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Adding strength to endurance training does not enhance aerobic capacity in cyclists

N. Psilander[1,2]*, P. Frank[1,2]*, M. Flockhart[1], K. Sahlin[1]

[1]Åstrand Laboratory, GIH, The Swedish School of Sport and Health Sciences, Stockholm, Sweden, [2]Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden
Corresponding author: Niklas Psilander, PhD, The Swedish School of Sport and Health Sciences, Lidingövägen 1, 11486 Stockholm, Sweden. Tel: +46 8 12053812, Fax: +46 8 120 538 80, E-mail: niklas.psilander@gih.se

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The molecular signaling of mitochondrial biogenesis is enhanced when resistance exercise is added to a bout of endurance exercise. The purpose of the present study was to examine if this mode of concurrent training translates into increased mitochondrial content and improved endurance performance. Moderately trained cyclists performed 8 weeks (two sessions per week) of endurance training only (E, $n = 10$; 60-min cycling) or endurance training followed by strength training (ES, $n = 9$; 60-min cycling + leg press). Muscle biopsies were obtained before and after the training period and analyzed for enzyme activities and protein content. Only the ES group increased in leg strength (+19%, $P < 0.01$), sprint peak power (+5%, $P < 0.05$), and short-term endurance (+9%, $P < 0.01$). In contrast, only the E group increased in muscle citrate synthase activity (+11%, $P = 0.06$), lactate threshold intensity (+3%, $P < 0.05$), and long-term endurance performance (+4%, $P < 0.05$). Content of mitochondrial proteins and cycling economy was not affected by training. Contrary to our hypothesis, the results demonstrate that concurrent training does not enhance muscle aerobic capacity and endurance performance in cyclists.

Endurance training is an efficient way to improve aerobic capacity that promotes both health and athletic performance. Traditional low- and moderate-intensity training is efficient in improving aerobic capacity in untrained individuals (Davis et al., 1979; Poole & Gaesser, 1985), whereas the effect in well-trained subjects is less prominent or absent (Laursen & Jenkins, 2002; Sandbakk et al., 2013). It is therefore essential to develop new training strategies to improve aerobic capacity and performance in trained individuals.

Strength training stimulates contractile protein synthesis and hypertrophy (Tesch et al., 1987; Green et al., 1999) and sometimes also mitochondrial biogenesis (Sale et al., 1990b; Wilkinson et al., 2008). It has also been shown that adding strength training to an endurance training program can increase endurance performance (Aagaard et al., 2010; Ronnestad et al., 2010). A combination of endurance and strength training (concurrent training) might therefore be a potential training strategy to promote muscle oxidative capacity. However, most studies of concurrent training show no or minor effects on mitochondrial enzyme activities (i.e., mitochondrial biogenesis) (Hickson et al., 1988; (Bishop et al., 1999; Bell et al., 2000). A possible explanation for this could be the study design. These studies used a training protocol where the two modes of exercise were separated by several hours or days. Only two previous studies, one acute exercise study from our laboratory (Wang et al., 2011) and a long-term training study (Sale et al., 1990a), have examined the effect of endurance training immediately followed by strength training. These studies show that, in relatively untrained subjects, concurrent training has a positive effect on the signaling leading to mitochondrial biogenesis (Wang et al., 2011) as well as on mitochondrial enzyme activity (Sale et al., 1990a).

Adaptation to training at the muscular level occurs by a sequence of events where transcription of DNA to mRNA is an early component in protein synthesis. Measurement of gene expression after one bout of exercise is a powerful tool to study the signaling response to exercise, which subsequently leads to training adaptation. Peroxisome proliferative-activated receptor-γ coactivator 1α (PGC-1α) is a major genetic marker of mitochondrial biogenesis because it has a key role in inducing several mitochondrial transcription factors such as the nuclear respiratory factors (NRF-1 and NRF-2) and mitochondrial transcription factor A (Wu et al., 1999; Jager et al., 2007). We have previously examined the acute effect of combined endurance and strength training on mRNA levels of PGC-1α and other genes related to muscle oxidative capacity (Wang et al., 2011).

*Contributed equally.

Table 1. Physical characteristics and training background of subjects

	E		ES	
	Pre	Post	Pre	Post
Age (years)	36 ± 2	–	34 ± 2	–
Height (cm)	184 ± 1	–	186 ± 2	–
Mass (kg)	80.3 ± 1.9	79.8 ± 1.7	79.6 ± 2.0	79.9 ± 2.3
Fat free mass (kg)	70.2 ± 1.8	69.9 ± 1.6	72.0 ± 1.8	72.0 ± 1.8
Cycling experience (years)	5.8 ± 1.4	–	5.0 ± 1.6	–
Training time (h/week)	6.8 ± 0.5	–	7.1 ± 1.0	–

Values are reported as means ± standard error.

E, endurance training only (n = 10); ES, endurance + strength training (n = 9).

In untrained subjects, we observed a twofold higher PGC-1α expression when resistance exercise was added in close vicinity to a bout of endurance exercise. These results together with the concurrent trainings study by Sale et al. (1990a) suggest that combining endurance and strength training within the same training session might be a potent stimulator of mitochondrial biogenesis. However, because these studies were performed on relatively untrained subjects, it remains to be shown whether concurrent training with this protocol is efficient also in trained subjects.

The purpose of the present study was to examine if muscle oxidative capacity and endurance performance can be enhanced by combining strength and endurance in the same training session. We hypothesized that this mode of concurrent training would enhance muscle oxidative capacity and cycling performance.

Methods

Subjects

Nineteen moderately trained male cyclists [34.7 ± 1.2 years; mean ± standard error (SE)] completed all parts of the study. The subjects were assigned to groups performing either endurance (E; n = 10) or concurrent training (ES; n = 9). Physical characteristics of subjects are shown in Table 1. One subject originally assigned to the ES group interrupted the training due to back pain and was excluded from the study. Prior to inclusion in the study, subjects were screened for endurance capacity with a lactate threshold test (LT4; i.e., work rate corresponding to 4 mmol lactate per l blood) and subjects with lactate threshold work rate below 2.5 W kg/bw were excluded. During this initial session, subjects were also familiarized with the experimental procedure to measure endurance performance (40-min time trial; TT40) and muscle strength (leg press) and were also interviewed about their training routines. Fat free mass was calculated from skinfold thickness measured with caliper (Harpenden, Baty International CTD, West Sussex, UK). The two groups were matched for endurance and strength capacity as well as for training background.

The subjects were instructed to keep a balanced diet throughout the intervention and to avoid diets with a low amount of calories or carbohydrates. The subjects recorded food intake during the 36 h preceding pretests and were instructed to duplicate the diet prior to post-tests. Pre- and post-tests were performed 3 h after the last meal at the same time of the day for each subject.

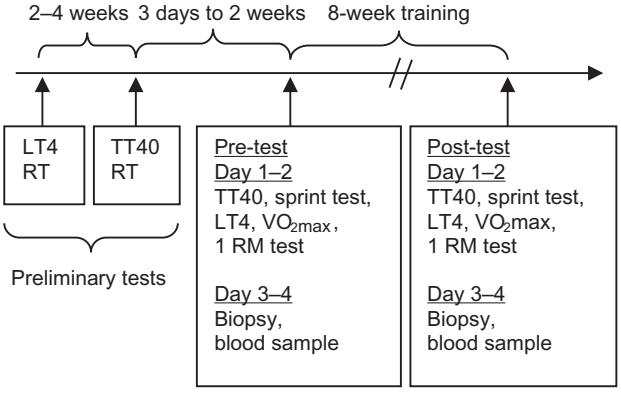

Fig. 1. Schematic diagram of the experimental protocol. LT4, work rate corresponding to 4 mmol lactate per l blood; TT40, 40-min time trial; RT, resistance training.

Subjects were informed about the possible risks and discomforts involved in the experiment prior to giving their written consent to participate in the study. The study design was approved by the Regional Ethics Committee of Stockholm, Sweden.

Experimental protocol (Fig. 1)

Time trial (TT40) and 30-s sprint tests

On the first day of testing the subjects performed TT40 on an ergometer (SRM, Konigskamp, Germany), preceded by warm up for 10 min. Seat height and handle bars were adjusted for each subject and used during all subsequent tests. To standardize the test, subjects received information about the pacing strategy that they used during the preliminary test and were instructed to repeat this during the pre- and post-test. Subjects completed TT40 without any feedback other than the remaining time. Power output, cadence, and heart rate (HR) were measured continuously. After TT40 subjects had a total of 20 min of easy pedaling (~70 W) followed by a 30-s maximal isokinetic sprint at 115 rpm in an "all out" fashion (Wingate test) with strong verbal encouragement. The sprint was performed seated and the subjects were informed of every 10 s elapsed. Peak power was defined as the highest mean power output during 0.5 s at any time during the sprint.

Tests of LT4, VO_{2max}, and strength

On the second day of testing, LT4 was determined during incremental submaximal exercise (5-min cycling at each step: 100, 150, 180, 210, etc. W). Capillary blood samples were collected from the

finger tip during the 1-min period of rest between each step and analyzed for lactate. The composition of expired gas and HR were measured continuously with Oxycon Pro (Erich Jaeger GmbH, Hoechberg, Germany) and Polar Electro Oy (Kempele, Finland), respectively. Lactate threshold work rate was defined as the interpolated work rate corresponding to 4 mmol lactate per liter blood. Cycling economy (CE), defined as work rate divided by VO_2, was determined at the same work rate (75–80% of VO_{2max}) pre- and post-training. The submaximal test was followed by 5-min unloaded pedaling before the max test where work rate, starting at LT4 (measured during pre-test), was increased by 20 W each min until fatigue (cadency 90–94 rpm). RPE was rated according to the Borg category scale (6–20 scale). VO_{2max} was measured as the highest recorded mean oxygen uptake during 40 consecutive seconds. The criteria for attaining VO_{2max} (RPE ≥ 18, RER ≥ 1.1, and a plateau of VO_2 with increased workload) were fulfilled for all subjects. Time to exhaustion during the VO_{2max} test (TTE-VO_{2max}) was defined as the time point when cadence was involuntarily dropped below 80 rpm. Following the max test, the subjects performed 15 min of easy pedaling on the ergometer before measurement of leg strength.

Maximal strength during leg press (243 Leg Press 45°, Gymleco, Stockholm, Sweden) was measured at a knee angle of 90°. The subjects performed three warm-up sets, and then the load was progressively increased until the subjects could not perform more than one single repetition. The rest between sets was 3–5 min.

Training

The subjects were instructed not only to continue their habitual cycle training but also to exchange two ordinary training sessions per week with supervised laboratory training. The first eight supervised training sessions consisted of 60 min of continuous cycling at a work rate corresponding to 90% of the mean power output during TT40 in the pre-test. The work rate was then raised to 92.5% (sessions 9–12) and to 95% (sessions 13–16). Following 10- to 15-min rest the subjects in the ES group performed strength training in a leg press machine (243 Leg Press 45°, Gymleco). The session started with a warm-up set with 10 repetitions at 50% of 1 RM (determined in pre-test) followed by sets at 65%, 70%, 75%, 75%, 70%, and 65% of 1 RM. The subjects were instructed to do as many repetitions as possible and if they were able to complete 15 reps the load was increased by 5% the next set. Instead of resistance exercise the E group cycled for 2.5–4.0 min corresponding to an equal amount of energy expenditure.

Subjects were instructed to record their training sessions outside the laboratory as a rough estimate of total training volume. When subjects missed supervised training sessions due to illness or of other reasons, the training intervention period was extended until at least 14 of the 16 scheduled training sessions were completed. No subject was absent more than three sessions altogether or two consecutive sessions. The last strength training session was performed with half the number of repetitions in order to minimize interference with the performance tests.

Analytical methods

Muscle and blood sampling

Muscle and blood samples were collected in the rested state 24–48 h following the last test after an overnight fast or at least 2 h after their last meal. The timing of blood and muscle sampling was the same pre- and post-training for each subject but varied between subjects. Muscle samples were obtained from the middle portion of the vastus lateralis muscle. After local anesthesia (2–4 mL Carbocain 20 mg/mL; Astra Zeneca, Södertälje, Sweden), an incision was made through the skin and fascia and muscle sample (50–100 mg) taken with the percutaneous needle biopsy technique with suction (Bergstrom, 1975). The sample was rapidly frozen in liquid nitrogen and stored at −80 °C. The frozen samples were thereafter freeze-dried, dissected free of blood and connective tissue, pulverized with forceps, and stored at −80 °C for later determination of enzyme activity and protein contents. Blood samples from an antecubital vein (4 mL) were centrifuged at 1500 *g* at 4 °C for 10 min and plasma stored at −20 °C for later analysis.

Analysis of blood samples

Capillary blood lactate was analyzed using an automated analyzer (Biosen 5140, EKF Diagnostics, Barleben, Germany). Venous plasma concentrations of cortisol and testosterone were determined with commercially available enzyme-linked immunosorbent assay kits (Calbiotech, Spring Valley, California, USA) using a plate reader (Tecan Infinite F200 Pro, Männedorf, Switzerland).

Analysis of muscle samples

For immunoblotting, samples were homogenized using a bullet blender (Bullet Blender 1.5, Next Advance, New York, USA) in ice-cold buffer (80 µL/mg) with the following composition (in mM): 2 HEPES, 1 EDTA, 5 EGTA, 10 $MgCl_2$, 50 β-glycerophosphate, 1% Triton X-100, 1 Na_3VO_4, 2 dithiothreitol, 20 µg/mL leupeptin, 50 µg/mL aprotinin, 1% phosphatase inhibitor cocktail (Sigma P-2850, St Louis, Missouri, USA), and 40 µg/µL PMSF. The homogenate was centrifuged at 10 000 rpm for 10 min to pellet the insoluble debris and the supernatant was stored at −80 °C. Protein concentration of the supernatant was determined with the bicinchoninic acid assay (Pierce Biotechnology, Rockford, Illinois, USA) by measuring the absorbance at 560 nm with a plate reader (Tecan Infinite F200 Pro). The samples were diluted with Laemmli sample buffer (Bio-Rad Laboratories, Richmond, California, USA) and homogenizing buffer (1:1) to a final protein concentration of 1.5 µg/µL and heated to 95 °C for 5 min to denature proteins. The diluted samples were stored at −20 °C prior to analysis. The proteins of the diluted samples were separated by SDS-PAGE (criterion cell gradient gels, Bio-Rad Laboratories) for 45 min at 300 V on ice and then transferred to polyvinylidene fluoride membranes (Bio-Rad Laboratories) for 3 h at 300 mA on ice. The amount of protein loaded to the membranes (20 µg) was kept constant for all samples and was verified by staining with MemCode™ Reversible Protein Stain Kit (Pierce Biotechnology). After blocking for 1 h at room temperature in 5% non-fat milk, the membranes were incubated overnight with primary antibodies. This was followed by 1 h incubation with anti-rabbit or anti-mouse HRP (1:10 000) as secondary antibody. The antibodies were visualized by chemiluminescent detection on a Molecular Imager ChemiDoc™ XRS system, and the bands were analyzed using Quantity One® version 4.6.3 software (Bio-Rad Laboratories). The used primary antibodies were citrate synthase (CS), hydroxyacyl-CoA dehydrogenase (HAD) (GeneTex, Irvine, California, USA, catalog number GTX 110624 and GTX 118325), PGC-1α (Millipore, Billerica, Massachusetts, USA, catalog number ST1202), and OXPHOS (Abcam, Cambridge, UK, catalog number AB110411). Anti-rabbit or anti-mouse HRP (Cell Signaling, Danvers, Massachusetts, USA) was used as secondary antibody.

For determination of enzyme activity, muscle samples were homogenized using a bullet blender in a buffer (150 µL/mg) with the following composition (in mM): 50 K_2HPO_4, 1 EDTA, and 0.05% Triton X-100 adjusted to pH 7.4. The homogenate was centrifuged at 10 000 rpm for 10 min, and the supernatant was collected and diluted three times. CS activity was measured in a reagent solution (in mM): 50 Tris-HCl, 0.2 DTNB, and 0.1

acetyl-CoA. The reaction was initiated by adding oxaloacetate (7 mM) and the change in absorbance at 412 nm was measured spectrophotometrically at 25 °C. HAD activity was measured in a reagent solution (in mM): 65 Triethanolamine HCL, 0.3 EDTA, and 0.3 NADH adjusted to pH 7.0. The reaction was initiated by adding acetoacetyl coenzyme A (4 mM) and the change in absorbance at 340 nm was measured spectrophotometrically at 25 °C.

Statistical analysis

Data are presented as means ± SE. Differences between E and ES were tested for statistical significance with two-way repeated measures analysis of variance (ANOVA). When a significant main effect of training was found, differences within groups were tested with a paired *t*-test using Statistica (StatSoft, Inc, Tulsa, Oklahoma, USA). Statistical significance was accepted at $P < 0.05$.

Results

Body composition, strength, and peak power

Body mass and lean body mass remained unchanged with training (Table 1). Muscle strength measured as 1 RM during leg press increased in ES (+19% ± 2%, $P < 0.01$) but not in E (+3% ± 2%, ns), resulting in a significant difference between groups ($P < 0.05$, Fig. 2). Peak power during the 30-s Wingate test increased in ES (+5% ± 2%, $P < 0.05$) but not in E (+1% ± 2%, ns) (Fig. 2), but mean power (W) was neither changed in ES (736 ± 26 pre vs 755 ± 30 post) nor in E (725 ± 19 pre vs 731 ± 18 post).

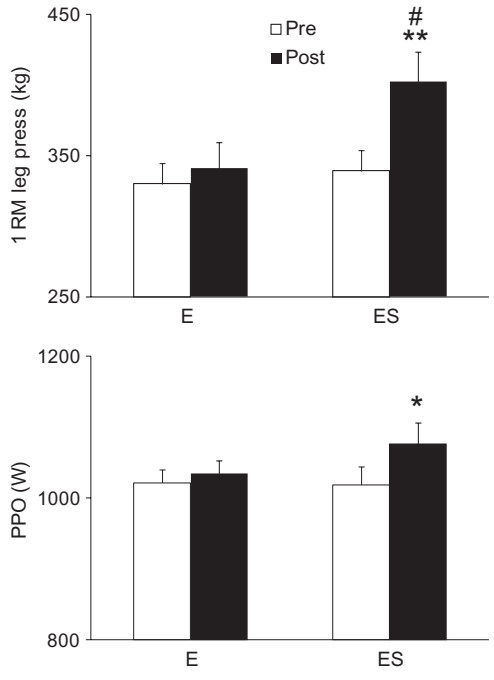

Fig. 2. Effect of 8 weeks of training on leg strength (1 RM leg press) and peak power output during the 30-s Wingate test (PPO). Values are reported as means ± standard error. E, endurance training only ($n = 10$); ES, endurance + strength training ($n = 9$). Main effect of training: *$P < 0.05$ vs pre-training; **$P < 0.01$ vs pre-training. Main effect of groups: #$P < 0.05$ ES vs E.

VO_{2max} and short-term endurance

VO_{2max} increased similarly in both groups (ES; +3% ± 1% and E; +4% ± 1%, $P < 0.05$, Table 2). Time to exhaustion during the VO_{2max} test increased in ES (+10% ± 3%, $P < 0.05$) but not in E (+7% ± 5%, ns) (Table 2).

Lactate threshold work rate, CE, and long-term endurance

LT4 increased in E (+3% ± 1%, $P < 0.05$) but not in ES (+1% ± 1%, ns) (Table 2). CE (W/mL O_2) was not changed following either type of training (ES: 75 ± 1 pre vs 76 ± 1 post; E: 75 ± 1 pre vs 75 ± 1 post). Mean power output during the 40-min time trial increased in E (+4% ± 1%, $P < 0.05$) but not in ES (+3% ± 2%, ns) (Table 2).

Muscle enzyme activities, hormones, and protein content

The ANOVA did not show a significant main effect of training for CS activity but a trend (paired *t*-test) toward increased CS activity was observed in E (+11% ± 5%, $P = 0.06$) but not in ES (–1% ± 7%, ns) (Fig. 3). HAD was unaffected by training in both groups (E: +9% ± 6% and ES: +3% ± 7%; both ns; Fig. 3). Training had no effect on the blood concentration of plasma testosterone and cortisol levels at rest or on muscle contents of proteins related to oxidative metabolism (Table 3 and Fig. 4).

Discussion

This is the first study to investigate if adding strength training immediately after endurance training alters mitochondrial biogenesis in trained individuals. The main finding was that 8 weeks of concurrent training did not enhance mitochondrial biogenesis and endurance performance.

Table 2. Effect of training on performance and performance related variables

	E		ES	
	Pre	Post	Pre	Post
VO_{2max} (mL/min/kg)	55 ± 1	58 ± 1**	57 ± 1	58 ± 1*
TTE-VO_{2max} (s)	402 ± 14	423 ± 15	407 ± 22	443 ± 21**
LT4 (W)	287 ± 11	295 ± 11*	283 ± 9	286 ± 8
TT40 (W)	284 ± 11	294 ± 11*	282 ± 10	290 ± 10

Values are reported as means ± standard error.
Main effect of training: *$P < 0.05$ vs pre-training; **$P < 0.01$ vs pre-training.
E, endurance training only ($n = 10$); ES, endurance + strength training ($n = 9$); LT4, work rate corresponding to 4 mmol lactate per l blood; TTE-VO_{2max}, time to exhaustion during the VO_{2max} test; TT40, mean power output during 40-min time trial.

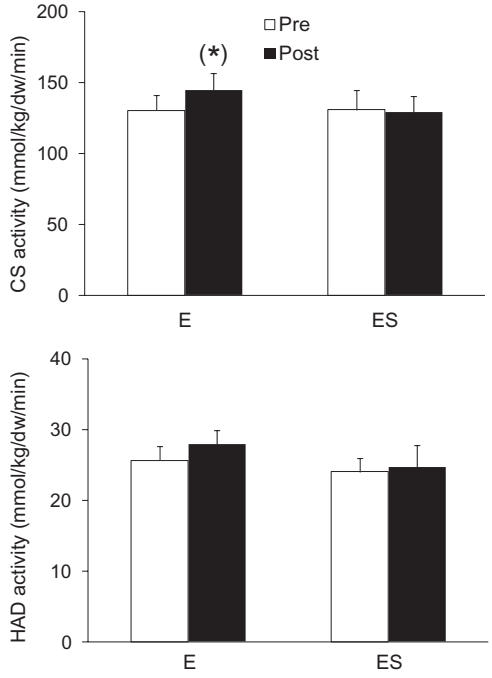

Fig. 3. Effect of 8 weeks of training on mitochondrial enzyme activities. Values are reported as means ± standard error. E, endurance training only ($n = 10$); ES, endurance + strength training ($n = 9$). CS, citrate synthase; HAD, 3-hydroxyacyl-CoA dehydrogenase. (*), $P = 0.06$ vs pre-training.

Table 3. Effect of training on hormones and muscle protein content

	E		ES	
	Pre	Post	Pre	Post
Plasma concentration (ng/mL)				
Cortisol	108 ± 18	94 ± 12	115 ± 22	111 ± 22
Testosterone	9.1 ± 0.7	9.2 ± 0.7	8.5 ± 0.8	8.5 ± 0.4
Protein content (AU)				
CS	6.6 ± 0.4	7.3 ± 0.5	8.0 ± 0.4	8.0 ± 0.6
HAD	5.0 ± 0.1	5.1 ± 0.3	5.8 ± 0.2	5.6 ± 0.3
PGC-1α-1 (113 kDa)	1.5 ± 0.2	1.4 ± 0.1	1.8 ± 0.2	1.6 ± 0.1
PGC-1α-4 (38 kDa)	3.1 ± 0.3	2.6 ± 0.3	3.3 ± 0.4	2.7 ± 0.3
Oxphos. C. I	3.4 ± 0.2	3.4 ± 0.2	3.7 ± 0.2	3.6 ± 0.2
Oxphos. C. II	3.6 ± 0.2	3.7 ± 0.2	3.5 ± 0.3	4.0 ± 0.4
Oxphos. C. IV	4.2 ± 0.2	4.2 ± 0.3	4.4 ± 0.4	4.6 ± 0.3
Oxphos. C. V	3.0 ± 0.4	3.0 ± 0.4	4.0 ± 0.3	4.2 ± 0.4

Muscle biopsies were obtained approximately 48 h before and after the first and last training session. Values are reported as means ± standard error.

E, endurance training only ($n = 10$); ES, endurance + strength training ($n = 9$); CS, citrate synthase; HAD, 3-hydroxyacyl-CoA dehydrogenase; Oxphos. C., oxidative phosphorylation complexes I–V; PGC-1α, peroxisome proliferative–activated receptor-γ coactivator 1α (isoform 1 and 4).

The subjects in the present study were cyclists with an average training volume of approximately 7 h/week. Part of their regular training was replaced with supervised laboratory training with the intention to keep the overall cycling training as constant as possible. However, CS

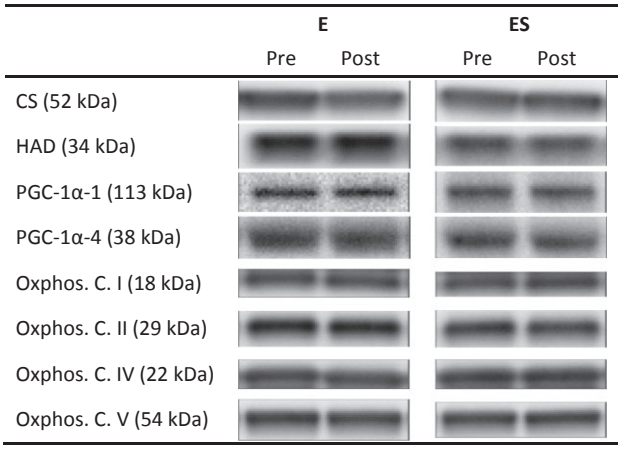

Fig. 4. Representative Western blots.

activity (trend), LT4, VO_{2max}, and TT40 were improved in the E group, indicating that the subjects were not in a steady-state mode. Obviously endurance training in the laboratory added training stimulus above that present during the subject's ordinary training, possibly due to more intense and regular training.

VO_{2max} increased similarly in both groups but, in contrast to the E group, endurance performance and markers of muscle oxidative capacity (LT4 and CS activity) were unchanged in the ES group. This suggests that adding strength training does not enhance muscle adaptation to endurance training, which is in opposite to our hypothesis as well as to the findings by Sale et al. (1990a). It is also in contrast with our previous finding that resistance exercise amplified the signaling response of mitochondrial biogenesis in muscle (twofold higher expression of PGC-1α compared with endurance exercise alone) (Wang et al., 2011). A possible explanation for this discrepancy could be that the subjects in the present study were trained cyclists whereas in our previous study and in the study by Sale et al. (1990a) the subjects were recreationally active. Previous studies have shown that relatively untrained subjects can increase their mitochondrial content with strength training (Sale et al., 1990b; Wilkinson et al., 2008), whereas this is normally not the case in trained subjects (Green et al., 1999; Wilkinson et al., 2008). The findings in our previous study of amplified expression of PGC-1α after concurrent exercise might therefore be due to an additive endurance stimulus from the resistance training session. The finding that resistance exercise alone increases mRNA of PGC-1α (Apro et al., 2013) supports this line of reasoning.

It has recently been shown that PGC-1α exists in several isoforms (at least four) with different roles in the signaling cascades preceding muscle adaptation. Studies in rat skeletal muscle have shown that PGC-1α1 is a marker of mitochondrial biogenesis, whereas another isoform, PGC-1α4, is more related to hypertrophy than to mitochondrial biogenesis (Ruas et al., 2012). In our

previous acute exercise study (Wang et al., 2011), we used primers that could not separate the different isoforms of PGC-1α, and it is therefore possible that the enhanced gene expression after concurrent exercise was due to increases in other isoforms than PGC-1α1 (e.g., PGC-1α4). However, recent human studies report that activation of PGC-1α4 (measured with a semi-quantitative technique) is not solely related to resistance exercise and hypertrophy (Lundberg et al., 2014; Ydfors et al., 2014) and the precise role of PGC-1α4 and other isoforms of PGC-1α in human skeletal muscle remodeling is therefore not clear.

Another possible explanation for the lack of training adaptation in muscle oxidative capacity (i.e., LT4 and CS activity) in the ES group might be a dilution of mitochondria as a result of hypertrophy (MacDougall et al., 1979; Chilibeck et al., 1999). Muscle or fiber cross-section area was not measured in the present study and, although lean body mass was unchanged, it is possible that our training protocol induced muscle hypertrophy and thereby dilution of the mitochondrial content. This idea is supported by a similar training study where increased type II muscle fiber hypertrophy was associated with reduced CS activity (Nelson et al., 1990).

After ES we observed the expected outcomes of strength training, i.e., increased muscle strength (1 RM during leg press), increased peak power output during 30-s Wingate cycling, and increased short-term endurance (TTE-VO_{2max}). Endurance performance during long-term exercise was, however, not improved, which was in contrast to our hypothesis. Several other studies have shown that adding strength training to the training program of endurance-trained subjects have a beneficial effect on long-term endurance performance (Paavolainen et al., 1999; Aagaard & Andersen, 2010; Ronnestad et al., 2010). The suggested mechanisms are that increased strength and power would result in attenuated blood flow restriction, faster force development in each movement cycle, or enhanced neuromuscular function (Aagaard & Andersen, 2010; Ronnestad & Mujika, 2013). Studies showing improved long-term performance after concurrent training have used training protocols where the two modes of exercise are separated by several hours or even days and it has been shown that strength training adaptations, such as strength and power, can be blunted by endurance training in close proximity (Sale et al., 1990a; Chtara et al., 2008; Wilson et al., 2012). Furthermore, improvements in long-term endurance performance seems to require strength training with high loads (85–95% 1 RM) and long durations (> 8 weeks) (Aagaard & Andersen, 2010), which was not the case in this study. The absence of performance improvement (TT40) in the ES group might therefore be explained by a combination of the relatively short training period, low training intensity, and the preceding endurance exercise.

Another possible explanation why the ES group did not improve in endurance performance could be that the subjects were not fully recovered when they performed the final TT40 test. We used a linear progression model in the present study and did not periodize the training. This in combination with the close vicinity between the endurance and strength training sessions might have induced some degree of overtraining. Even though we reduced the training volume of the last strength session and separated it from the post-tests by at least 48 h some subjects reported that they felt tired in their legs during these tests. However, self-reported training volume and intensity as well as markers of overtraining (testosterone and cortisol levels, Table 1) did not differ before and during/after the intervention period.

Perspectives

Traditional low- and moderate-intensity training is efficient in improving aerobic capacity in untrained individuals whereas the effect in well-trained subjects is less prominent or absent. Finding new models of training is therefore important especially for athletes, where the already large volume of training prevents further increases. Although strength training primarily induces muscle hypertrophy, there are some reports indicating that adding strength training immediately after endurance training have positive effect on the signaling leading to mitochondrial biogenesis as well as on mitochondrial enzyme activity. These studies were performed in relatively untrained subjects, and the aim of the present study was to investigate the effect of concurrent training in endurance-trained subjects. The major findings of this study was that combining endurance and strength training in the same exercise session resulted in improved strength, peak power, and short-term endurance. However, in contrast to endurance training only, long-term endurance and markers of muscle oxidative capacity (LT4 and CS activity) were not improved after concurrent training. The findings suggest that adding strength training in close vicinity after endurance training does not enhance the training-induced adaptation of muscle oxidative capacity. Endurance athletes are therefore recommended to train strength and endurance in separate training sessions.

Key words: Mitochondrial biogenesis, exercise, resistance training, gene expression, PGC-1α.

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