controlled by feedback of current power output (Quadspect, Ergospect GmbH, Austria). ^{31}P MRS exercise studies were carried out after at least 24 h activity break. Subjects attended twice, to first perform an incremental and then a submaximal exercise protocol. In both cases, subjects performed metronome-timed full knee extensions at 0.5 Hz. The incremental exercise protocol started at 4.5 W, increasing by 1.5 W every 2 min until exhaustion. After completion, PCr recovery was monitored for 4 min. Unlocalized ^{31}P free induction decay (FID) sequences (no localization sequence) were acquired throughout 10-scan groups with repetition time (TR) 1 000 ms, giving 10 s time resolution. Subsequently a T1-weighted spin-echo sequence (TR=550 ms, TE=12 ms) was performed which covered both upper legs with 40 slices, with a 320 mm field of view, 10 mm slice thickness with 2 mm interslice gap and a 320×320 matrix. Muscle volume for each slice was obtained by multiplying measured planimetric areas by the interslice distance, summed for all the slices, and converted to total quadriceps muscle mass assuming a density of 1.056 g/ml. In the second session, subjects performed submaximal exercise at 80% of the maximal power output from the first session. Spatially-resolved spectra were acquired every 2 min from the portion of quadriceps that was covered by the coil as determined by a localizer matched to the first session. After 2 min rest (baseline measurement) the protocol consisted of 2 exercise increments (E1 and E2) and 2 measurements during recovery (R1 and R2). A chemical shift imaging (CSI) spin echo sequence was used, with TR=790 ms, TE=2.3 ms, field of view 200×200 mm^2 with weighted phase encoding (8×8 phase-encoding steps) and a slice thickness of 80 mm, giving a time resolution of 2 min with voxel size 50 cm^3. PCr signal to noise ratio values for the different muscle groups were ca. 15 (7.5/0.5 for the medials muscle at baseline, see **Fig. 1**).

Spectral data were processed with the commercial software package LUISE (Siemens, Erlangen, Germany). Baseline correction was performed semi-automatically by setting the peak ranges of inorganic phosphate (Pi) and PCr as references, and

phase correction was applied semi-automatically using 12 iterations with Pi and PCr as reference peaks; supplementary manual phase- or baseline correction was performed if necessary. The peak areas and peak positions of PCr and Pi were fitted in the frequency domain. Concentrations of Pi and PCr during rest were estimated from signal ratios to ATP, corrected for NOE and T1 saturation effects [3,20]. Intracellular concentrations of PCr and Pi were calculated relative to β-ATP, assuming a resting β-ATP concentration of 8.2 mmol.L^{-1} and corrected for magnetic saturation [21]. For kinetic analysis, PCr changes were expressed relative to baseline values, and PCr recovery was fitted to a monoexponential curve to determine τ. Intracellular pH was calculated from the chemical shift of Pi relative to PCr (δ) as pH = 6.75 + log(δ–3.27)/(5.69–δ) [33].

Statistical analysis was carried out using R software, version 2.15.1 [37]. Descriptive statistics are given as mean and standard deviation. To answer the first question, whether the PCr recovery time would be different between the 3 groups, we applied one-way analysis of variance (ANOVA). For the second set of questions, whether the 3 groups of athletes/individuals and whether the component of the quadriceps responded differently to the exercises, we applied two-way ANOVAs (between and within design) separately for each component muscle and separately for each group, respectively. The significance level was set to 0.05. Post-hoc analyses were performed using pairwise t-tests without correction for multiple testing due to the exploratory character of the study.

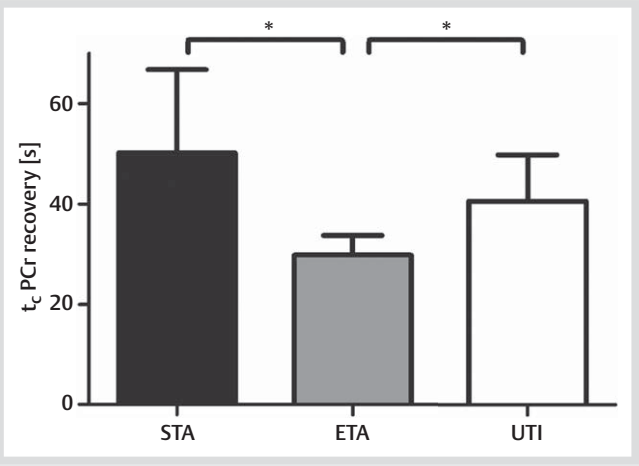

Fig. 2 Mean ± SD of PCr recovery time derived from unlocalised spectra of the quadriceps for STA (black), ETA (grey) and UTI (white). * denotes significant differences between the groups at the 0.05 level.

Results

PCr recovery τ of the 3 training groups was significantly different (ANOVA; $P<0.05$). It was shortest in the ETA (30 ± 4 s) and longest in STA (50 ± 17 s), while the UTI (41 ± 9 s) was in the middle. Posthoc comparisons revealed that the values of the STA and the UTI differed significantly ($P<0.05$) from the ETA with respect to a slower recovery in these groups (**Fig. 2**).

This is consistent with the spatially-resolved data, which show an average decline of the component muscles during E1 and E2 in the ETA of 60 and 58 % compared to the UTI (38 and 24 %) and STA (55 and 35 %) from baseline, respectively (**Fig. 3**). During R1 and R2, PCr recovered fastest and almost completely to 92 and 99 % in the ETA compared to 68 and 82 % in the UTI and slowest to only 49 and 76 % in the STA, respectively. There was no significant difference in PCr depletion between the different component muscles within each group (right panel of **Fig. 3**). **Fig. 3** depicts the change of PCr for each training group separately for each compartment of the quadriceps. Details about means and standard errors are given in **Table 1**.

Analysis of variance showed highly significant time and interaction effects (ANOVA; $P<0.01$) for each component muscle. The group effect was highly significant in intermedius and significant in lateralis and medialis, respectively (ANOVA; $P<0.01$ and $P<0.05$). However, this effect could not be found in the rectus.

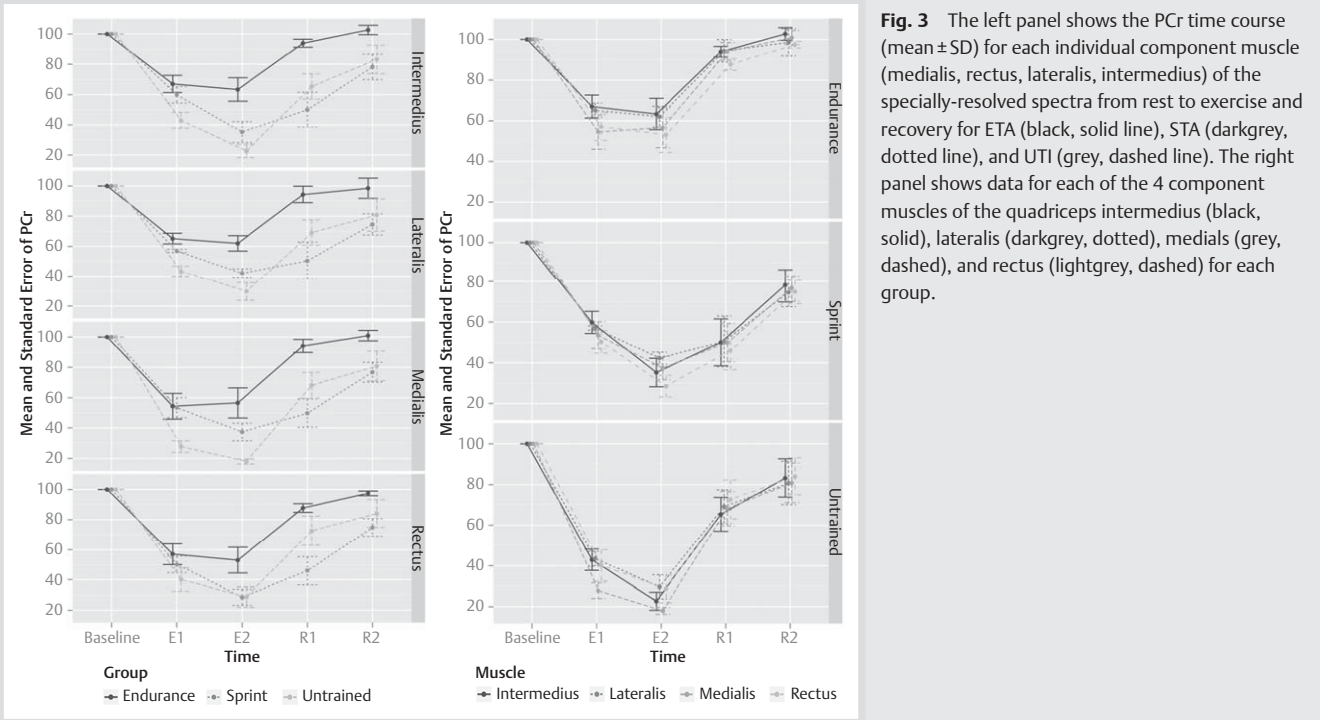

Fig. 3 The left panel shows the PCr time course (mean ± SD) for each individual component muscle (medialis, rectus, lateralis, intermedius) of the specially-resolved spectra from rest to exercise and recovery for ETA (black, solid line), STA (darkgrey, dotted line), and UTI (grey, dashed line). The right panel shows data for each of the 4 component muscles of the quadriceps intermedius (black, solid), lateralis (darkgrey, dotted), medials (grey, dashed), and rectus (lightgrey, dashed) for each group.

Table 1 Means and standard deviation are given for PCr (upper panel) and pH (lower panel) for every component muscle and every group over the time course of the exercise test, E-exercise; E-recovery.

PCr						
Muscle	**Group**	**Baseline**	**E1**	**E2**	**R1**	**R2**
intermedius	ETA	100.0 (0.0)	67.0 (11.3)	63.3 (15.5)	93.9 (5.2)	102.6 (6.2)
	STA	100.0 (0.0)	59.8 (12.2)	35.2 (15.6)	50.0 (25.7)	78.2 (18.7)
	UTI	100.0 (0.0)	42.9 (14.1)	22.6 (11.5)	65.3 (22)	83.2 (25)
lateralis	ETA	100.0 (0.0)	65.2 (7.1)	62.0 (10.2)	94.3 (11.1)	98.5 (13.3)
	STA	100.0 (0.0)	57.0 (2.4)	42.2 (6.6)	50.5 (27.9)	74.5 (15.6)
	UTI	100.0 (0.0)	43.5 (9.4)	29.6 (15.5)	69.1 (22.2)	80.8 (28.3)
medialis	ETA	100.0 (0.0)	54.4 (16.9)	56.6 (19.8)	94.1 (8.7)	100.9 (6.8)
	STA	100.0 (0.0)	53.5 (14.8)	37.3 (12.8)	49.8 (21.1)	76.7 (14.6)
	UTI	100.0 (0.0)	27.8 (10.1)	18.0 (4.5)	68.2 (22.4)	80.8 (25.8)
rectus	ETA	100.0 (0.0)	57.1 (13.7)	53.0 (17.3)	87.6 (5.7)	97.4 (3.2)
	STA	100.0 (0.0)	50.2 (12.4)	28.5 (11.9)	45.9 (21.1)	74.8 (12.9)
	UTI	100.0 (0.0)	40.1 (20.4)	28.6 (17.8)	72.7 (25.5)	84.0 (24.1)
pH						
Muscle	**Group**	**Baseline**	**E1**	**E2**	**R1**	**R2**
intermedius	ETA	6.97 (0.04)	6.99 (0.03)	6.98 (0.06)	6.99 (0.10)	6.96 (0.05)
	STA	7.02 (0.04)	6.95 (0.01)	6.9 (0.030)	6.86 (0.04)	6.87 (0.05)
	UTI	7.04 (0.05)	6.99 (0.04)	6.88 (0.05)	6.90 (0.12)	7.02 (0.13)
lateralis	ETA	7.04 (0.08)	7.03 (0.03)	7.02 (0.02)	6.95 (0.09)	6.95 (0.04)
	STA	7.03 (0.05)	7.01 (0.03)	6.98 (0.02)	6.92 (0.11)	6.88 (0.08)
	UTI	7.09 (0.06)	7.05 (0.02)	6.95 (0.08)	6.98 (0.14)	7.08 (0.14)
medialis	ETA	7.03 (0.07)	7.01 (0.05)	6.98 (0.02)	6.92 (0.05)	7.01 (0.09)
	STA	7.03 (0.08)	7.00 (0.04)	6.93 (0.07)	6.87 (0.06)	6.89 (0.10)
	UTI	7.07 (0.07)	6.99 (0.05)	6.90 (0.09)	6.90 (0.05)	6.98 (0.05)
rectus	ETA	7.00 (0.04)	7.01 (0.03)	6.98 (0.04)	6.90 (0.10)	6.97 (0.04)
	STA	7.11 (0.06)	6.99 (0.02)	6.87 (0.07)	6.80 (0.06)	6.80 (0.06)
	UTI	7.06 (0.06)	7.02 (0.04)	6.88 (0.04)	6.87 (0.08)	6.97 (0.07)

Post-hoc tests showed that the ETA had higher values than the STA during recovery in all quadriceps compartments (R1, $P<0.01$; R2, $P<0.05$). Also during E2 the values were higher in the intermedius ($P<0.01$) and rectus ($P<0.05$).

Additionally, the ETA maintained higher relative PCr concentrations than the UTI at all times for the intermedius and medialis (E1, $P<0.01$; E2, $P<0.01$; R1, $P<0.01$; R2, $P<0.05$). Except for R2, this was also the case in lateralis, while in rectus there was only a difference at E2.

Except for the intermedius, PCr was higher in the UTI than in the STA at R1 ($P<0.05$). Furthermore, during exercise, PCr in the medialis was higher in the UTI than the STA (E1, $P<0.01$; E2, $P<0.05$).

Fig. 4 shows pH for the 3 groups for each quadriceps component muscle. Details about means and standard deviations are given in **Table 1**. The pH data reveal significant differences between training groups. When analysing the pH values of the 3 groups (separately for each quadriceps muscle) we find that there is a highly significant time effect for all compartments (ANOVA; $P<0.01$), while a significant group effect can only be found for rectus (ANOVA; $P<0.05$). The interaction effect was highly significant in intermedius and rectus (ANOVA; $P<0.01$) and significant in lateralis (ANOVA; $P<0.05$).

Subsequent posthoc tests showed that pH for the ETA was higher than for the UTI at E2 and R1 ($P<0.05$) and also higher than for the STA at R1 ($P<0.01$). At R2, pH for the STA was much lower than for the UTI ($P<0.01$). At R2, the UTI showed a higher pH than the ETA ($P<0.05$) and the STA ($P<0.01$) in lateralis. In rectus, pH for the STA ($P<0.01$) was higher than for the ETA at baseline and lower than the ETA at E2 ($P<0.01$), R1 ($P<0.05$) and R2 ($P<0.01$). pH in the STA was also lower than for the UTI during recovery (R1, $P<0.05$; R2, $P<0.01$).

The right panel of **Fig. 3, 4** depicts changes of PCr and pH for the 4 compartment muscles of the quadriceps at the 5 time points, separately for each training group. Analysis of variance for PCr revealed that there is a significant time effect for all training groups (ANOVA; $P<0.01$). No significant group or interaction effect could be found. Similarly for pH, only time was significant (ANOVA; $P<0.01$) for all 3 groups. In addition, in the STA group a significant interaction effect could be observed (ANOVA; $P<0.01$), showing different changes in the compartments over the course of time. No Post-hoc tests were performed.

Muscle volume calculated from 40 slices of the quadriceps muscle of one leg was 2866 ± 339 cm^3 and 2066 ± 179 cm^3 for the STA and the UTI, respectively ($P<0.05$) but not different from ETA (2406 ± 250 cm^3).

No significant differences in resting concentrations of Pi and PCr were found among STA (PCr: 43 ± 12; Pi: 4.7 ± 1.8 mmol.L^{-1}), ETA (PCr: 40.0 ± 8.5; Pi: 4.7 ± 1.6 mmol.L^{-1}) and UTI (PCr: 45.0 ± 2.3; Pi: 4.4 ± 0.9 mmol.L^{-1}). The absolute values of PCr at the end of exercise, however, are lowest in the STA (17.4 ± 5.1 mmol.L^{-1}) and significantly ($P<0.05$) different from end-exercise values of the ETA (25.4 ± 2.6 mmol.L^{-1}). The UTI (22.5 ± 5.7 mmol.L^{-1}) did not differ from ETA and STA, respectively.

Discussion

To our knowledge, this is the first study to assess muscle metabolism by dynamic spatially-resolved ^{31}P MRS in sprint- and endurance-trained athletes and untrained subjects, representing, next to the untrained state, 2 extremes of muscular adaption, and to ask whether heterogeneity of skeletal muscle results in local metabolic differences between different regions (rectus, medialis, lateralis, intermedius) of the quadriceps. The main dif-

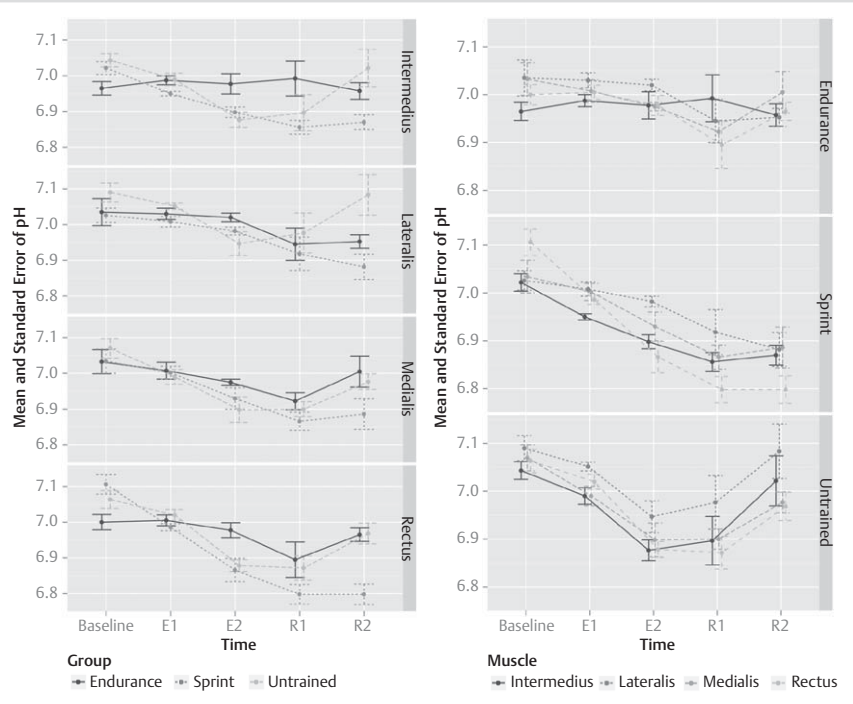

Fig. 4 The left panel shows the pH time course for each individual component muscle (medialis, rectus, lateralis, intermedius) of the spatially-resolved spectra from rest to exercise and recovery for ETA**,** STA and UTI**.** While pH did not drop in ETA, STA ($P<0.01$) and UTI ($P<0.05$) showed a lower pH during recovery. The right panel shows data for each of the 4 component muscles of the quadriceps for each group; see **Fig. 3** for abbreviations.

ferences among the groups were the smaller pH changes and PCr changes during exercise, and 40% faster PCr recovery in ETA compared to the STA and UTI. The exercise differences probably reflect higher contractile costs in STA and UTI, and lower capacity for oxidative PCr recovery within the exercise-recovery protocol used here.

PCr recovery kinetics mainly reflect mitochondrial function, and when there is no significant drop in pH or ATP [22,29], as in our experiments, PCr recovery follows first-order exponential kinetics and τ is independent of exercise intensity and end-exercise PCr [45]. Our findings therefore indicate differences in functional mitochondrial performance between STA and ETA compared to UTI and since O_2 supply is non-limiting after exercise in normal volunteers, PCr recovery should reflect mainly intrinsic mitochondrial enzyme function and mitochondrial numbers [13]. It is well established that endurance athletes with a history of low volume training and speed athletes with a history of mainly high-intensity strength and speed training show distinct patterns of muscle enzyme activities [8] whereas untrained people, according to their demand, possess an approximately uniformly distributed muscle fibre spectrum [46]. Our finding of ~40% higher oxidative capacity in ETA is in agreement with Crowther [6] who reported ~50% higher oxidative capacity and with Costill [4] who reported ~40% higher SDH activity in distance runners. However, we did not observe a difference as large as the 2.1-fold difference in τ between sprinters and distance runners reported by McCully [31]. This might be related to performance-related differences in the populations studied. The τ reported by Takahashi [42] following moderate intensity exercise in untrained controls (41±3 s) as well as in runners (34±2 s) is well in the range of our findings for the UTI (40±9 s) and the ETA (30±4 s), respectively. However, τ increased when subjects from [42] were exposed to more severe exercise, being influenced by a significant pH drop.

Our ETA subjects were able to maintain PCr during exercise, their high oxidative capacity allowing them to reach a PCr steady state after only 2 min exercise. Indeed, one subject, at the time of the study a semi-professional cyclist who is now participating in the Tour de France as a professional, achieved partial PCr recovery in the second increment of exercise. This might be related to the variation of motor units activated in these athletes, which helps to spread the work load across the pool of motor units and hence reduce the requirements on a given motor unit. This selective recruitment of motor units by the central nervous system has been observed during submaximal exercise [10].

In the STA subjects, PCr kinetics during exercise behaved quite differently. In contrast to the purely oxidative kinetics of PCr recovery, PCr changes during exercise reflect both mitochondrial function [7] and anaerobic metabolism. It is evident from the submaximal exercise session that STA as well as UTI showed a progressive PCr depletion. In contrast to the STA, the UTI as well as the ETA showed a rather quick recovery of PCr after cessation of exercise. Recovery, however, was clearly delayed in the STA. The absolute values of PCr depletion at the end of exercise also reflect the markedly different situation in the muscles of the 3 groups. This is in line with the concept of delayed onset of oxidative phosphorylation due to marked PCr hydrolysis [11].

This major difference between STA, ETA and UTI therefore probably reflects fundamental differences in muscle phenotype. However, it is not known whether skeletal muscle remodelling to a homogenous metabolic phenotype is characteristic in response to a long training process or whether genetic endowment might be the reason for a uniform musculature in these high-performance athletes.

In some subjects we observed an increase of PCr concentration above the resting level after termination of exercise. This overshoot was most pronounced in 2 subjects from the STA (recovery to 112 and 111%). A moderate overshoot was observed in one subject from the ETA (106%), whereas it was absent in the UTI. The PCr recovery overshoot is well known and related to stressing factors such as greater acidification of muscle cells [47] which is in line with our observations of a pronounced pH decline in the STA (right panel of **Fig. 4**). The reason might be a slow decay during recovery of the direct activation of oxidative

phosphorylation during muscle work [24]. The reason for the slight increase in pH at the end of exercise is unclear. We speculate that the exercise induced acidosis might be followed by an augmented ventilatory demand causing a slight respiratory alkalosis upon cessation of exercise in the UTI.

Muscles adapt to training by changing fibre type distribution [35]. Adaptations to endurance training include increased oxidative enzyme activities and mitochondrial density. In contrast, strength training reduces mitochondrial density [43], and sprint-trained athletes predominantly express fast type IIA and IIX fibres, which have lower mitochondrial enzyme activities than type I fibres [27], higher force, and preference for the glycolytic pathway. Consequently, although type II fibres, especially type IIA fibres, can contain many mitochondria, even if the absolute number of mitochondria is unchanged by strength training, as fibres enlarge these are 'diluted' as the cytoplasmic volume per myonucleus (myonuclear domain) increases, especially in fibres expressing fast MHC [39]. This is reflected in reduced activity of oxidative enzymes relative to protein content. Endurance athletes show metabolic adaptations and higher mitochondrial capacity related to specific long-term training which distinguish them from sprint- and strength-trained athletes [1,17]. Further, the cardiovascular system of sprint-trained athletes is less developed than that of endurance-trained athletes. Metabolic differences between these 2 groups have also recently been shown during blood flow restriction [41]. Their faster PCr recovery kinetics are in this sense a system property.

Data on the fibre composition in different muscles of the same subject are scarce. Autopsy data indicate ~50/50% type I and II fibres in vastus lateralis and rectus femoris [9]. Biopsy studies in untrained subjects suggest ~50% type I fibre content in vastus lateralis [40]. No data are available on intermedius and vastus medialis. The published data on adaptation to training is derived from biopsy analysis of vastus lateralis [44]. We found that different muscle groups of the quadriceps showed a similar metabolic behaviour, which supports the notion that the quadriceps constitutes a homogenous muscle group in trained athletes as well as untrained individuals. Specialized long-term training leads to metabolic adaptations in the whole muscle in terms of the biochemical properties, perfusion and composition of fibre types [44]. This metabolic response of the whole muscle has been discussed before in highly trained athletes [5] and surely increases the metabolic potential of the quadriceps.

The muscles of these athletes represent the full range of properties responsible for their extreme performances with untrained individuals somewhere in between of the metabolic extremes. We found no differences in apparent PCr and Pi concentration at rest, in line with earlier observations [6] but in contradiction to others [2]. However, we did not attempt a calibrated absolute quantification of resting metabolites, which poses its own technical difficulties [20].

Most historic MRS studies on humans have focused on calf or forearm muscle for practical reasons relating to the bore size. Other things being equal, the larger mass of the quadriceps makes it a better surrogate for whole-body oxidative capacity. Further, it is the main locomotor muscle for untrained people and is used in endurance and sprint athletes during training and competition. Although the methodology and temporal resolution of the FID and spatially resolved CSI spectra are very different, the implications for PCr kinetics are in good agreement. There are substantial technical limitations on the acquisition of CSI data with sufficient time resolution for formal fitting of PCr recovery kinetics.

Taken together, our study nicely shows marked differences regarding high-energy phosphate metabolism and pH in the muscles of high-performance athletes representing 2 extremes of endurance and sprint performance as well as in untrained individuals assessed by non-invasive in vivo MRS.

Limitations

The number of subjects might be a limitation. It is, however, very rare to have subjects at hand that show such a high level of performance in their field. Due to their tight training schedule, these subjects are difficult to recruit for a scientific study. For the same reason we could not obtain a muscle biopsy sample from the athletes to determine their fibre type profile.

Conclusion

Our study shows that post-exercise PCr recovery kinetics are closely related to the physical adaptations and performance level of the population studied, presumably reflecting the higher type I fibre type content and other metabolic adaptations of endurance as compared to sprint training. These changes may be associated with improved exercise efficiency in highly trained athletes if compared to untrained subjects. Furthermore, spatially-resolved data during exercise and recovery show a close similarity between the 4 components of quadriceps within each group, suggesting that the whole muscle functions, at least to a first approximation under these conditions, as a consistent metabolic unit. The results show the suitability of ^{31}P MRS as a way to quantify the divergent metabolic adaptations of sprint-trained and endurance-trained athletes as well as the response of untrained individuals to exercise. ^{31}P MRS qualifies as an alternative, non-invasive tool to study mitochondrial function in different populations.

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